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A
TEXT-BOOK OF MEDICINE

FOR STUDENTS AND PRACTITIONERS

BY

DR. ADOLF STRÜMPELL

PROFESSOR AND DIRECTOR OF THE MEDICAL CLINIQUE AT
THE UNIVERSITY OF ERLANGEN

Third American Edition

*TRANSLATED BY PERMISSION FROM THE THIRTEENTH
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WITH ONE HUNDRED AND EIGHTY-FIVE ILLUSTRATIONS
IN THE TEXT, AND ONE PLATE

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AUTHOR'S PREFACE TO THE THIRTEENTH EDITION

THE extent to which my text-book has been used, both in Germany and in other countries,* justifies me in the assumption that it has exercised some little influence upon the professional thought and practice of numerous medical readers, and imposes upon me the obligation of employing every opportunity for the improvement and perfection of my work. Yet I must confess that this task seems more difficult to me with every new edition of the book, for scientific workers are making deep and extensive investigations into the many subdivisions of internal medicine, with such zeal and skill that it becomes difficult for any one man to keep step with the unceasing progress of investigation in this wide domain, and to maintain that complete and universal command of facts and theories which is essential for a presentation of the subject, which shall be at once brief and true to the present standpoint of science. The author has often been painfully sensible of these limitations of his personal knowledge and ability, but he hopes that this will be regarded not merely as subject for criticism, but also as an excuse and an incentive to clemency, in case a reader who is expert in any specialty finds here and there an omission or an error. Above all, I beg the reader to consider that no text-book such as the present one ought to replace, or could replace, the vast material of the larger reference books. My purpose was not to collect all the facts of pathology which have been discovered up to date, nor all the methods of treatment which may have been recommended, wisely or unwisely, nor all the theories or views which have been propounded. My wish was to give a complete presentation of the essentials of our present knowledge and views with regard to the various diseases, from a scientific and individual standpoint; and I desired particularly to impart to the reader an insight into the origin and relation of the various morbid phenomena. To this end I have brought the facts of clinical experience into the closest possible relation with the data of pathological anatomy and of general pathology, and have endeavored, also, in discussing therapeutics, to deduce from the nature of the symptoms a basis for rational medical opinion and treatment, although I have not undervalued the importance

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* So far as I have been informed, translations of my text-book have appeared in the following languages: French, English, Italian, Spanish, Russian, modern Greek, Turkish, and Japanese; and some of these translations have had several editions.

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of simple experience. A text-book intended for students and a larger circle of graduate readers ought not to incline too decidedly toward any one of the prevailing currents of scientific opinion. It should, however, acquaint the reader to such an extent with the questions at present under discussion relating to internal medicine, as to give him at least a fair start in his further studies.

The author has made an honest effort to bring the present thirteenth edition of this book up to the level of contemporary medical thought and knowledge. Large portions of the book have been almost completely rewritten—in particular, the whole doctrine of gastric diseases and several chapters in other sections, including gall-stones, intestinal parasites, etc. Almost all portions of the book have received numerous additions and improvements, so that this edition may be said with justice to be completely revised. That even now my work is imperfect I am well aware. I shall, therefore, be thankful to any one who will inform me of the mistakes or omissions he discovers.

A. STRÜMPELL.

TRANSLATORS' PREFACE

THE work of Professor Strümpell has been so long familiar that it is hardly necessary for the translators to introduce it again to the English-speaking portion of the medical profession or to express again their admiration of the work. Since the second edition of this translation was published in 1893 seven new editions have appeared in Germany, so that the book has been almost wholly rewritten, although it still retains all the merits of the earlier production, with the advantage of being fully in line with the most recent medical investigations. The translators have kept as close to the original as seemed consistent with clearness. The doses in the metric system have been changed to the approximate equivalents in apothecaries' weight, the original doses being retained in parentheses. As in the former editions, the translators have substituted specimens of handwriting in English in the chapter on general paralysis for the original in German script. These specimens were kindly furnished them by the late Dr. E. P. Elliot, of the Danvers Insane Hospital. The translators have also added a chapter upon the plague and various notes which they hope may prove of assistance to the American practitioner. These additions are inclosed in brackets and signed by the initials of the translators, to distinguish them from the (unsigned) additions, also in brackets, of Dr. Shattuck.

BOSTON, *June, 1901.*

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ACUTE GENERAL INFECTIOUS DISEASES

CHAPTER I

TYPHOID FEVER

(Typhus abdominalis. Enteric Fever. Ileotyphus)

Ætiology.—The sole cause of every attack of typhoid fever is an infection of the body by a definite pathogenic bacillus, the "typhoid bacillus" discovered by Eberth and Koch, and later made better known by the researches of Gaffky and others. Their length (see Fig. 1) is about one third the diameter of a red blood-globule, and their breadth equals one third their length, but sometimes they may grow out in long fibers. It is not yet certain whether spore formation takes place in their interior or not. The typhoid bacilli show a very active spontaneous movement in water, caused probably by very fine filiform threads which Löffler has discovered on the ends of the rods and which he regards as the motor organs of the bacilli.

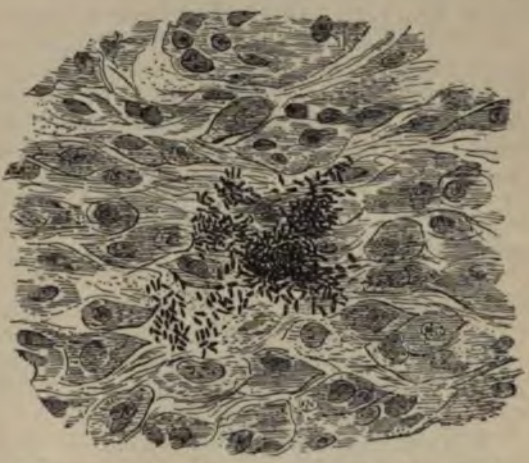

FIG. 1.—Typhoid bacilli. Section from the spleen. 800 : 1. (From FLÜGGE.)

That these typhoid bacilli are specific, is shown, however, as in the case of many micro-organisms, less by their external form than by their peculiarities, as observed in pure cultures of them, and by their other biological peculiarities (virulence, chemical reaction, etc.). By the latter the pure typhoid bacillus is distinguished from a form of bacillus morphologically similar and perhaps allied to it, which often is found in the intestine, and is called *bacterium coli commune* (Escherich). The attempt has even been made to regard the typhoid bacillus merely as a virulent form of the bacterium coli, but the weight of evidence is at present in favor of regarding the typhoid bacillus as an independent form.

In cultures on gelatine the typhoid bacilli develop along the line of puncture in little whitish or yellowish clusters, while a thin gray pellicle extends slowly over the surface, but the nutrient gelatine never liquefies. The manner of their growth on the cut surface of boiled potatoes is still more characteristic, and especially valuable for their recognition. The whole cut surface is covered with a dense pellicle, scarcely discernible with the naked eye, and forming an absolutely pure culture of typhoid bacilli. It is an important fact that typhoid bacilli may also thrive if deprived of oxygen, because we can thus understand their increase within the intestine.

The typhoid bacilli have thus far been found chiefly in the typhoidal infiltrations in the intestine, where they lie between the cells, and also in single foci in the mesenteric glands, the spleen, the liver, the kidneys, the pleura, the meninges, in typhoidal foci in the bones, etc. By suitable methods of investigation they are often found in the stools of typhoid patients and also in the fluid obtained by puncture of the fresh splenic tumor, in the gall bladder, sometimes in the urine, and also in the blood taken from a spot of roseola. As a rule, they are not found in the rest of the blood.

Numerous attempts have been made to produce typhoid fever artificially by introducing pure cultures of the typhoid bacilli into the bodies of animals, but the results of these efforts have not yet proved perfectly harmonious. The main cause of the discrepancy is probably that animals are in general very slightly susceptible to the disease. At any rate, the attempts at artificial infection up to this date have proved successful only in cases when the animals subjected to the experiment (rabbits, guinea-pigs) have received large amounts of the typhoid bacilli directly into a vein or into the abdominal cavity (E. Fränkel and Simmonds), or when the bacilli have been introduced into the duodenum (A. Fränkel). Probably, however, we have here to do rather with the intoxication of the animals caused by the poisonous matters generated in the cultures of bacilli than with an actual infection, for the pathological changes of typhoid fever are but little developed in the animals, and the injected bacilli themselves appear to be for the most part destroyed within the body of the animal experimented upon (Flügge and Sirotinin, and others). Attempts to produce the disease by mixing the dejecta of typhoid patients with the animal's food have thus far proved invariably unsuccessful. Probably the bacilli are immediately destroyed by the hydrochloric acid in the stomach.

Investigation of the ætiology of typhoid fever must consequently be directed to ascertaining in what manner and through what channels the specific typhoid bacilli penetrate into the human body, and what circumstances are then essential to their further development and to the display of their pathogenic properties. It must be confessed that the ability to answer these questions accurately is a goal from which we are quite distant.

It is almost universally believed that, as a rule, typhoid bacilli do not have any permanent, independent existence outside the human body, but sometimes under favorable conditions they seem to be able to remain for years in a "latent condition" in the external world (in moist earth or stagnant water). Often, however, the conditions essential to an abundant development of the bacilli arise in certain places, and thus make it possible for a greater or less number of persons to absorb the pathogenic poison, and, as a result, to be attacked by typhoid fever. In this way occur the numerous greater or smaller epidemics of typhoid fever in contrast to the sporadic cases, which are likewise possible, and are not infrequent. If an epidemic of typhoid appears in a place till then entirely free from the disease, we must always refer it to an importation of the disease-germs, and seek their source in some previous case of typhoid. We must, therefore, take for granted that the poison of typhoid can in some way escape from the body of the patient into the outer world. If we believe this, we shall be sure to think, first of all, of the intestinal discharges as the source of infection. These discharges, as already stated, are known to contain the typhoid bacilli or perhaps their spores.

As to the exact manner of infection, views are still widely different. Up to the present time there are chiefly two contrasted theories, called, respectively, the "ground-soil" and the "drinking-water"* theories. According to the former, which is maintained principally by Pettenkofer and his pupils, the ground-soil is to be regarded as the chief place of development for the schizomycetic fungus of typhoid fever. Whether this will flourish depends chiefly on the condition of the soil (varying at different times and in different places), and this alone should explain all the peculiarities observable in the spread of the disease—e. g., that single houses, streets, or wards of a city should suffer. According to Pettenkofer, a soil that air and water easily penetrate—e. g., one made up of alluvial or detrital deposits—is most favorable for the spread of the disease, while a firm, rocky bottom makes its further development impossible; and, where this "tendency of the ground-soil" is wanting, the disease can neither be introduced nor, if brought in by the sick, spread any further; for, according to Pettenkofer, the typhoid poison is seldom if ever transferred directly from one person to another. The poison in the stools must first be changed by the soil before it becomes infectious. The "ground-air," which is continually rising, carries the poison not only into the open atmosphere, but into the air of dwelling-rooms, and, being then inhaled, produces infection. We can thus understand why Pettenkofer regards typhoid fever as not directly contagious. The chief support of the ground-soil theory, beyond the results of comparing the character of the soil with the extent of the epidemics, consists in the proof which Buhl and Pettenkofer have given (taking Munich as an example, and later Berlin and other places) that a relation exists between the variations of the standing water in the soil and the frequency of typhoid cases. It appears that, when the water stands high (near the surface), fewer cases occur, and when it falls below the mean height cases are more numerous. Pettenkofer explains this relation by the fact that the level of the ground-water is certainly an index of the moisture and other conditions of the soil upon which the development of the typhoid bacilli depends.

To be contrasted, or rather compared, with the "ground-air" theory is the view held by most physicians, despite the vigorous protest of Pettenkofer, that drinking-water plays an increasingly important *rôle* in the origin of many epidemics of typhoid. In fact, in the case of numerous epidemics, whose extent bears an unmistakable relation to the water-supply, we are forced to believe that the typhoid germs are brought into the body by means of water used in drinking or otherwise. Even then we are by no means wholly to disregard the character of the soil, for the disease-producing poison—not to speak of direct pollution—is probably often communicated to the well-water from the soil. The possibility of this will be especially great if the wells are near drains or cess-pools containing typhoid discharges. In epidemics spread by drinking-water, the typhoid bacilli have lately been repeatedly found in the suspected water.

We believe the idea is continually gaining ground that no single "theory" can fully explain all the facts, and that the possibility of infection occurring in several different ways must be considered. The final source of infection is always to be sought for in a previous case of typhoid fever. From this patient the typhoid bacilli reach the external world through the dejections, and may sometimes directly reach another human body and infect it. Hence the experience that nurses, laundresses who wash the soiled linen, etc., often have typhoid fever; but typhoid fever is not contagious by conduction through the air. In most hospitals typhoid patients are not isolated from the rest of the sick, and yet outbreaks of typhoid hardly ever occur among them, provided they are not in close intercourse with the typhoid cases or do not carelessly use the same dishes, rectal thermometer, etc. In the further extension of the disease—that is, when there is an epidemic—water contaminated with typhoid bacilli certainly plays a frequent and most important part. All persons who indulge directly or indirectly (rinsing cooking utensils, etc.) in water from an infected spring or infected aqueduct are in danger of becoming ill. For example, it has been remarked in England, and lately in Cologne, that the fever in certain epidemics was limited to individuals who had their milk from one common source. In such cases, however, the probable cause is not a disease in the cows, but a pollution of the milk or the milk-cans by water. It is as yet doubtful if animals can have typhoid fever. This fact makes it uncertain whether the illnesses which have been observed to follow the ingestion of the flesh of diseased calves (e. g., the epidemic of Kloten) are actually to be considered typhoid fever, although the pathological changes are said by Huguenin to be very similar to those found in typhoid.

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* Compare with what follows the statements concerning the ætiology of cholera, where the same disputed points are considered.

[It is not probable that sewer-gas in itself is an exciting cause of typhoid fever. Especially in large cities typhoid dejections are constantly finding their way into the sewers, which afford all the conditions favorable to the further growth and development of the poison. If, then, the drainage of any house is defective, the seeds of the disease can readily gain access to the interior of the house and infect susceptible individuals.

One of the most instructive epidemics on record is that in Plymouth, Pennsylvania, a town of eight thousand inhabitants. In the spring of 1885 a disease, at first supposed to be of a strange character, broke out in the place, and, before it ceased, affected twelve hundred persons, causing one hundred and thirty deaths. It was soon found that the malady was typhoid fever, which arose from one case, briefly in this wise: In January, February, and March there was a case of typhoid in a house on a hill sloping toward a water-supply of the town. The dejections were thrown out on the snow, under which the ground was deeply frozen. On March 25th a sudden and great thaw occurred, the water did not sink into the ground, but ran immediately into the natural surface channels, and on April 10th the epidemic began. There were reasons, which it is not necessary here to detail, why the above source of water-supply was drawn upon to an unusual degree just at that time, but it has been shown that those who derived their water from other sources were spared by the disease. The original case came from Philadelphia.]

In almost all cases the intestine seems to be the actual gate of entrance for the typhoid poison into the human system. This is shown by the fact that in all cases which come to autopsy in early stages of the disease, the typhoid bacilli are mainly confined to the lymphatic tissues of the intestine. The typhoid poison (bacilli or spores) is probably swallowed, either directly with water or polluted food, or after being inhaled or in some other way introduced into the mouth. [Raw oysters grown in impure waters may convey the infection.—V.] If not destroyed in the stomach, it passes on in viable condition into the alkaline contents of the intestine, and here finds the conditions essential to its further development. It penetrates at first into the lymphatic follicles and Peyer's patches, and thence goes on into the mesenteric glands, the blood-current, the spleen, and other organs.

As in the case of most other infectious diseases, the occurrence of infection in typhoid is dependent not only on outward conditions, but also on an individual predisposition. Details of the circumstances attending this latter are as yet not at all accurately understood. Even in the worst typhoid centers, where the possibility of infection must be universal, many escape the disease.

Age has an indubitable influence upon the liability to the disease. Typhoid is especially a disease of youthful, vigorous individuals, of fifteen to thirty years. Above that age it is noticeably less frequent, although cases do occur at sixty and even seventy years. Formerly it was often said that young children were never attacked; but this was because the disease was not recognized, for in reality it is only children under one year old who seem to be seldom infected. At a later age, cases are by no means rare.

Sex can not be shown with certainty to have an especial predisposing influence upon the frequency of typhoid fever.

Mental excitement and gross errors in diet seem to predispose to the disease. On the other hand, a certain immunity has been alleged to be given by many circumstances, especially pregnancy, the puerperal state, and other diseases already existing (tuberculosis, heart disease). Most of these statements are shown, however, by more extended experience, to be very doubtful. It is certain, however, that the occurrence of typhoid fever gives very probable though not absolute immunity against any later new attack. Recent bacteriological investigations have suggested a reason for this immunity. Certain chemical albuminoid substances (protective substance or "alexine" of Buchner) probably remain in the blood-serum and fluids of the tissues and prevent a new infection.

Finally, it must be mentioned that the necessary conditions for an abundant development and conveyance of the typhoid germs are beyond doubt dependent on the season. According to statistics, most of the typhoid epidemics come in the months from August to November, while generally the number of cases greatly diminishes from December to spring.

General Course of the Disease.—Extended experience shows that, after infection with the typhoid poison has taken place, a certain time must elapse before the symptoms of the disease appear. The length of this time, the "stage of incubation," is, unlike that of many other infectious diseases, not perfectly definite. On the average, it lasts two to three weeks, sometimes less time, sometimes longer. During this period the patient either feels perfectly well, or has certain slight symptoms, to which he pays more or less attention, according to his individual susceptibility. These prodromata consist of languor, disinclination to exertion, anorexia, slight headache, pain in the limbs, etc. Often they last only a few days. Not infrequently the patients state afterward that they had felt the disease coming on for weeks.

The transition of the prodromata into the regular disease takes place sometimes so gradually that it is utterly impossible to take any one day as the first of the illness, in order to reckon from it its duration. It is usually, however, the first symptoms of a high temperature, chilliness, feverishness, and the accompanying increase in general discomfort, which allow one to fix, with at least some accuracy, the beginning of the disease. A decided initial rigor is certainly exceptional.* After the fever begins, most patients soon take to their beds, although it happens often enough that the sick feel either unable or unwilling to give up, and keep on at work for days!

There have been manifold attempts to divide the whole course of the disease into separate periods. The most natural division seems to be into the three stages of development, height or fastigium, and decline (*stadium incrementi, s. acmes, s. decrementi*). Usually, however, physicians reckon according to the week of the disease. The first week corresponds to the developmental stage, the second, and in all severer cases the third as well, to the fastigium, the fourth (in light cases the third) to the decline. The course of the disease is very variable, however, and naturally there is the greatest diversity in the departures from this general plan.

In the first week, the initial period, the general symptoms augment rapidly. The patients become, in severe cases, very languid and feeble, have generally an intense headache, and complete anorexia, with great thirst. The fever, which is all the time gradually rising, is recognizable subjectively by alternating sensations of heat and cold, and objectively by the hot, dry skin, the parched lips, and the dry and coated tongue. The sleep is disturbed. For the most part there are no prominent thoracic or abdominal symptoms, except that at times there is a sense of oppression in the chest, or some cough. The pulse is quickened, sometimes even now dicrotic. There is often a temporary epistaxis. The belly is not much swollen as a rule, and but little if at all tender. There is generally constipation. Usually the spleen, even at this time, exhibits a swelling that can be easily demonstrated.

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* According to the representations of many authors, a marked initial rigor seems to occur rather often in some places. In Leipsic, and also in Erlangen, a pronounced rigor at the beginning of typhoid is scarcely ever seen.

Generally the fastigium has begun before the end of the first week. The severe general symptoms persist or even increase. The fever maintains constantly a considerable elevation. The patients become more stupid. Often delirium appears, especially at night. In the lungs there is developed a more or less intense and extensive bronchitis. The abdomen becomes more swollen. On the skin of the trunk appear, generally at the beginning of the second week, a number of small, pale-red spots, roseolæ. Instead of constipation, there is a moderate diarrhœa. There are daily about two to four soft, thin, bright-yellow dejections.

The third week, during which in the severe cases the symptoms already mentioned persist, is the chief time of the numerous complications and of especial clinical events, about which we shall speak below at length. If the disease takes a favorable course, there comes at the end of the third week a decline of the fever; and then the general symptoms also improve as a rule. The mind becomes clearer, the patient sleeps better, and gains some appetite. The pulmonary and digestive symptoms abate, and convalescence gradually begins.

This short sketch of the course of the disease corresponds to most of the cases of medium severity. There are, however, besides these, so many forms and so many variations from the usual picture, that it seems almost impossible to enumerate completely all the events of typhoid fever. And besides, the separate epidemics vary in their general character according to the time and place of their occurrence. In many epidemics the cases run a peculiar course and have certain special complications not seen in others.

We will begin the presentation of the chief peculiarities by speaking of the course of the fever.

Course of the Fever.—Observation of the temperature in typhoid is so absolutely essential for the estimation of each individual case that no scientific physician ought to treat a case without regular measurement of the temperature. The measurements should be taken, if possible, in the rectum. Their frequency must of course be modified by circumstances, but it will probably be possible to have three or four measurements daily. At night, especially if the patients are asleep, it is generally not requisite to take the temperature. A general idea of the course of the fever can be gained only by representing the separate measurements graphically in a continuous "temperature curve."

The typical curve of typhoid fever (see Fig. 2) falls naturally into three or four divisions. The first division is the initial period, or the pyrogenetic stage, and is seldom observed, since at this time the patients are generally not yet under the doctor's care. The initial period of the fever lasts, as a rule, some three or four days, seldom longer; and during this time the temperature rises, generally by gradual steps, so that the morning as well as the evening temperature is each day 2° or 3° (1°–1.5° C.) higher than on the day before. A sudden and considerable rise of temperature, such as occurs in many other diseases, is very rarely seen in the beginning of typhoid fever.

The second division of the curve represents the so-called fastigium, and corresponds to the height of the disease. During this time the fever presents, in most of the severer cases, the general character of "*febris continua*"—i. e., the spontaneous remissions of the fever seldom exceed 2° (1° C.). Almost always the lower temperatures come in the morning hours and the higher in the evening. In cases of average severity the morning remissions touch 102°–103° (39°–39.5° C.), and the evening exacerbations 104°–105° (40°–40.5° C.). Temperatures which reach or exceed 106° (41° C.) are seen only in very severe cases. Considerable morning remissions are always a favorable symptom, while morning temperatures of 104° (40° C.) or higher generally show the case to be severe. The duration of the fastigium varies with the severity and obstinacy of the case. It may last only a few days or one and a half to two weeks; in violent cases still longer.

In many cases of slight or average severity the period of decline follows directly on the fastigium; but in severe cases there frequently intervenes another stage, which Wunderlich has graphically named the "ambiguous" period. The temperature-curve becomes irregular and more variable. The morning remissions may be great, even reaching the normal, while the evening temperatures are often still very high. This stage has accordingly been termed the "period of the steep curves." It may be said that in general the longer a case of typhoid lasts the more irregular will be the course of the fever.

The last stage—i. e., in cases of slight or average severity the third stage, and in severe cases commonly the fourth—is the period of defervescence or recovery. The peculiarity of this period in typhoid fever is that the fall of the fever is never by crisis, but always gradually, by lysis. Commonly the temperature descends by degrees, so that on each new day the morning remissions as well as the evening exacerbations are 1° to 2° (0.5°–1° C.) lower. The zigzag form of curve, in which there are of course very frequently slight irregularities, must be taken as the rule. The duration of the defervescence generally exceeds

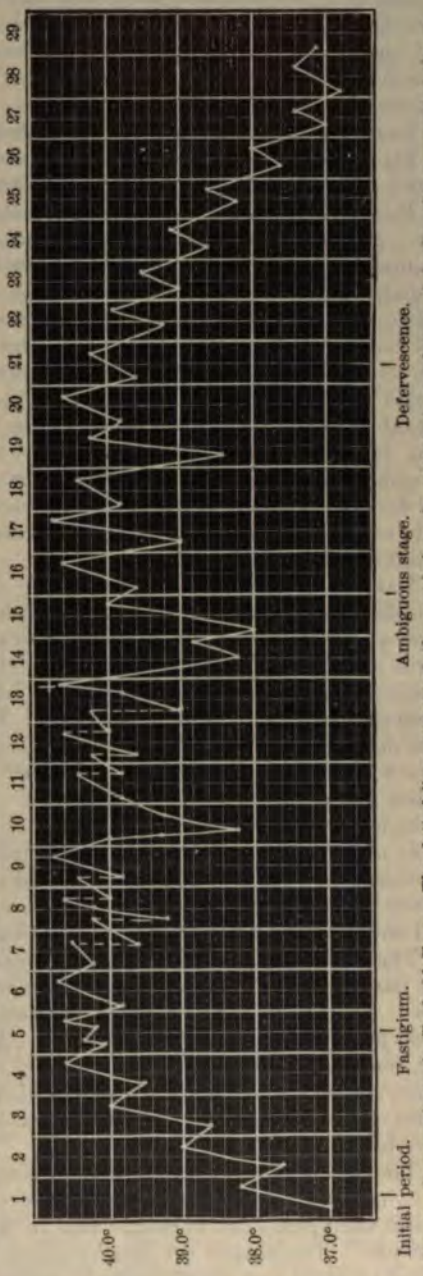

FIG. 2.—*Temperatures in Typhoid Fever.*—The dotted lines represent the remissions in temperature after cool baths, the crosses (+) the exhibition of antipyretics.

that of the initial period. It lasts five to eight days, often longer. It is not very seldom that in defervescence the morning remissions become from the first very marked, even reaching the normal temperature, while the evening exacerbations become daily less and less, until they too are not above the normal. A third form of decline is much less frequent, in which the morning remissions become every day greater, while the evening temperature persists for some days at about the same height. Several times we have seen the fever take on a tertian type during recovery.

To this outline must be added a number of observations of practical importance.

The initial period does not exhibit especial variations from the course we have stated. Its entire duration is bounded by certain relatively narrow limits.

The fastigium presents, as already mentioned, the greatest varieties in its duration. In light cases it is wholly wanting, so that these consist only of a period of gradually rising fever, and of a gradual defervescence almost immediately consecutive to the rise. The entire duration of such light cases is only one and a half to two weeks. In other and tolerably frequent cases, which are often tedious, but still for the most part are light, the fever is not continuous, but remittent. The difference between the morning and evening temperatures amounts to 3° or 4° F. (1.5°–2.0° C.), but the absolute height of the temperature is often not very considerable, so that the temperature curve at first leads to error and excites suspicion, for example, of tuberculosis. We have seen in Leipsic a number of cases in which the fever was perfectly intermittent during almost the entire illness, and for two to three weeks afternoon elevations reaching 104° (40° C.) or more daily succeeded normal morning temperatures. These cases had the general course of light attacks.

Various influences, not to speak of therapeutic interference, may produce a considerable temporary remission of temperature in the course of the fastigium. Such a remission sometimes occurs spontaneously from the seventh to tenth day of the disease. If a marked intestinal hæmorrhage happens (*vide infra*), the temperature generally falls several degrees centigrade, and the less frequent instances of severe epistaxis have the same effect. If, in female patients, abortion or premature delivery occurs, we often observe a similar considerable fall of temperature, even without severe attendant hæmorrhage. Perforation of the intestine often causes the temperature to fall rapidly. At times the occurrence of mental disturbances effects a moderate though noticeable lowering of temperature. Those great and sudden depressions of temperature remain to be mentioned which are accompanied by a very small but exceedingly rapid pulse and general prostration. Every such collapse, if severe, is a most dangerous event, and demands prompt and energetic medical treatment (*vide infra*).

The development of local complications, such as pneumonia or inflammation of the parotid gland, is generally accompanied by a considerable rise of temperature. The fever in such cases often becomes more irregular. This indicates the great practical value of thermometry. Almost every new rise of temperature or any considerable change in the ordinary course of the temperature has its special cause, and is therefore a warning to the attending physician to be vigilant. The cause of the change in the temperature is often not apparent until two or three days later.

The period of defervescence departs most frequently from its typical behavior by being lengthened out into a "stage of retardation." The morning temperature is then generally normal, while in the evening slight or moderate elevations continue. The reason for this long continuance of the fever may frequently be found in some not yet completely healed local complication, but often no such lesion can be demonstrated. Then we are commonly inclined to surmise sluggish intestinal ulcers which will not heal, or trouble in the mesenteric glands, etc.

This slow fever may continue for weeks. It is prone to follow severe cases, but lighter attacks, especially in elderly or feeble patients, may also take on this sluggish character at a relatively early period. To these last-mentioned cases must finally be added a few others in which during the whole course of typhoid fever no febrile temperature at all or only a very slight rise can be detected.

Entrance into complete convalescence is shown with far greater certainty by the absence of elevations of temperature than by any other single symptom. There sometimes come, however, temporary elevations of temperature during convalescence, following some error in diet, long-continued constipation, or mental excitement. In other cases the new fever depends on some local sequela, e. g., a boil or a glandular abscess. Often, however, the most accurate investigation fails to demonstrate a cause. Especially in the beginning of convalescence there sometimes comes a high fever, or even a rigor, which may recur several times, but which soon gives place to a normal temperature. Generally no definite cause for these brief but decided elevations of temperature can be pointed out. Perhaps we might consider the possibility of some affection of the mesenteric lymph-glands. These sudden and great elevations have seldom any grave significance.

This new fever which we have just described is best termed febrile recrudescence, or febrile sequela, in contrast with the proper typhoid relapse. That is, after typhoid fever has ended, the whole process may be repeated; and this occurrence is called a relapse. Particulars as to the behavior of the fever in such cases will be considered below, in connection with all the other peculiarities of typhoid relapses.

PHENOMENA AND COMPLICATIONS RELATING TO THE SEPARATE ORGANS *

Before we undertake a detailed discussion of the individual symptoms of typhoid fever, we must first make a brief general statement which is of very great significance for the correct understanding of almost every infectious disease. We include among the direct typhoidal symptoms all those morbid phenomena which are produced immediately by the typhoid bacillus itself or by its toxic chemical action; but, on the other hand, every patient sick with typhoid fever is exposed to many secondary infections (from the intestinal ulcers, from the mouth, in the lungs, etc.) whose invasion is rendered possible, or at least rendered easier, by the pre-existing typhoid infection. All the morbid symptoms arising in this way, which unite with the pure typhoidal symptoms in making up the whole picture of the disease, must, strictly speaking, be termed complications of typhoid. In the individual case it is often hard to decide whether a particular symptom is of a typhoidal nature or a complication. We must, however, hold fast to the essential difference between these two kinds of morbid phenomena, if we would obtain a deep insight into the nature and origin of the whole course of the disease.

1. **Digestive Organs.**—We think it best to begin our consideration of the more special symptoms with the phenomena referable to the intestinal canal, for the reason that the anatomical changes in the intestine are pathognomonic. Indeed, these alterations may sometimes become of surpassing import in a clinical point of view, although in the majority of cases the intestinal symptoms are clinically not nearly so prominent as the general symptoms that result from the infection of the system as a whole.

The characteristic typhoid lesion of the intestine consists of an affection of Peyer's patches, most marked in the lower part of the ileum. In the first week the patches swell gradually (stage of medullary infiltration). The rest of the mucous membrane exhibits at the same time more or less marked symptoms of simple catarrhal inflammation. In the second week, necrotic crusts form on the surface of the patches, which are cast off in the third week, leaving behind the typhoid ulcers. Toward the end of the third week the ulcers clean up, and then in the fourth week, if the case takes a favorable course, the ulcers heal. Smooth scars are formed, often diffusely pigmented. Experience shows that these scarcely ever lead to stricture of the intestine. The same process also goes on in a greater or less number of the solitary follicles as well as in the Peyer's patches themselves. We may add that probably in lighter cases of typhoid (*vide infra*) there is often no actual ulceration. The swelling of the lymphatic tissue subsides in this case before sloughing occurs. We have already mentioned the occurrence of typhoid bacilli in Peyer's patches and the intestinal follicles.

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* To avoid repetition, we have in what follows united a description of the anatomical changes with the presentation of the clinical symptoms.

The number and size of the ulcers formed have no direct relation whatever to the severity of the case. Although very extensive lesions in the intestine are often found in cases that end fatally, yet, on the other hand, we observe fatal cases in which only a few ulcers are found in the intestine. In cases with extensive intestinal lesions we often see follicular ulcers in the colon as well as in the small intestine (colo-typhoid).

The clinical symptoms referable to the intestinal canal are, as we have said, prominent only in exceptional cases. In the beginning of typhoid fever there is usually constipation. This may last throughout the illness, so that the patients have but one dejection in every two or three days, or often none at all unless an enema be given. As a rule, a moderate diarrhœa begins during the second week. There are two to four stools, or sometimes more, each day. These usually have a characteristic bright-yellow color. On standing, they divide into an upper, cloudy, and quite liquid layer, and a lower layer composed of yellow, crumbly masses. They have generally an alkaline reaction, and upon microscopic examination they are found to contain, besides remnants of the ingesta and granular detritus, a few epithelial cells, round cells, many crystals of triple phosphate, and numberless bacteria. Pfeiffer and other investigators have been able frequently, although not invariably, to demonstrate the true typhoid bacilli in the dejecta by means of special methods of cultivation.

Severe diarrhœa (ten or more stools daily) is relatively infrequent. In some bad cases we have seen the stools take on a dysenteric character. The autopsy showed in these cases unusually severe lesions of the colon and a diphtheritic inflammation of its mucous membrane. They were probably secondary complications.

Gaseous distention affecting the intestine, and especially the colon, is very frequent; a slight but evident gaseous distention, with a fluctuating, "air-cushion" feeling, is a very valuable symptom in the diagnosis of typhoid fever, but, as a rule, the meteorism is moderate. Indeed, severe cases of typhoid are observed in which the abdomen always remains concave. Marked tympanites is always an unpleasant complication. We saw one case, which ended fatally, with very great tympanites, in which the lesions were almost exclusively in the colon, and it was the enormous distention of its entire length which had so swollen the abdomen. The noise that can often be produced by pressure in the ileo-cæcal region (gurgling) used to be regarded, but probably erroneously, as especially characteristic of typhoid fever. Abdominal pain is often entirely absent. Some patients, however, complain of abdominal pain during almost the entire illness. On pressure, the belly is generally somewhat sensitive, but the tenderness is seldom extreme. It is more apt to be marked when there is constipation. Often such tenderness is due to a participation of the peritoneum in the disease, even when there is no perforation (*vide infra*).

There still remain two symptoms of the greatest practical importance, both of which have a direct connection with the intestinal lesions: they are intestinal hæmorrhage and perforation.

Intestinal hæmorrhages in the course of typhoid are almost always due to the erosion of the walls of blood-vessels in connection with the formation and throwing off of the crusts of the ulcers. The hæmorrhages occur, therefore, most frequently toward the end of the second and during the third week. The blood pours out into the intestine, and is passed with the stools. Its amount may be small, or it may reach to one or two pints, or even more. Its color is generally rather dark. The later discharges are generally tarry. Liebermeister states that he has observed intestinal hæmorrhages in 7.3 per cent. of typhoid patients, and Griesinger in 5.3 per cent. We have ourselves seen, in the medical wards in Leipsic, 45 intestinal hæmorrhages in 472 cases, i. e., in 9.5 per cent. In individual epidemics the frequency varies greatly. It rose in 1880 to eighteen per cent.

Intestinal hæmorrhage is always a grave symptom. Even slight hæmorrhages deserve consideration, for they may be the precursors of severer ones. Yet intestinal hæmorrhage, even if profuse, is not necessarily fatal. Of the above forty-five cases of typhoid with hæmorrhage, twenty-six ended in complete recovery. In eight cases, death occurred as the immediate result of the loss of blood. Eleven ended fatally after a time.

After every considerable intestinal hæmorrhage, the symptoms of general anæmia, often even of collapse, appear. The fall of the bodily temperature has been already mentioned. The hæmorrhage has sometimes a favorable influence on severe cerebral symptoms, for consciousness succeeds to the previous stupor or delirium. Often the hæmorrhage is directly followed by recovery from the disease.

Much more ominous than the intestinal hæmorrhage is the occurrence of perforation, as a result of the breaking through of a typhoid ulcer into the abdominal cavity, because, almost without exception, this is followed by a purulent or even ichorous peritonitis. Peritonitis is never caused by the typhoid bacillus itself, but by pyogenic organisms (cocci, perhaps bacterium coli?) which enter the abdominal cavity with the contents of the intestine. The amount of fluid peritonitic exudation is, as a rule, not very great. The serous membrane is often found covered merely with a fibrino-purulent or purulent-hæmorrhagic deposit. The occurrence of perforation is sometimes marked by a violent pain suddenly felt by the patient; but it may also, even in severe cases, take place insidiously. The abdomen is generally (not always) greatly distended and very tender on pressure, so that even in stupor patients groan while being examined. If gas has entered through the opening into the peritoneal cavity, we often observe absence of the ordinary dullness over the liver; but this symptom is to be employed cautiously as a factor in diagnosis, for absence of hepatic dullness may also result from distended intestines lying in front of the liver. When perforation has occurred, the patient soon looks collapsed, with cheeks fallen in and sharp, cool nose. Frequent eructations and vomiting often follow. The pulse becomes small and very frequent. The temperature generally falls as the peritonitis begins, and later it usually undergoes great variations.

Perforation of the intestine occurs most frequently in the third or fourth week of the disease, and much oftener in men than in women. In sluggish cases, however, we can not be without apprehensions of it till a late period. The perforation generally takes place in a coil of the lower part of the small intestine, and with marked relative frequency in the right side of the pelvis, though seldom in the vermiform appendix or in the colon. With few exceptions, death comes quickly, after a few days at latest. Only when the perforation is small and the intestines have become agglutinated at the onset is the course of the peritonitis slower, so that the symptoms are less violent and cause death in a week or ten days. Out of fifty-six fatal typhoid cases in the Leipsic medical clinique we lost five, or nine per cent., from peritonitis following perforation. Here and there a case of recovery has been reported, probably resulting from a limitation of the peritonitis through speedy adhesion of the intestines.

It should be mentioned here that sometimes in typhoid fever a local or general peritonitis may occur through direct extension of the process to the serous membrane without actual perforation. We have seen in one case, as the result of the peritonitic bands and false membranes, complete occlusion of the intestine (ileus), and death.

Swelling of the mesenteric lymph-glands (less often of the retro-peritoneal glands as well) is found in typhoid almost as constantly as the anatomical changes in the intestine. Sometimes they break down, i. e., suppurate. In cases that have passed through the disease we often find considerable deposits of lime in the glands. These changes have a certain clinical importance; for, as already mentioned, we may often venture to refer a more or less tedious recurrent febrile state which has no other demonstrable cause to this lesion of the mesenteric glands. In some rare cases a general peritonitis has been observed as a result of the bursting of a suppurating gland.

The swelling of the spleen (acute splenic tumor) is, in typhoid fever as well as in many other acute infectious diseases, one of the most constant symptoms. The enlargement of the spleen can often be demonstrated as early as the end of the first week, and is therefore of considerable diagnostic importance; but percussion of the spleen is sometimes decidedly difficult and deceptive in this disease because of the existence of tympanites. The surest demonstration of splenic enlargement is therefore always by means of palpation, which, after a little practice, gives a positive result in the majority of cases. Absence of splenic tumor is most frequently observed in elderly typhoid patients. The spleen may also diminish considerably in size after severe intestinal hæmorrhage. Pain in the splenic region, resulting from tearing of the distended capsule, is comparatively rare. The splenic infarctions which sometimes occur may, in exceptional cases, prove the starting-point of a peritonitis.

Hepatic symptoms are seldom seen in typhoid fever, except that there may be a moderate swelling of the organ. The anatomical changes of "parenchymatous degeneration," and the frequent formation in the liver of the small lymphomata which Wagner discovered, have no clinical significance. The bile secreted is generally pale and scanty. This is a partial explanation of the light color of the stools. A very rare complication, which we ourselves observed in one case, is acute yellow atrophy of the liver.

The stomach presents no especial anatomical changes in typhoid. Anorexia is an almost invariable symptom in the beginning and during the course of all severer cases. There is seldom any desire for food till recovery begins; but then, if convalescence is undisturbed, the appetite soon attains an enviable keenness. Vomiting in the beginning or course of the disease is an exception, unless after some error in diet. We have already mentioned it as a symptom of peritonitis.

The changes in the mouth and throat of typhoid patients deserve the careful attention of the physician. The lips and tongue are in severe cases dry and fissured. The lips are often covered with dry, black crusts, sometimes described as a "fuliginous coating." The tongue is apt to be thickly coated at first, but later cleans off from the edges and tip. In severe cases, especially if the mouth is not properly cleansed, a rather severe stomatitis may occur and produce superficial ulceration of the buccal mucous membrane and of the edges of the tongue. The gums sometimes become spongy, and are apt to bleed, as if scorbutic.

Actual sore throat, at least according to our experience in Leipsic, occurs but seldom at the beginning of typhoid fever. The difficulty in swallowing, often complained of by patients, is generally due to dryness of the pharynx. In certain epidemics, however, the occurrence of sore throat at the beginning of the illness has been frequently observed. It may even happen that this early sore throat is accompanied by an erythema diffused over the body, so that at first suspicions of scarlet fever arise. The cases are very interesting and quite rare (although we have repeatedly seen them) in which disturbances of swallowing exist with the general typhoid symptoms from the beginning. On examining the fauces we see on the tonsils peculiar white, slightly elevated spots, which later form superficial ulcers. After a time these places heal, and in other respects the disease pursues a normal course. The suspicion is justified that in these cases there is a specific typhoidal disease of the tonsils (due to the typhoid bacillus itself), and such cases are called tonsillar or pharyngeal typhoid (analogous to the laryngeal typhoid, pneumo-typhoid, and nephro-typhoid, to be mentioned later). In such cases the typhoid bacilli have probably attacked the tonsils at their first invasion. It should also be mentioned that in severe cases there is often an extensive growth of thrush in the mouth and throat, and this may spread quite a distance down the œsophagus.

The changes in the mouth and throat are of especial interest, for the reason that they may be directly propagated to important neighboring organs. Starting from the pharyngeal cavity, the pathogenic agent, probably in most cases the staphylococcus, may penetrate through the Eustachian tube into the middle ear. Thus arise those inflammations of the middle ear which are not very rare in severe cases of typhoid, and which lead to perforation of the membrana tympani and to purulent discharges from the ear. The not infrequent inflammation of the parotid gland is also, as we believe, occasioned in a similar way, the inflammatory agent reaching the parotid gland from the mouth by way of Steno's duct. We do not regard the otitis and parotitis as especial localizations of the typhoid poison, but as secondary diseases, for the occurrence of which typhoid fever merely furnishes the occasion, as when the mouth is imperfectly cleansed. Purulent otitis in typhoid fever can easily be overlooked at first, since stuporous patients only rarely complain, of themselves, of pain in the ear or deafness. The parotitis appears most frequently in the third week, and generally on one side, though sometimes on both. It almost always becomes purulent, and discharges either externally or into the external auditory meatus, unless there is a timely incision.

2. **Organs of Respiration.**—Affections of the lungs are among the most frequent and important complications of typhoid fever, but are for the most part not a direct result of the typhoid infection, but pure complications. The bronchitis very often found in severe cases, and especially in patients who do not come till late under proper care, certainly is dependent on the imperfect expectoration of the bronchial secretions and on the inhalation of inflammatory agents coming from the mouth and throat.

Numerous cases of typhoid of slight or average severity, under proper care, run their course without any considerable bronchitis. In many other cases, and even severe ones, the bronchitis remains within moderate bounds, especially if the patient is brought promptly under proper care and treatment; but in severe cases, when marked disturbances of the nervous system arise, and the patient in his stupor expectorates little, swallows things the wrong way, and lies all the time on his back, passive and collapsed, the occurrence of a severe, diffuse bronchitis, especially in the lower lobes of the lungs, can hardly be avoided. Nor in such cases is there generally a mere bronchitis, but a more or less extensive catarrhal, lobular pneumonia, to be classed therefore under the so-called inhalation pneumonias (cf. chapter on lobular pneumonia). What was formerly termed " hypostatic pneumonia " is also almost invariably to be put in this group.

From the way in which these pulmonary disorders arise, we can understand why the bronchitis sometimes takes on a putrid character, and why the lobular infiltrations are, in severe cases, transformed into genuine gangrene. If such spots touch the pleura, they occasion the development of a pleurisy which is almost always purulent. In rare cases, pneumothorax may arise as a sequel to the perforation of a gangrenous infiltration into the pleural cavity. Various circumstances promote the occurrence of pulmonary symptoms. Thus we find it especially easy for a severe bronchitis and its sequelæ to be developed, in the case of elderly persons, or the kyphoskoliotic, or the corpulent, or patients who have previously suffered from emphysema or cardiac disease.

The subjective thoracic symptoms, in typhoid patients who have pulmonary complications, are generally not very prominent. It is only occasionally that patients complain in the early stages of typhoid fever of pain, and of a sense of oppression in the chest, or of cough, or of a stitch in the side; and even when such symptoms exist, the physical examination may give comparatively insignificant results. The severer pulmonary complications are seen mainly in those whose intelligence is more or less blunted, and who, therefore, make little complaint, are not much disturbed by the dyspnœa, and cough and expectorate little. A careful physical examination alone can enlighten us as to their condition. On auscultation, sibilant rhonchi are the chief signs observed in the milder cases. In the severer ones there are moist, fine, and coarse râles, especially numerous toward the base of the chest. If there are abundant moist râles, we may infer that there is a lobular pneumonia, although this can not be demonstrated with certainty till the separate islets of infiltration unite into a more extensive solidification, so as to afford dullness on percussion.

In addition to the pulmonary lesions already mentioned, genuine croupous or lobar pneumonia does occur in typhoid fever. This is often an actual complication, dependent upon a secondary infection with the genuine pneumonia diplococcus. Such a pneumonia may come on early or sometimes during convalescence, and affect both the upper and lower lobes. There is probably also a true typhoid pneumonia caused by the entrance of typhoid bacilli into the lungs. Such a pneumonia can not at present be recognized anatomically, but only by bacteriological investigation. Especial interest attaches to those cases of typhoid fever which begin with a lobar pneumonia. Often there is at first not the slightest suspicion of a typhoid fever, for the disease is regarded as an ordinary croupous pneumonia; but it is usually to be noticed that the illness does not begin suddenly with a rigor, but more gradually, and that from its incipiency the constitutional symptoms, the headache and splenic tumor, are more prominent than is usually the case in pneumonia. At the end of the first week's illness there is no crisis, but persistent fever. Now the pulmonary symptoms often retreat more and more to the background, while, on the contrary, diarrhœa and rose-spots appear. The spleen is enlarged. In short, the clinical picture of typhoid is developed. It is not unnatural to suppose, although there is yet no absolute proof of the fact, that in these cases, which are fittingly termed "pneumotyphoid," the infection with the typhoid bacilli has taken place exceptionally in the pulmonary tissue, and that, therefore, the first pathological changes are developed in the lungs.

Laryngeal Lesions.—The same causes which produce the bronchitis result also in a simple catarrhal laryngitis, with hoarseness. This is in severe cases accompanied by superficial ulcers on the vocal cords or the posterior wall of the larynx. Sometimes, again, the lesion is due to mechanical causes, constituting the so-called "*decubitus laryngis.*" The disorders which attack the less superficial structures of the larynx are fortunately rare. Chief among them is a laryngeal perichondritis of the arytenoid cartilages. This complication is justly regarded as of bad omen, and may lead to the rapid development of œdema of the glottis, with great laryngeal obstruction and threatening suffocation. These severe laryngeal affections in typhoid are regarded by some authorities, especially by Klebs, as always the direct effect of the infecting poison; but in most cases they are probably due to an invasion of staphylococci or some similar microbes. We have several times seen croup in typhoid fever, and it is a very dangerous symptom. In regard to their origin most of these mild and severe laryngeal affections in the course of typhoid fever are to be regarded as secondary inflammations; but specific typhoid disease of the larynx also seems to exist. The cases are of interest where the whole morbid process begins with a severe laryngitis (" laryngeal typhoid ") and is followed later by the ordinary symptoms of typhoid (*vide supra*, pharyngeal typhoid).

Among symptoms referable to the mucous membrane of the nose, epistaxis is important. It occurs in the beginning of typhoid with tolerable frequency, and is in one way not unfavorable, for it often mitigates the patient's headache. At a later period nose-bleed may become a most unpleasant complication, as it is sometimes very difficult to check. We have even seen one fatal case due to persistent nose-bleed. Other nasal symptoms are exceptional. There is an old saying that typhoid never begins with a coryza.

3. **Nervous System.**—The old term " nervous fever," which is still used by the laity, shows how frequent and severe are the nervous derangements which occur in typhoid. In cases of any severity there is almost always a certain dullness of intellect, often amounting to apathy and somnolence. The patients give monosyllabic and incomplete answers to all questions, and their statements about their previous history are often disordered and contradictory. There may even be sopor or a deep coma in the worst cases. All cases of this sort in which there was a condition of intellectual enfeeblement were termed by the old physicians "*febris nervosa stupida*," in contrast to the "*febris nervosa versatilis*," that form in which abnormal mental activity or delirium predominates. In severe cases delirium is very frequent. It is generally worse at night, and at times when the patient happens to be left alone. Very often he tries to leave his bed, because of his delusions, and talks of persons and things with which he was formerly familiar; or he is very noisy and restless, sometimes shrieking from groundless fears. We may add that these diverse nervous symptoms frequently succeed one another, or appear in combination. Sometimes a soporose patient may be heard softly whispering to himself in " muttering delirium."

Certain motor disturbances are often combined with considerable impairment of consciousness. There is a slight twitching of the muscles of the face and extremities. The old authorities gave the name *subsultus tendinum* to the sudden leaping into prominence of the sinews thus caused. It is best seen on the back of the hands. In severe cases the patient is sometimes observed to grind the teeth together; this is due to a cramp-like condition of the muscles of mastication, and is justly regarded as ominous. We often see persistent tremor of the extremities and lower jaw; and it is especially in these cases, as we have demonstrated upon numerous patients, that the tendon reflexes and the mechanical excitability of the muscles are much increased. If deep coma comes on, the muscles become lax, the motions of the eye are not co-ordinated, and reflex excitability diminishes, or is wholly extinguished.

Headache is one of the most constant symptoms in the beginning of the disease. It is usually referred to the forehead or temples. The pain may be very violent, and sometimes takes on almost a neuralgic character. It almost always subsides in the second week.

If we seek the cause of these nervous symptoms, which are often so severe, we find that the anatomical changes in the nervous system, including the brain, bear no relation whatever to the severity of the symptoms observed during life. We sometimes meet with minute hæmorrhages in the cerebral meninges, or meningeal opacity or œdema, or a moist condition of the cerebral parenchyma; but the connection of these and similar changes with the symptoms of the disease is often more than doubtful. Nor can the microscopic alterations in the brain, which have been reported, be regarded as important and authoritative. It is only in very rare cases that large cerebral hæmorrhages or purulent meningitis have been found. As to this last, we should always be very cautious in making a diagnosis, as symptoms which would seem to be most conclusively meningeal—such as stiffness of the neck, rigidity of the whole spinal column, and occipital headache—may appear in typhoid patients, and yet the autopsy may show no trace of meningitis.

One theory, which has Liebermeister for its chief supporter, and which has won a tolerably widespread acceptance among physicians, is that the nervous symptoms are chiefly a direct result of the febrile temperature. It is impossible, however, for us to regard this view as universally true. The unprejudiced consideration of a large number of personal observations prevents it. Although it is undeniable that elevated temperature has a harmful influence on the nervous system, yet in numerous cases there is no relation between the height of the fever and the severity of the nervous derangements. There are cases in which the fever remains continuously high for days, while the patient feels perfectly comfortable and presents no symptoms of any important cerebral disturbance. The opposite class of cases is still more numerous, in which from the very start there is always a low temperature, and, notwithstanding, the most severe nervous symptoms arise. Fräntzel and others have published very striking cases of this sort.

Hence we must seek for some other special cause of the severe nervous symptoms, and according to our present views this cause must be the intoxication resulting from the specific infection. We know that all bacteria produce, by their own tissue-metamorphosis and the chemical processes which they excite in their neighborhood, certain chemical matters which, especially in the case of the so-called "pathogenic bacteria," seem to be similar to the alkaloids ("ptomaines" and "toxines"), and exercise a decidedly poisonous influence upon the body, and especially upon the nervous system. These products are formed by the typhoid bacilli, enter the blood, and are the chief cause of the nervous phenomena. The difference in the violence of the latter in different cases probably depends mainly on a difference in the amount, and perhaps also in the quality, of the toxines produced by the typhoid bacilli, and probably also in the different susceptibility of individuals to the poison. The reason that the influence of these poisons is not much greater than it is, is that they are in part destroyed within the body and in part excreted with great rapidity, the channel of exit being mainly the kidneys. Thus is explained the interesting fact observed by Lépine, Bouchard, and others, that the urine of typhoid patients possesses poisonous properties not present in normal urine.

That the appearance of the nervous symptoms is dependent not only on the material causes, but also on the susceptibility of the individual, is shown by the fact that certain patients are especially prone to exhibit marked nervous phenomena; for example, hard drinkers, "nervous" individuals, and also those who have suffered violent emotional disturbances shortly before the onset of the disease.

Actual insanity is not very infrequent during the course of typhoid, or in convalescence. It generally takes the form of melancholia. We have repeatedly seen patients in such a state that they would lie almost motionless in bed, with eyes open, and perhaps assert that they were dead! In other cases there is mental excitement, sometimes combined with hallucinations, or there is confusion of ideas. In one case, in a girl who was evidently predisposed to nervous disorders, we saw typical hysterical insanity break out during the fever. Sometimes the mental excitement at the beginning of a relapse terminates in actual insanity. Few of the psychoses which arise during or at the end of typhoid outlast convalescence.

We have still to mention a number of nervous diseases that develop in the course of typhoid or after its decline. Neuralgia is sometimes seen, as well at the beginning as at the end of the disease. It is most frequent in the regions supplied by the trigeminus and the occipital nerves. Great hyperæsthesia of the skin and muscles is not rare during convalescence. It attacks the lower extremities by preference. Paralysis of single muscles (e. g., of the serratus magnus), or paralysis of a single extremity, has been repeatedly observed as a sequela. The paralysis is generally of the atrophic variety, and is probably, as a rule, due to neuritis. Ataxia and spastic paralysis of the lower extremities are rare sequelæ. Finally, there are sometimes developed, either in the course or at the conclusion of typhoid fever, the symptoms of a localized cerebral disorder (e. g., hemiplegia and aphasia), the anatomical cause of which varies. There may be a hæmorrhage or an embolism, and probably in still other cases a localized encephalitis.

4. **Circulatory System.**—Disturbances of the heart such as to produce striking anatomical changes are rare. The pericardium macroscopically almost always appears normal; the slight mitral or aortic endocarditis sometimes found has no clinical significance. The disturbances in the cardiac muscle seem more important. It is often more flabby than normal. The cavities, especially on the right side, are often dilated. With the naked eye we frequently see in the muscle itself cloudiness or fatty changes. Microscopic lesions are usually present and are much more pronounced. They consists usually of a granular (" albuminoid ") or more rarely of a fatty or hyaline degeneration of the fibers, and of genuine inflammatory foci of interstitial myocarditis (Hayem, Romberg). No marked disease of the cardiac ganglia has as yet been found.

The clinical significance of these changes should not, we believe, be overestimated. In all probability they are often without serious consequences and disappear completely with recovery from the disease. Sudden death (heart-failure?) occurs in typhoid, but it is very rare (see the chapter on diphtheria). Permanent disturbances of the heart after typhoid are also rare. When they occur they are perhaps due to the passage from an acute myocarditis to a chronic interstitial degeneration. [This slowness of the pulse as compared with the fever is common enough to have some diagnostic value, provided brain trouble is excluded. The pulse is almost always rapid, although often not so much so as the height of the temperature might lead one to expect.—V.]

It averages from 90 to 110, and often more. When it keeps at 140 or higher, in adults, it is always an unfavorable symptom. The abnormal frequency is often due in part to the high temperature; but there are other factors. Temperature and pulse do not correspond in all cases. Sometimes the pulse will have a normal or even subnormal frequency throughout the entire attack, despite the fever. Temporary accelerations are easily produced by mental excitement or bodily exertion, as by sitting up in bed. In convalescence the rate is frequently subnormal.

Slight irregularities of the pulse are not rare, either in the acme or the decline of typhoid. Marked irregularity is always a grave symptom, although in many cases it passes off and the patient recovers.

Dicrotism, due to loss of tension in the wall of the artery, is so common that it is still regarded by elderly physicians as characteristic of typhoid, although it often occurs in the same way in other acute diseases. In many severe cases the height and strength of the pulse may cause no alarm, but often the pulse is notably weak and small. This is due not only to the influence of the disease, but also to the previous condition of the individual.

The diminished cardiac activity may result in venous thrombosis, especially in the lower extremities and in a crural vein. This sometimes causes swelling of one of the lower extremities during the later stages or convalescence. The swollen member generally regains its normal size after some weeks. In other cases the thrombosis occurs earlier, and in patients who are still too vigorous to suffer from cardiac weakness, so that we are forced to the conclusion that there is some local specific cause, a local thrombo-phlebitis due to the typhoid bacillus itself, or, more probably, to the invasion of the walls of the vein by some secondary infection. A possible, but fortunately infrequent, result of these thrombi in the lower limbs is pulmonary embolism and sudden death.

In severe cases, which end in death, cardiac thrombi are sometimes found, with emboli in the lungs, spleen, kidneys, or other organs.

Œdema of the ankles and legs is very often seen in convalescents, especially when they first get out of bed. It is due to the weakness of the heart and changes in the vascular walls. Once we saw a general dropsy develop at the end of a severe attack in a girl of fourteen. The autopsy disclosed no other possible cause for it than the extreme atrophy and flabbiness of the heart.

5. **Blood.**—As in most febrile diseases associated with great emaciation, so in typhoid in severe cases the number of red blood-corpuscles (and correspondingly the amount of hæmoglobin in the blood) is much diminished. We found, for example, 2,800,000 to 3,200,000 in a cubic millimetre. In milder cases the figures do not differ materially from normal. It is a fact of greater interest and also of distinct diagnostic importance that many observers have found that leucocytosis is absent in typhoid fever, contrary to what is found in many other acute febrile diseases, pneumonia, erysipelas, sepsis, etc. We have often confirmed this fact in our clinique. We sometimes find with the general emaciation a decrease in leucocytes; for example, only 3,000 or 5,000 in a cubic millimetre instead of the normal number of about 8,000.

6. **Skin.**—The eruption seen in typhoid fever is characteristic and extremely important in diagnosis. The roseolæ or rose-spots (light red, slightly elevated spots) appear at the beginning of the second week, usually on the trunk, and chiefly on the abdomen and back. The number varies greatly. Rarely they are entirely absent, most often in elderly persons. Sometimes they are very abundant, and extend to the thighs, the arms, and even to the neck and face. Often they vanish after a few days, but they may persist much longer. In the latter case they may become to a very slight degree petechial, so that they will not entirely disappear on pressure. They often occur in successive crops. We have even seen several cases where new rose-spots kept coming for some days after the fever had disappeared.

As to other cutaneous eruptions, we may mention first of all that herpes labialis is so rare in typhoid that in cases of doubtful diagnosis it is a factor in excluding that disease. Miliaria, urticaria, and superficial pustules are sometimes observed. Occasionally little bluish spots appear, especially on the trunk. These used to be called "*taches bleuâtres*" (pelioma typhosum); but later observations show that they are not connected with typhoid fever particularly. They are due to pediculi. We might use the term pelioma typhosum to designate the kind of vesicles which we have repeatedly seen on the abdomen in severe cases. They are about the size of peas, and have sero-hæmorrhagic contents. Boils and superficial abscesses are frequent, especially as disagreeable sequelæ in convalescence from severe attacks. There are often abscesses of the sweat-glands in the skin of the axilla during convalescence. All these and similar cases of suppuration in typhoid fever do not depend, as a rule, upon the original cause of the disease, but upon secondary pathogenic germs, especially the staphylococcus, for whose entrance the typhoid process has merely prepared the way. Extensive ecchymoses are very rare, and are symptomatic of a general hæmorrhagic diathesis. Petechiæ are frequent during recovery. They are generally seen in the follicles of the skin below the knee. There have been a few cases of gangrene in the lower extremities, especially in the toes. We saw in one patient an extensive gangrene of the skin of the abdomen. Its cause could not be determined (ice-bag?).

Finally, we must mention that bed-sores are prone to develop in severe or neglected cases. The localities most often attacked are the nates, the furrow between them, and the heels. A bed-sore may be so extensive, and accompanied by such undermining of the skin, as to be a dangerous or even fatal complication.

The epidermis often scales off to a considerable extent during convalescence after a severe attack of typhoid. Everybody knows how the hair falls out after the fever, but it is sure to grow again. The nails also are not infrequently affected, becoming rough and brittle, or even falling off.

7. **Muscles, Bones, Joints.**—Zenker has discovered a degeneration of the voluntary muscles which occurs in typhoid as well as in other severe diseases. It is called the "granular" or "waxy" degeneration. Whether it has clinical symptoms can not be determined. Perhaps it may explain the great muscular hyperæsthesia which is often observed, and the muscular pains, which may be very trying. Severe cases sometimes have hæmorrhages into the muscles, particularly the rectus abdominis.

Lesions of the bones and joints occur but seldom, although other observers and we ourselves have repeatedly seen periostitis and ostitis in the ribs, tibia, etc., following typhoid. It is an interesting fact that lately typhoid bacilli have often been found in such foci of disease, so that it is a true typhoid localization. Swelling of the joints is rare. When of a purulent nature it is usually due to secondary "septic" infection.

8. **Genito-urinary Apparatus.**—Genuine, acute, hæmorrhagic nephritis is a very rare complication. It does occur, however, and has even given rise to the establishment of a special "renal form of typhoid fever" (nephro-typhoid). This name applies especially to those cases in which a severe acute nephritis is the predominant symptom at the start, while at a later period the course of the fever, the intestinal symptoms, the rose-spots, etc., show the disease to be typhoid fever. Nephro-typhoid is analogous to pneumo-typhoid and tonsillo-typhoid. A simple so-called febrile albuminuria occurs very frequently at the acme of typhoid, and is not to be interpreted unfavorably. It is probably the result of that slight parenchymatous degeneration of the kidneys which occurs in typhoid with the same frequency as in most of the other severe infectious diseases. There does not seem to be a direct relation between the albuminuria and the fever, although some authors assume it to exist. It is more likely that the renal epithelium is injured by the noxious products which have been formed in the body and excreted by the kidneys. In other respects the urine in typhoid presents the same peculiarities as in most other severe febrile diseases: its amount is diminished; its color dark; its specific gravity increased; the excretion of urea greater than normal. It should be added that the urine at the height of the disease presents Ehrlich's "diazo-reaction" in almost all cases.* Cystitis is not a rare development toward the end of the illness. It is probably always secondary.

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* Ehrlich's test is carried out as follows: Solution 1 consists of strong hydrochloric acid, one part mixed with twenty parts of a one-half-per-cent. solution of sulphanilic acid. Solution 2 is a one-half-per-cent. solution of sodic nitrite in water.

To 250 c. c. of solution 1 is added 6 c. c. of solution 2; an equal volume of urine is then added, and, lastly, about 60 c. c. of ammonia. On shaking, the urine becomes crimson.

In men, orchitis is sometimes observed. Women often have their catamenia at the beginning of typhoid. Later in the course of the disease, and in convalescence from severe attacks, the menses are often absent for several periods. In pregnant women there is considerable danger of abortion or miscarriage.

Peculiarities in the Course of the Disease

The above statements show an almost inexhaustible variety in the possible symptoms and complications of typhoid. The course of the disease as a whole may likewise present many diverse forms and peculiarities. We shall attempt merely to cite the most essential.

The numerous light and rudimentary attacks (*typhus levissimus*) are first to be mentioned. It was not recognized till lately that they belonged to typhoid fever at all (Griesinger). They used to have all sorts of names applied to them, the favorite term being "gastric fever." This light form lasts eight to fourteen days. The fever is moderate and often decidedly remittent. There is almost no proper fastigium. The typhoid symptoms are but slightly developed. There is no severe pulmonary or cerebral disturbance. There is generally a moderate diarrhœa, the spleen is plainly enlarged, and often rose-spots can be found. The diagnosis of these cases is of course difficult in proportion to the scanty development of typhoid symptoms. It is best established by demonstrating an ætiological relation between these cases and others which are plainly typhoid fever.

Abortive typhoid is justly distinguished by Liebermeister from *typhus levis*. The name belongs to cases which begin with severe symptoms and high fever, as if they were going to be grave, but in which these violent symptoms disappear after a few days and give place to a rapid convalescence.

On the other hand, there are cases which for a long time cause so little subjective discomfort that the patient does not even go to bed (walking typhoid). It is not till quite late that there occurs a sudden change for the worse, or some severe complication. Thus it has happened that persons who were apparently healthy have suddenly had all the symptoms of a severe peritonitis due to perforation and have died, the autopsy disclosing the lesions of the third week of typhoid fever; or a mild typhoid assumes the walking type and is not detected, but later a severe or dangerous relapse may occur.

The individual circumstances are very important in weighing each case, for they may modify the disease in many ways.

In children it is a remarkable fact that typhoid ulcers are much less frequent than in adults. This explains why intestinal hæmorrhages and peritonitis are much rarer in children. Marked cerebral symptoms are, on the other hand, very frequent. In severe cases children sometimes exhibit the peculiar symptom of a continuous penetrating screaming. In other, mild, cases the children are soporose.

In the aged the diagnosis of typhoid is often very difficult, since the course of the disease is frequently irregular. Generally the fever is not very high, and it very seldom exhibits distinctly the type described above. The pulmonary or cerebral symptoms predominate as a rule.

In the corpulent, typhoid fever is often very severe, so that our prognosis must always be rather grave, especially if pulmonary symptoms arise.

Hard drinkers are also in especial peril in this as in all other acute diseases. Dangerous cardiac weakness is prone to appear. Severe cerebral symptoms are frequent. It is, however, surprising that true delirium tremens is relatively infrequent, although so common in pneumonia.

The influence of previous strong mental excitement and of certain already existing diseases (cardiac disease, emphysema, kyphoskoliosis, etc.) has been already mentioned. Finally, we repeat that often the different epidemics present certain peculiarities. For instance, in one the type of the disease will be severe, in another mild. In one epidemic relapses are comparatively frequent, in another exceptional. The same is true with regard to the frequency of the appearance of certain symptoms, such as intestinal hæmorrhage, perforation, pneumonia, or nephritis. Indeed, it has even been observed that those cases which occur during a given epidemic in the same family or house or block sometimes present striking resemblances to one another ("group typhoid" of E. Wagner and others).

Relapses of Typhoid Fever

Typhoid fever exhibits in many cases the peculiarity of repeating itself completely after having run its entire course and disappeared. This process is called a relapse. It is in all probability the result, not of a fresh infection of the system from without, but of a renewed development of the germs already present, or possibly of a fresh auto-infection. A typical relapse is like a first attack in all clinical and anatomical particulars, with this difference that everything is more condensed, and lasts a shorter time than in the first attack. The interval between the two, during which there is no fever, lasts seven to ten days. It may be longer, and is often shorter. Sometimes the relapse follows immediately upon recovery. Indeed, it may even happen that, before the patient has completely recovered, his temperature begins to rise again in the characteristic step-like way. To such cases as this last the term recrudescence is applied. Except in the time of its beginning, it may be just the same as a genuine relapse. In the interval between the two attacks many persons are perfectly comfortable, and appear to be fully convalescent. There is often, however, a slight evening rise of temperature. It is noticeable that the splenic tumor does not completely disappear after the first attack in many cases which are followed by a relapse.

The relapse is generally briefer, as we have said, than the first attack, seldom lasting more than fifteen to eighteen days. The temperature rises more rapidly, perhaps in two or three days. The fastigium is shorter, the decline more abrupt. The absolute height of the temperature may be very considerable, even exceeding that in the first attack. Rose-spots appear as soon as the third or fourth day. The stools become liquid, the spleen enlarges again, and all sorts of complications may arise. The danger occasioned by a relapse may, however, be overestimated. On the whole, a relapse is not so very dangerous, and it is especially noticeable that the subjective symptoms, such as headache, are often slight. A severe relapse may follow a mild case. In other instances the relapse may prove merely rudimentary.

The frequency of relapses varies considerably in different epidemics. In Leipsic, we had relapses in about nine per cent. of all cases, but in separate years the percentage varied between four and sixteen. Out of about five hundred cases we have seen three in which there were two successive and typical relapses.

Diagnosis.—The diagnosis of typhoid fever may be perfectly easy, but, if the case be anomalous, or come under observation at a late period, it may be extremely obscure. Inasmuch as a search for the specific bacilli is too difficult and troublesome for the practicing physician, the diagnosis of the disease must be made from its course and symptoms. Important factors are the gradual onset, then the height and course of the fever, with no demonstrable localized disease, and the rose-spots. Less characteristic, but still of value, are the stools, the tympanites, and the swelling of the spleen. Ætiological factors, such as the occurrence of undoubted cases of typhoid in the neighborhood, are of great diagnostic value in obscure cases. Sometimes the diagnosis can not be established till the appearance of certain symptoms, like intestinal hæmorrhage, a characteristic mode of convalescence—viz., by lysis—or a relapse. It is an important rule not to make a diagnosis of typhoid after a single examination. It is generally necessary to observe the case accurately for several days before the diagnosis can be established. The differential diagnosis from other acute diseases, such as miliary tuberculosis, acute endocarditis, meningitis, etc., will be considered in discussing these diseases.

The diagnosis is usually most difficult in the cases with an imperfect history which are first seen in a severe "typhoidal" state, with high fever, disturbances of consciousness, etc. These are the cases in which we must also consider, beside typhoid fever, miliary tuberculosis, acute septic or pyæmic infection (including acute malignant endocarditis), meningitis, severe "typhoid pneumonia," etc. The accurate differential diagnosis between typhoid and these conditions will be spoken of later. We may state here that in such cases a careful examination of the blood, especially the absence of leucocytosis in typhoid, may afford very valuable diagnostic criteria. The ordinary diagnostic features are also to be considered, such as the way the disease began, so far as can be learned, the ætiological conditions (i. e., the connection with other cases of typhoid), rose-spots, thin pea-colored stools, meteorism, intestinal hæmorrhage, etc.

Widal has lately reported a method for diagnosticating typhoid (Widal's serum reaction), which is very interesting theoretically, but which, of course, can be used only in cliniques and hospitals. It is based on previous researches by Gruber and others. If we mix ten or fifteen drops of a fresh culture of typhoid bacilli with a drop of blood-serum from a typhoid patient, the bacilli lose their mobility in a few minutes, or at most in a quarter to half an hour, and come together in little clumps ("agglutinating" action of the serum). If we add blood-serum from a typhoid patient to a uniformly cloudy mixture of typhoid bacilli in bouillon, the bacilli again clump together and sink to the bottom, while the fluid becomes perfectly clear. Since the blood-serum of healthy persons, or those not suffering from typhoid, does not give this reaction at all, or only in a slight degree, a very positive result from this test is quite a sure diagnostic sign. The reaction often occurs in typhoid even in the first weeks of the disease, but it may be delayed. It persists long after the disease has subsided. Widal's reaction can also be obtained with serum from a blister or from serous inflammatory exudation in a typhoid patient. In otherwise doubtful cases a positive result is of definite diagnostic significance.

Prognosis.—A perfectly favorable prognosis should never be made. Cases which seem the mildest may become dangerous (from intestinal perforation, etc.). Yet, if there are good nursing and good treatment, typhoid fever is not a particularly dangerous disease, and we may hope for recovery even in very severe attacks. The danger lies, first, in the severity of the infection, as shown chiefly (though not wholly) by the height of the fever and the intensity of the general symptoms. A further danger is the appearance of the complications already enumerated and discussed. Thirdly, the constitution and condition of the individual are important. The circumstances coming under this head have likewise been repeatedly mentioned above. All these factors must be carefully estimated before we decide as to the danger in each case and make our prognosis.

The mortality in typhoid varies greatly in the separate epidemics. The severe cases are undoubtedly more frequent at some times than at others. This renders it difficult to give statistics which are universally applicable. We may in general reckon on an average mortality of about ten per cent., and measure the severity of separate epidemics by this standard. Numerous observers agree that the treatment now in vogue has decidedly diminished the mortality. It was formerly not rare for it to reach twenty or twenty-five per cent.

Treatment.—A specific cure for typhoid—i. e., some remedy to destroy the specific cause of the disease within the system, or to render it harmless—is as yet unknown. The different remedies recommended for this purpose (carbolic acid, iodine, thalline, ergotine, naphthaline, etc.) have none of them proved their value, and we can not advise further trial of them. The interesting experiments lately reported of injecting sterilized pure cultures of typhoid bacilli or the *bacillus pyocyaneus* into the patient (Rumpf and Fränkel) has as yet led to no definite practical result. At present there is only one remedy, whose efficiency, of course, should in no way be overrated, which we would be unwilling wholly to give up in treating typhoid fever, and that is calomel. Wunderlich noticed that if a few rather large doses of calomel be given at the beginning of the disease, it will on the average run a lighter and more favorable course than otherwise would have been the case. Wunderlich believed that typhoid fever may sometimes be aborted by this method. Although we can hardly expect this, it is really an efficient means of procedure, which we have almost always employed, to give two or three powders of calomel, of five grains (0.30 gramme) each, as the first prescription, to patients who come under treatment in the first week or the beginning of the second. As there is generally constipation, the laxative effect is also beneficial. Moreover, it often lowers the temperature somewhat. A moderate diarrhœa is not a contra-indication, but, if the bowels be very loose, the calomel should be omitted.

In other respects the treatment of typhoid must still be chiefly dietetic and symptomatic, and in one sense prophylactic. We must fight the symptoms already present, and further seek, as far as possible, to defend the patient from the attack of certain dangerous secondary disorders. Starting out with this view, the proper treatment of typhoid fever is a task of the highest importance, and by no means a thankless one.

We will begin by considering the general treatment. The sick-room must not be too warm, and must be frequently and thoroughly aired. The sick-bed must be well cared for. If effort be made to prevent bed-sores, we shall obviate one source of pain and danger (*vide supra*), and save ourselves and the nurse much trouble. Those who are very ill should therefore be laid on an air-cushion, or, if possible, a water-bed. The patient should be told not to lie always upon his back, but to change now and then upon his side. The back, the region of the sacrum, and the heels are to be often bathed with spirits of camphor or brandy. The minutest bed-sore is to be treated carefully. It should be cleansed twice a day (rinsed off with a weak solution of salicylic or carbolic acid), and dressed with an ointment containing Peruvian balsam, 1–30.* If the bed-sore be extensive, dusting with iodoform is very efficient treatment. We should be particularly careful not to let the skin be undermined. If this has already occurred, we must be prompt in the use of the knife or drainage-tube.

We can not recommend too strongly that the mouth should be kept clean. In a light case the patient can see to this himself, but otherwise the mouth and tongue must be frequently cleansed with a linen cloth wet in cold water or a solution of borax (1 to 30). Perhaps we need hardly repeat the reason for this excessive cleanliness. It lies in the causative relation between stomatitis and inflammation of the parotid gland, and of the middle ear. If the tongue and lips be dry, they may be touched with glycerine.

The diet must be at once liquid and nourishing. Milk is excellent, and it should always be ordered, but it can, unfortunately, be taken by very few patients continuously. It is often better borne if coffee or a little brandy be added to it. Cocoa made with milk may also be given for a change. In severe cases Nestlé's food (Kindermehl) has been often employed by us with benefit. Broths and soups, thickened with sago or rice, are also good. They may be made more nourishing by adding an egg to them. If the patient is very anxious to have more solid food, as often happens, we need not hesitate to give him a roll or rusk (Zwieback) that has been softened by soaking. If a patient becomes exceedingly enfeebled we should give him fine shavings of raw beef, regardless of the fever. A little dilute hydrochloric acid might be given with the meat. Beef-tea would be still better than the raw meat, and is to be strongly recommended. The various preparations of meat which are now made (meat-juice, meat-peptones, etc.) may be useful. When the fever takes a sluggish course, we must often begin to give stronger nourishment before the fever has ended. The best drink is cold water, which must be offered to the patient even when he does not ask for it. Lemonade and similar preparations generally become distasteful in time. Drinks containing carbonic dioxide are to be avoided, because they increase the meteorism. Cold tea with milk is good. In severer cases we should give some good strong wine, such as port, Malaga, or Hungarian wine, but it is not necessary to force the patient to take wine if he does not care for it. If the patient desires beer, we need not hesitate to give it in moderate amount. During convalescence we should be very careful about diet, since errors often have disagreeable consequences. We must wait till there has been no fever at all for seven to ten days before we allow a solid, animal diet, and return by degrees to common sorts of food.

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* The unguentum balsami peruviani is made by mixing one part of balsam very thoroughly with thirty parts of the glycerite of starch (B. P.). It is not officinal in Germany.—TRANS.

The general and dietetic treatment which we have thus far discussed is very important. Outside of this, it is our opinion that there is only one method of treatment to be chiefly considered—at least under the present limitations of our therapeutic ability. This method consists in the persistent use of cool baths, as first practiced by Brand in Stettin. We do not indeed believe that the indications for this method of treatment are exactly what its original promoter held them to be, and we think some of the minutiæ of the treatment should be changed. Yet there is at present no other single method of treating typhoid fever which has so numerous and evident advantages for the patient when properly and moderately used. To carry it out in private practice may often be more difficult than in a well-appointed hospital. However, even in private houses it will generally be possible to manage it, and we regard it as the duty of every physician who undertakes to treat a severe case of typhoid to try his best to have the baths employed.

The great advantages of the treatment by baths are: 1. The baths diminish the fever, if their temperature be only sufficiently low, by direct absorption of heat. The baths thus obviate, as far as possible, all the bad effects which might result from a rise of temperature. 2. The direct influence of the baths upon the nervous system is still more important. The intellect becomes clearer, the apathy and dullness diminish. In fact, if baths be used, we do not see nearly so often as formerly the grave "typhoid condition." It is thus evident that bathing not only causes an improvement in the subjective sensations of the patient, but brings in its train many other beneficial effects. The patient takes his nourishment better, does not so often swallow the wrong way, coughs more effectively, is easier to move, and his body and his mouth can be better cleansed. 3. The influence of the baths upon the respiratory organs is of the greatest importance. We refer especially to the stimulation to deeper inspiration, and the promotion of expectoration. The best proof of the benefit of this influence is the circumstance that if patients are subjected to baths from the start, it is comparatively a rare thing for severe bronchitis, atelectasis, and catarrhal pneumonia to develop. 4. The good care of the skin, which the bathing makes possible, is not to be despised. Since this treatment has been introduced, bed-sores are much rarer in typhoid than before. 5. Lastly, the baths are sometimes observed to have a diuretic effect.

What has been said shows that the height of the fever is by no means the sole indication for the employment of baths, at least in our opinion. The condition of the nervous system and of the respiratory organs is also to be considered. It is true that numerous mild cases run a favorable course without a single bath; but we should always remember that this treatment is not only directed against the symptoms already existing, but has also a prophylactic importance, since it tends to prevent any severe cerebral or pulmonary manifestations.

We will pass on to the special method of carrying out balneo-therapeutics in typhoid. Full baths are generally employed, immersing the patient to his neck. The tub must stand, if possible, by the bedside. In hospitals, where there are beds on rollers, it is a better way to wheel the patients into the bathroom. All who are severely ill should be lifted into the bath and there held and supported, to avoid any bodily fatigue. During the bath the skin should be gently rubbed. This averts unpleasant sensations of chilliness. The temperature of the water should not be set too low, especially for the first baths. We begin at 85° to 90° (24° to 26° R.), or, if the individual be elderly or sensitive and timid, at even warmer temperatures. When the patient has become accustomed to the temperature of the water, we can cool off the bath still further. Baths below 73° (18° to 20° R.) have scarcely ever been used by us, and we believe that they are seldom needed. A very satisfactory average temperature is 80° to 85° (20° to 24° R.). A bath lasts on the average ten minutes. If the patient feels very cold or very uneasy in the bath, it must be cut short. After the bath the patient is at once lifted into bed, wrapped up in a sheet previously made ready, and wiped dry, with rather vigorous rubbing of the extremities and back. The moist sheet is then removed. The patient is covered up rather warmly, and is given some hot broth or a sip of good strong wine. The effect of the bath upon the temperature is measured about half an hour later by the rectum. If the temperature be 2° to 3° (1° to 2° C.) lower than before, the result is deemed satisfactory. Often the difference is greater, but in severe cases the fever may be so obstinate that the temperature remits only a small fraction of a degree. In such cases it is sometimes permissible to lower the temperature of the bath still more, or continue it a little longer. If cool baths are ill-borne, protracted baths of lukewarm water are sometimes very efficient (Riess, and others).

In so far as the height of the fever furnishes an indication for baths, we may accept 103.6° (39.8° C.) in the rectum, as the temperature calling for a bath, but spontaneous variations of the temperature of the body are always to be regarded. In cases which show a low morning temperature without special interference, the height of the evening temperature is seldom an indication for a cold bath. We regard it as very important that the baths be not given too frequently, since their advantages may thus be overbalanced by their incontestable evil consequences. At present we rarely prescribe more than two to four baths a day. At night we have given baths only very seldom, when forced to by extremely high temperatures or other bad symptoms. It must be a mistake to wake a patient who is quietly sleeping, and put him into cold water, even if his temperature is above 104° (40° C.). On the other hand, even if the temperature be not excessive, or even if it be normal, there is, as we have said, no better remedy than the baths for severe pulmonary or cerebral symptoms. In such cases we often raise the temperature of the baths a little, and during them we have colder water poured upon the head and back. If we do this, the ears must be stopped with cotton-wool, lest the cold water find its way into them.

Advantageous as the bath treatment of typhoid generally is, it must, however, like every other therapeutic method, be used with wise moderation and with constant regard to individual conditions. If the patients are very weak, show a strong aversion to the baths, or feel dull and exhausted instead of refreshed after them, it is well to consider whether we really do good by continuing their employment. In such cases we have of late years often advised cold wet packs of the whole body in bed, instead of the baths, and we consider such a use of cold as very advisable in these cases. The antithermic action of the pack is, of course, much less than that of the bath, but it can easily be aided by the exhibition of antipyretics internally. The respiration and the nervous system, however, are almost always most favorably affected. Most patients readily consent to the packs and lie quiet for an hour or two in the wet envelope. We would urgently recommend the more frequent use of packs, especially in private practice, where the bath treatment often meets with greater difficulties than in hospitals. We should here always begin with higher temperatures, and only gradually go on to cold packs. There are a number of contra-indications to the use of baths which can not be disregarded. First, the occurrence of intestinal hæmorrhage, however slight, and likewise the suspicion that peritonitis is developing, prohibit bathing. In these cases quiet is the very first requirement of the patient, and the baths must be at once discontinued. The onset of otitis, severe laryngeal affections, and acute nephritis are also complications in which we should properly omit the baths entirely or employ them very cautiously, giving them warmer and less frequently. Sometimes rheumatic pains in the limbs or an attack of boils render the continued use of baths difficult. In short, we see that nothing could be more foolish than to try to establish a general rule for the bath treatment of typhoid.

Passing to the consideration of the further symptomatic treatment of typhoid, the first important question is about giving drugs to reduce the fever. The one-sided point of view which considered that the chief task of the physician in the treatment of acute febrile disease was to lower the temperature has been gradually abandoned. In describing the bath treatment of typhoid we have emphasized the fact that the action of the bath in reducing the temperature was only one factor, and perhaps neither the most efficient nor the most important factor, in the curative action aimed at. In giving antipyretics internally their antifebrile action is of chief importance, but some of them also have a certain sedative action on the nervous system. The important influence of the bath on the respiration and skin they do not possess. If we had to decide whether to treat typhoid exclusively by baths or exclusively by antipyrine and similar drugs we should decidedly prefer the former. We would not wholly exclude the use of antipyretics internally in the treatment of typhoid, but we would limit their use more than is often the case. In our opinion they are indicated only when with existing high fever baths are for any reason impossible or contra-indicated (*vide supra*), or when, in spite of the baths, the fever remains constantly high. In such cases the baths can often be judiciously combined with the use of antipyretics internally if the patient bears the drug well and feels better subjectively after the reduction of temperature than before. We often treat typhoid patients by baths in the day, and, if the fever be high, by a dose of antipyrine (fifteen to thirty grains, grammes 1–2) at night. With severe headache, nervous restlessness, etc., this remedy is especially indicated; but we regard it as at least useless and often improper to give a patient with moderate fever large doses of an antipyretic without any sufficient reason, as unfortunately is often the case in practice; the only permanent result is to make the patient feel worse and upset his stomach.

In regard to the different antipyretics so frequently brought forward and recommended of late, we are of the opinion that antipyrine is most to be recommended. It was introduced by L. Knorr and first recommended by Filehne. In doses of fifteen to thirty grains (grammes 1–2), best given in wafers, it usually reduces the temperature considerably, although we may find that the height and the obstinacy of the fever are not identical terms. We do not often see unpleasant effects from antipyrine (such as vomiting, profuse sweating with defervescence, a chill when the temperature rises again, and sometimes a measles-like eruption). The patient often feels better than before while under the influence of antipyrine, since the drug, as we have said, also acts favorably on the nervous symptoms (headache, restlessness). The dose of fifteen to thirty grains (grammes 1–2) can in severe cases be repeated several times a day, but, as a rule, we should not exceed eighty or ninety grains (grammes 5–6) in the twenty-four hours. One or two doses a day are usually all-sufficient. Lactophenine is also much recommended and even held as a specific in typhoid. In doses of seven to fifteen grains (gramme 0.5–1) it reduces the temperature considerably and usually causes a marked improvement in the general condition. A total of eighty or ninety grains (grammes 5–6) can be used in a day. Of the many other antipyretics which, of course, have often been tried in typhoid, we may first mention antifebrine (acetanilide), which in doses of four to seven grains (gramme 0.25–0.5) has a similar action to antipyrine (Cahn and Hepp), and it should be used in practice among the poor because it is much cheaper. If we avoid too large doses we seldom see unpleasant results. The appearance of a pale-cyanotic hue of the skin is the only disturbing feature; this, as in aniline poisoning, is probably due to a change in the coloring matter of the blood, and therefore warns us to be cautious. Other new antipyretics are phenacetine (fifteen grains, gramme 1, at a dose), salipyrine, etc., which have no special advantages. Quinine in doses of fifteen to thirty grains (grammes 1–2) and salicylate of soda in doses of seventy-five grains (grammes 5) are now little used as antipyretics, and properly, since their unpleasant action (vomiting, tinnitus, sweating, etc.) is much greater than that of the other drugs mentioned above.

[Water or an acid drink should be given frequently by the nurse without waiting for the patient to ask for it, unless the mind is unusually clear. Phenacetine seems to have proved itself less depressing to the heart than its predecessors. The antipyretic dose usual in this country is five grains. A strong protest should be entered against the routine or frequent use of any of these internal antipyretics. If the temperature seems, in itself, to cause restlessness and discomfort, an occasional dose may be given. When used early in the course of the disease, antipyretics may seriously embarrass the diagnosis in doubtful cases. The method of Brand has been slowly working its way in America of late years, and perhaps would have spread more widely and rapidly were it not that we have long been in the habit of frequently sponging our typhoid patients with alcohol and water.]

Another important symptom which needs special treatment is intestinal hæmorrhage. It has been already mentioned that if this occurs, the baths should cease at once. Further than this, the chief remedies are ice and opium. Flat ice-bags are laid upon the abdomen. They should not be too heavy, and should, if possible, be suspended from a hoop. Internally, the patient is given every two hours fifteen or twenty drops of laudanum or a powder of one-half grain or one grain (gramme 0.03 to 0.05) of opium, either pure or combined with acetate of lead (opii, gr. ss., grm. 0.03; plumbi acetatis, gr. j, grm. 0.05; sacchari albi, gr. j, grm. 0.05). The object of the opium is to check peristalsis, and thus promote the formation of a clot in the bleeding vessel. In severe cases we may try injections of ergotine or fluid extract of hydrastis, twenty drops three or four times. Liquor ferri chloridi (five to ten drops in water every hour) is often employed, but is of extremely doubtful value. The baths can not be resumed till there has been no bleeding for at least three or four days—and then only cautiously.

If peritonitis occurs, the treatment is much the same. Above all, opium must be used in still larger doses, but, unfortunately, as a rule, in vain. The surgical treatment of peritonitis has rather more of a future and more success, but experience of this is still scanty.

If there is considerable diarrhœa, we can give mistura gummosa [P. G., gum arabic and sugar, each 15 parts; water, 170 parts], or small doses of opium, the latter sometimes combined with tannin, tannigen, etc. In general it is certainly proper not to check the ordinary moderate diarrhœa of typhoid. Persistent constipation is always to be avoided. Constipation at the beginning of the disease is overcome by calomel (*vide supra*). In later stages we always try enemata first, to produce an operation. If this does not succeed, then we must employ rhubarb or castor-oil. Great tympanites may be diminished by laying cold wet cloths or ice-bags upon the belly. Considerable amounts of gas may often be removed by introducing a long rectal tube. As to puncturing the greatly inflated intestines, a method practiced by some physicians, we have no personal experience.

If there are severe pulmonary symptoms, baths or wet packs are, as we have said, the best remedies. Internally we may try liquor ammonii anisatus [P. G., olei anisi, 1 part; aquæ destillatæ, 24 parts; aquæ ammoniæ, 5 parts] and benzoic acid (grains ij to iij, gramme 0.1 to 0.2, in powder). If the pulse be very rapid, we may put an ice-bag over the heart. If at the same time the pulse is small and weak, we give stimulants. Of late we have most frequently given tincture of strophanthus (ten to fifteen drops several times) with the best results. We only rarely use digitalis in typhoid. If symptoms of severe cardiac weakness—so-called collapse—suddenly occur a rapid and energetic interference is urgently demanded, Subcutaneous stimulation, injections of ether or camphorated oil,* should be given, with wine, stropanthus, etc., internally. If the respiration stops it can sometimes be started up again by douches of cold water on the back of the neck, or we may often restore it by artificial respiration. Digitalis (one-half grain of the leaves, gramme 0.03, two or three times daily) may also be employed if the pulse be rapid; but it should be used with great caution.

For nervous symptoms the baths and douching are the most effective remedies. The head is meanwhile covered by an ice-bag. Antipyrine sometimes acts favorably on the nervous symptoms. If there be great excitement, as shown by excessive restlessness or delirium, small doses of morphine internally, or, better, subcutaneously, are often very useful.

The numerous other complications and sequelæ which may occur, but which can not all be mentioned here, should be treated on general principles.

The prophylactic measures to avoid the spreading of the disease can be only briefly referred to. Of chief importance is careful disinfection of the excreta. The best means for this purpose is a two-per-cent. solution of corrosive sublimate or a five-per-cent. solution of carbolic acid. We may also pour on milk of lime until the reaction becomes alkaline, or use concentrated mineral acids. We should take care that bed-pans, bed-clothes, linen, the rectal thermometer, etc., should be handled by other persons as little as possible, and should be carefully disinfected. If there seems reason to suspect that the disease was due to bad water, of course the source of such suspected water must be cut off.

[Recent experiments tend to show that the above solution of carbolic acid does not kill spores except after prolonged contact.

The following are the measures of disinfection recommended by the American Public Health Association. It will be observed that they apply to all infectious diseases, and it seems well to give them here nearly *in extenso*, as the directions for disinfection in most text-books are far too vague.

Disinfection of Excreta, etc.—The infectious character of the dejections of patients suffering from cholera and from typhoid fever is well established; and this is true of mild cases and of the earlier stages of these diseases as well as of severe and fatal cases. It is probable that epidemic dysentery, tuberculosis, and perhaps diphtheria, yellow fever, scarlet fever, and typhus fever, may be transmitted by means of the alvine discharges of the sick. In cholera, diphtheria, yellow fever, and scarlet fever, all vomited material should be looked upon as infectious; and in tuberculosis, diphtheria, scarlet fever, and infectious pneumonia, the sputa of the sick should be disinfected or destroyed by fire. It seems advisable also to treat the urine of patients sick with an infectious disease with one of the disinfecting solutions below recommended.

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* One part of camphor to four of olive-oil in doses of seven to fifteen minims (gramme 0.5 to 1) every hour or two.

Chloride of lime, or bleaching powder, is, perhaps, entitled to the first place for disinfecting excreta, on account of the rapidity of its action. The following standard solution is recommended:

STANDARD SOLUTION No. 1

Dissolve chloride of lime, of the best quality,* in pure water, in the proportion of four ounces to the gallon. Use one quart of this solution for the disinfection of each discharge in cholera, typhoid fever, etc. Mix well and leave in vessel for at least one hour before throwing into privy-vault or water-closet. The same directions apply for the disinfection of vomited matters.

STANDARD SOLUTION No. 2

Dissolve corrosive sublimate and permanganate of potash in pure water, in the proportion of two drachms of each salt to the gallon. This is to be used for the same purposes and in the same manner as Standard Solution No. 1. It is equally effective, but it must be left a longer time in contact with the material to be disinfected—at least four hours. The only advantage this solution has over No. 1 consists in the fact that it is odorless. It costs about two cents a gallon. It is very poisonous, and will injure lead pipes if passed through them in considerable quantities. Solutions of corrosive sublimate should not be placed in metal receptacles.

Disinfection of the Person.—The surface of the body of a sick person, or of his attendants, when soiled with infectious discharges, should be at once cleansed with a suitable disinfecting agent. For this purpose solution of chlorinated soda, diluted with three parts of water, or Standard Solution No. 1, diluted with three parts of water, may be used. A two-per-cent. solution of carbolic acid is also suitable for this purpose, and, under proper supervision, the use of a solution of corrosive sublimate (1–1,000) is to be recommended.

In diseases like small-pox and scarlet fever, in which the infectious agent is given off from the entire surface of the body, occasional ablutions with solution of chlorinated soda, diluted with twenty parts of water, will be more suitable than the stronger solution above recommended.

In all infectious diseases the body of the dead should be enveloped in a sheet saturated with Standard Solution No. 1, or with a five-per-cent. solution of carbolic acid, or a 1–500 solution of corrosive sublimate.

Disinfection of Clothing.—Boiling for half an hour will destroy the vitality of all known disease-germs, and there is no better way of disinfecting clothing or bedding which can be washed than to put it through the ordinary operations of the laundry. No delay should occur, however, between the time of removing soiled clothing from the person or bed of the sick and its immersion in boiling water, or in one of the following solutions; and no article should be permitted to leave the infected room until so treated.

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* Good chloride of lime should contain at least twenty-five per cent. of available chlorine. The cost of the solution is less than one cent a gallon. The sediment does no harm.

STANDARD SOLUTION No. 3

Dissolve four ounces of corrosive sublimate and one pound of sulphate of copper in a gallon of water. Two fluid ounces of this standard solution to the gallon of water will make a suitable solution for the disinfection of clothing. The articles to be disinfected must be thoroughly soaked with the disinfecting solution, and left in it for at least two hours, after which they may be wrung out and sent to the wash.

Clothing may also be disinfected by immersing it for four hours in a two-per-cent. solution of carbolic acid. Soiled mattresses, pillows, feather beds, and articles of this nature can not be effectually disinfected by sulphur fumigation, owing to the fact that the gas does not penetrate to their interior in sufficient amount. For articles of this kind, and in general for articles of little value, which have been soiled by the discharges of the sick, destruction by fire will be advisable.

Disinfection of the Sick-Room.—No disinfectant can take the place of free ventilation and cleanliness, and it is impracticable to disinfect an occupied apartment. Neutralizing bad odors is not disinfection.

All surfaces should be thoroughly washed with Standard Solution No. 1, diluted with three parts of water, or with a 1–1,000 solution of corrosive sublimate. Standard Solution No. 3, diluted in the proportion of four ounces to the gallon of water, may be used.

The walls and ceiling, if plastered, should be brushed over with one of these solutions, and subsequently washed over with a lime-wash.

Especial care must be taken to wash away all dust from window ledges and other places where it may have settled, and thoroughly to cleanse crevices and out-of-the-way places. After this application of the disinfecting solution, and an interval of twenty-four hours or longer for free ventilation, the floors and woodwork should be well scrubbed with soap and hot water, and this should be followed by a second more prolonged exposure to fresh air, admitted through open doors and windows.

As an additional precaution, fumigation with sulphur-dioxide gas is to be recommended, especially for rooms which have been occupied by patients with small-pox, scarlet fever, diphtheria, typhus fever, and yellow fever. All apertures into the room should be carefully closed, and not less than three pounds of sulphur for each thousand feet of air-space should be burned. To secure complete combustion, the sulphur, in powder or small fragments, and moistened with alcohol, should be placed in a shallow iron pan, and this should be placed on bricks in a tub partly filled with water to guard against fire.—EDITOR.]

[Formaldehyde gas is superior to sulphur dioxide, because of its greater efficiency and rapidity of action, and because it does less harm to household goods. Both gases have merely a superficial action.—V.]

CHAPTER II

TYPHUS FEVER

(Spotted Fever. Ship Fever)

TYPHUS fever is an acute infectious disease, perfectly distinct from typhoid fever, but formerly often confounded with it. The similarity of the two diseases, which led to their similar names, consists only in the grave general condition with fever, and in a number of complications which may appear in both. There is, however, an essential difference in the whole course of the two diseases, and especially in the circumstance that the intestinal lesion which is characteristic of typhoid is never seen in typhus. The chief distinction between the two affections, which must undoubtedly lie in the difference in their causes, can not yet be demonstrated. We do not yet know the organized pathogenic agents of typhus fever, although it must be presupposed that they exist.

Ætiology.—As to the way in which infection occurs, we have much less information even than in relation to typhoid. We know nothing as yet as to the supposed specific agents which produce typhus, although micro-organisms have been repeatedly found in the blood. It is an incontestable fact that the appearance of typhus in a place previously free from the disease is always to be referred to an importation of the pathogenic poison from without. It is likewise determined, through numerous observations, that typhus is one of the contagious diseases—that is, that the specific poison can be directly transferred from the patient to others around him. How it is transferred we have no certain knowledge. Perhaps the poison is contained in the expired air; or, as is still more probable, in the scales of epidermis; or, perhaps, in the other excretions and secretions of the patient. We are equally ignorant through what channel the infectious agent enters the system—whether it is inspired or swallowed. It is certain that the poison may be transferred in the clothes, etc., of the patient (fomites).

Favorable hygienic surroundings decidedly diminish the contagiousness of typhus fever. For example, in the well-ventilated pavilions of the Leipsic hospital there have rarely been cases of transfer of the disease to physicians, nurses, or other patients. On the other hand, if the hygienic influences be unfavorable, typhus fever may appear in very widespread epidemics. Those terrible epidemics which have been described under the names of "famine fever," "camp fever" (Hungertyphus, Kriegstyphus), etc., were for the most part typhus fever. In the smaller epidemics it is often possible to trace the disease to some wretched, overfilled tenement-house.

At present typhus fever appears constantly in Great Britain. Ireland has been notorious for many years as a breeding-place of the disease. It is also frequent in the eastern part of Germany (Posen, East Prussia and West Prussia, Silesia), in Poland, Galicia, Russia, and in parts of southern Europe. The isolated cases which occur every year here and there in central Germany, though more or less numerous, are, almost without exception, to be referred to an importation of the disease.

Typhus fever attacks by preference young adults of twenty to forty years; but it occurs in children, and is comparatively frequent in elderly persons. There is no marked dependence of the epidemics upon any particular season of the year. As in the case of typhoid fever, a person who has once had the disease seems to enjoy immunity from any fresh attack.

[The practical acquaintance of American physicians with typhus fever is, fortunately, limited. Many of the outbreaks which have occurred were traceable to immigrants, especially from Ireland.

During our civil war the disease broke out neither among the armies in the field nor among the prisoners of war. A number of cases were reported at the time, but great doubt has since been thrown upon the correctness of the diagnosis.]

Course and Symptoms of the Disease.—If we try to sketch the characteristic behavior of typhus fever, especially as contrasted with typhoid, we may say that the disease begins much more abruptly and rapidly, and that the fever quickly becomes very high and the general disturbance very severe, but the illness lasts a shorter time, seldom more than two weeks, and generally passes by crisis into recovery.

The length of incubation seems to vary. Murchison thinks it is usually more than nine days. Sometimes, though not invariably, slight prodromata precede by some days the actual outbreak of the disease. These are languor, anorexia, headache, and pain in the limbs. Then the regular illness begins, as a rule, rather suddenly, and often with a pronounced rigor. With this the temperature rises quickly, and may on the very first evening reach 104° or 105° (40°–40.5° C.). Vomiting is not rare, and may be repeated. A grave general condition, with fever, is developed in a few days. The patient feels exhausted. There is often violent pain in the loins and extremities. Nervous symptoms soon appear: persistent and intense headache, vertigo, spots before the eyes, ringing in the ears, and in many cases quickly increasing stupor and delirium. In severe cases the fever often reaches 106° (41° C.), and may be even higher, and it is almost constant, with but slight morning remissions. The skin is hot and dry, the tongue dry and thickly coated, the respiration moderate, the pulse very rapid. We very frequently find in the chest the signs of an extensive bronchitis. Nasal catarrh and conjunctivitis also occur. Serious intestinal symptoms are generally absent, although there may be slight tympanites or diarrhœa. The spleen is almost always greatly enlarged. Only in a few epidemics is the splenic tumor said to have been wanting (?). The urine is concentrated and scanty, and sometimes has a trace of albumen.

On the third to the seventh day of the disease the characteristic eruption appears. To this the disease owes its name of "spotted fever." The eruption consists of rose-spots, generally very numerous and widespread, upon the trunk and extremities, often also on the face. Sometimes the spots are larger, and may then bear great resemblance to a fresh eruption of measles. The skin between the separate rose-spots is not infrequently diffusely reddened. After two or three days the roseolæ become hæmorrhagic, and change into lighter or darker petechiæ. It is commonly only in the lighter cases that the rose-spots fade away without first becoming petechial. In rare though well-substantiated cases the eruption has been scanty, or even wholly wanting. Herpes does occur, but only seldom.

The fever begins to abate in light cases as early as the second week, coincidently with an improvement in the general symptoms. Often this change is indicated about the seventh day by a considerable remission in the temperature. On the other hand, in severe cases, all the symptoms grow worse. The weakness increases. The nervous derangement reaches the extreme of a severe "typhoidal state," expressed either by marked stupor, which sometimes passes into complete coma, or by violent delirium. Lobular pneumonia attacks the lungs, and the fever continues with unabated violence. These symptoms may end with death, but in favorable cases they decline rapidly. Sometimes this decline is preceded by a great rise in temperature (*perturbatio critica*), especially about the seventeenth day, rarely a few days earlier or later. In such cases the temperature is apt to fall by crisis, sinking in a day or two, with but slight interruption, down to the normal level. Even in those cases in which the descent is by gradations it is always decidedly more abrupt than in typhoid. The eruption quickly fades, the patients gradually improve, and, as a rule, become completely and permanently convalescent. It is true that some observers have seen relapses, but they are, at least in our present epidemics, extremely rare.

Complications and Varieties in the Course of the Disease.—From what we have said of its course, it is evident that the symptoms are essentially those of an intense general infection of the system. The sole demonstrable local lesion which is almost invariably present is the characteristic eruption, and this has evidently no causal relation to the severe symptoms of the disease. It is likewise extremely probable that most of the complications, which not infrequently arise in severe cases, are secondary, and occur in the way already described with considerable detail in the preceding chapter. They are just such complications as are possible in every severe general disease, and embrace otitis, parotitis, extensive lobular pneumonia, more rarely gangrene of the lungs, and pleurisy; also furunculosis, purulent cellulitis, bed-sores, dysentery, icterus, etc. Whether some of the local lesions which are observed may not be direct results of the pathogenic poison, we can not at present decide. Among these would come, first of all, the rare cases of lobar pneumonia and nephritis. Sequelæ are, on the whole, rare, though sometimes there is a tedious anæmic condition, or neuralgia, paralysis, etc.

The separate epidemics of typhus present considerable variety, not only as regards the occurrence of individual complications, but more especially in the general course and character of the cases. For instance, some epidemics are distinguished by the greater frequency of light attacks (*typhus exanthematicus levissimus*, unsuitably termed by some "febricula"). Here the entire attack runs its course in five to eight days. The fever is generally comparatively moderate; there are no severe general symptoms, and complications are exceptional.

Diagnosis.—It may be very difficult for a time to distinguish typhus from typhoid. The following factors are of chief importance: 1. The onset is much more abrupt in typhus than in typhoid, and is often accompanied by a pronounced rigor. 2. In typhus, the nervous disturbances usually appear earlier and are more severe than in typhoid. 3. The rash is seldom so extensive in typhoid as in typhus, and in typhoid it hardly ever becomes petechial. 4. In typhus the pains in the loins and limbs are generally much more pronounced. 5. If we still find it hard to decide, the manner of recovery will almost always settle the question. Recovery in severe cases of typhoid is, on the average, much more tardy and gradual, by lysis. In typhus it occurs generally by the seventeenth day, and by crisis.

The **prognosis** is chiefly determined by the severity of the fever and of the nervous symptoms. Extensive lobular pneumonia is the most frequent dangerous complication. The mortality varies greatly in the separate epidemics. It is sometimes only six or seven per cent., but may rise to twenty per cent.

Treatment is based on the same principles as in typhoid fever. There is no specific remedy. Besides good nursing, a judicious employment of baths is certainly our chief reliance for lessening the severity of many of the symptoms, such as febrile, nervous, and pulmonary disturbances, as well as for averting many dangerous complications. For all details of treatment we may refer to the preceding chapter.

CHAPTER III

RELAPSING FEVER

(*Relapsing Typhus—Febris recurrens*)

Ætiology.—This disease was first named by English pathologists relapsing fever, and by Griesinger *febris recurrens*. It has a peculiar course, made up of separate attacks, and is further of great interest because it is one of the first infectious diseases in which the specific pathogenic organisms became known, and, being easily demonstrable in each separate case, were utilized for the speedy and certain diagnosis of the disease. Obermeier discovered in Berlin, in the year 1873, that in relapsing fever the blood, at certain times, invariably contains peculiar thread-like micro-organisms. This discovery has since been universally confirmed; and it may be maintained that if once the presence of these "spirilli" be demonstrated in the blood, we are justified in making an absolute diagnosis of relapsing fever.

In Germany the disease did not become epidemic till the year 1868. In 1872 and 1873 there were considerable epidemics in Breslau and Berlin. Its last extensive appearance was in 1879 and 1880, when it spread over most of northern and central Germany, and was accurately studied by numerous observers. People of the poorer classes were almost exclusively attacked, and especially the " tramps." The uncleanly dens where these people lodge were found everywhere to be the chief centers of infection.

The precise manner of infection is as yet almost wholly unknown. All observers agree that the disease is directly contagious; but it can not be very contagious if the hygienic influences be good. At least the results of our late epidemics would imply this. In the Leipsic hospital, where at that time over two hundred and fifty cases were treated, and isolation could not be at all perfectly carried out, not one case of infection occurred. It is certain that the disease can be transmitted by direct inoculation with the blood of patients. This has been established by a Russian physician, by the experimental inoculation of healthy persons. Doctors have been repeatedly inoculated at the autopsy of those who have died of relapsing fever. The disease may likewise be transferred by inoculation to monkeys, while other mammals seem to enjoy an immunity from it.

[The first cases of relapsing fever observed in this country were in Irish immigrants coming over in the same vessel in the year 1844. At several periods since then more or less limited outbreaks traceable to immigration have occurred, but the disease has never acquired any foothold with us, and comparatively few physicians have ever seen it. So far as I can learn, only one case has ever been seen in Boston, and that was in the person of a physician from another city, who brought the disease with him and passed through it in the Massachusetts General Hospital.]

Clinical History.—The stage of incubation lasts about five to eight days. It is only exceptionally that some slight prodromal symptoms present themselves just before the outbreak of the disease proper. As a rule, it begins suddenly, with a more or less pronounced chill and intense constitutional symptoms. There are violent headache, great languor, anorexia, and especially marked pains in the loins and extremities. The temperature rises rapidly, reaching generally 106° (41° C.) or higher as early as the first or second day. The skin is hot and dry, and usually quickly assumes a very characteristic dirty-yellowish color. In Leipsic, we often saw herpes labialis, which seems, however, to have been rarer in epidemics elsewhere. The tongue becomes dry and thickly coated. Sometimes there is vomiting. The bowels are constipated, or there is a slight diarrhœa. The spleen becomes rapidly enlarged, being, as a rule, even larger than in typhoid or typhus. The liver is slightly enlarged. The chest presents the signs of a bronchitis, generally moderate, but in exceptional instances severe. The pulse is much quickened. It is seldom that there are severe cerebral symptoms beyond a certain apathy and stupor. We have seen delirium tremens sometimes, in drunkards. A very characteristic symptom, already mentioned, is the marked hyperæsthesia of the muscles, especially in the calves.

After these symptoms, accompanied by persistent and generally very high fever, have lasted five days to a week, there is a critical decline of temperature, with profuse sweating. The patient now improves so rapidly and decidedly that he thinks himself completely cured, and generally gives little credence to the physician's prophecy of a relapse. In rare but well-attested cases there has been really but one attack. The rule is that, after about a week, a second attack occurs, often a third after that, and, infrequently, even a fourth and fifth. In each of these, the above-mentioned symptoms are repeated more or less completely and violently. As the only certain and constant sign of the recurring attacks (the so-called relapses) is a fresh rise of temperature, it will be well to consider their peculiarities at the same time that we describe the course of the fever. During the intervals of normal temperature the other objective symptoms of disease are usually absent, except an evident splenic tumor, and, not infrequently, the peculiar pale-yellow hue.

Course of the Fever (see Fig. 3).—The beginning of the fever in the first attack is, as we have said, almost always sudden, so that it may even in a few hours reach a considerable height. The fever lasts, as a rule, five to seven days, but not infrequently as short a time as three or four days, or as long as ten or twelve days. During this time it may keep a tolerably uniform height, but oftener there are considerable remissions, which may even come to deserve the name of pseudo-crises. In such cases the temperature sinks in the morning to normal or even lower, so that we might believe the fever ended; but in the evening the temperature rises again to its former level. These pseudo-crises are most frequent toward the end of the attack, but do occur sometimes in the very first days. The absolute height of the fever is, as a rule, very considerable. Temperatures between 105.5° and 106.5° (41° and 41.5° C.) are very often observed, and in themselves are not especially ominous in relapsing fever. The highest temperature we have observed was 107.9° (42.2° C.). Sometimes the temperature is more moderate (between 102° and 104°, 39° and 40° C.). The fever almost always ends at the close of the attack by crisis, only rarely by a rapid, gradual decline. The crisis is often preceded by an especially great rise the evening before (*perturbatio critica*); so that the subsequent fall of temperature is very considerable. It generally occurs at night, and is accompanied by profuse perspiration. The fall may amount to 9° or 10° (5° to 6° C.). The temperature sinks almost always below normal, often as low as 95° (35° C.) or thereabouts. Once we saw it fall to 92.1° (33.4° C.).

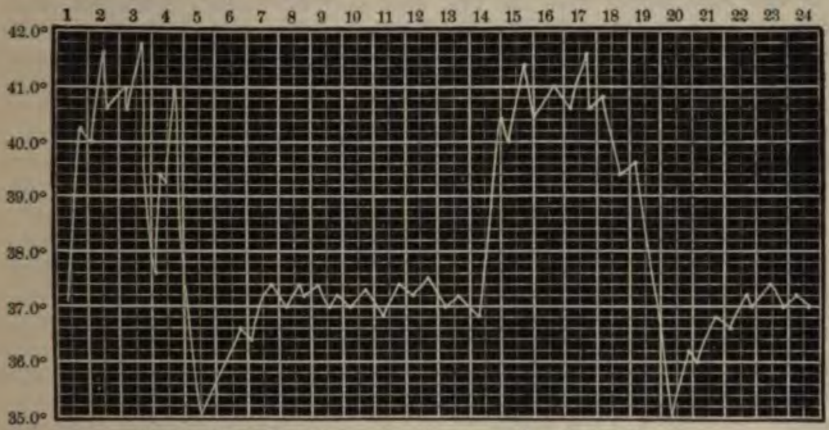

Fig. 3.—Example of the temperature curve in relapsing fever.

To the first attack succeeds an interval during which there is no fever (apyrexia), which lasts on the average about a week, sometimes a less time, and often a greater. The longest interval we have ever observed lasted seventeen days. During this interval the temperature, which, as a rule, is at first subnormal, rises to normal, and then generally remains there. Exceptionally there are slight evening exacerbations to above 100.5° (38° C.). These may have no demonstrable cause, or may result from some complication, such as otitis, or a furuncle. Then comes another change, and generally a sudden one, ushered in with a chill, and a new rise of temperature, the beginning of the second attack or first relapse. During this attack the fever has the same general peculiarities as in the first attack. Generally the first relapse is briefer by a day or two than the first attack; but the reverse is sometimes true. We will add that we have observed not infrequently a rather high evening temperature (101.5°, 38.5° C.) for one or two days before the second attack began, as also before the third.

Relapsing fever seems in many epidemics to have been made up of two attacks, so that not more than one tenth of the cases had a third attack. On the other hand, the majority of the cases in the last epidemic had two relapses. In these cases the rule was for the interval between the second and third attacks to be one or two days longer than the first apyrexia; but earlier epidemics seem to have had the second apyrexia, if there was one at all, briefer than the first. The third attack is set down in all reported cases as decidedly shorter than either of its predecessors. It lasts generally two or three days. Exceptionally we have seen it persist for four or even six days.

A fourth and even a fifth attack may occur, but only exceptionally. If they do happen, they are usually imperfectly developed, and often are limited to a fever of one day's duration. The more accurately and persistently we take the temperature during convalescence, the oftener do we find slight rises of temperature occurring at intervals late in the history of the case. These are probably to be interpreted as final, rudimentary attacks.

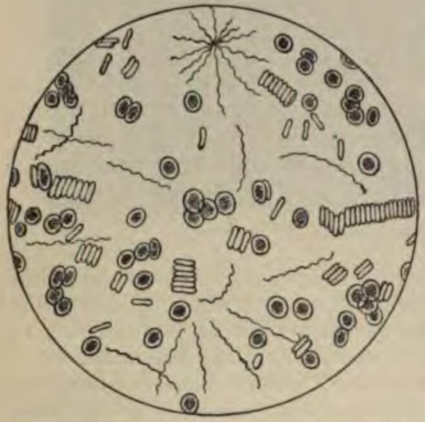

Fig. 4.—Spirilli of relapsing fever in the blood.

THE SPIRILLI.—The number of cases of relapsing fever in which no spirilli can be demonstrated in the blood, if the examination be accurate, has become so small that it can be disregarded, when we compare it with the much greater number of cases where such demonstration is made with ease and certainty. The best way is to get a drop of blood by pricking the skin, and examine it as it is, without mixing anything with it. Staining (with the basic aniline colors) is easy but unnecessary. With any good dry lens of 400 to 500 diameters the spirilli are seen with perfect distinctness. It requires a little practice to make them out; but this is easily obtained. Often the attention is first caught by little jogglings and motions of the red blood-corpuscles, and then we see the delicate, narrow threads. Their length equals about three to six times the diameter of the red globules (Fig. 4). They exhibit an active and almost continuous motion, like snakes. Often the whole thread bends upon itself, and then stretches out again. They are partly separate and partly tied up in knots composed of four to twenty individuals. The whole number visible in one field varies greatly in individual cases, and has no direct relation to the severity of the case. Often it requires long searching to find a few, while in other cases there may be twenty or more in the field at once. A very interesting fact is that their appearance in the blood depends upon the attacks of fever. On the first day of the attack we rarely find spirilli, and then only one or two. Upon succeeding days their number increases. Shortly before the end of the attack—that is, before the crisis—they generally disappear entirely; but even after the crisis they have been found, exceptionally and in very small numbers. They have very often been found by the author as well as other observers during the pseudo-crisis described above, so that, after the temperature has become normal, the presence of spirilli makes it very probable that another rise of temperature is impending. The spirilli have thus far been found in the blood only, in the catamenia, in bloody urine, or in blood coughed up from the lungs, and never in the organs or secretions (urine, milk, sweat, contents of herpetic vesicles). There can hardly be any doubt that the spirilli which appear in the separate attacks are to be regarded as separate generations. As to their manner and place of development we have as yet no knowledge. In the final, rudimentary attacks, we find few if any. If the patient dies during an attack, they are to be found in the blood after death. Artificial cultivations have not been very successful; nor have pure cultures of them, to our knowledge, ever succeeded. Albrecht states that they will subsequently develop in blood taken from a patient during the interval when he has no fever.

The blood is otherwise modified during relapsing fever. We very often find a slight increase in the white corpuscles. There is often a noticeable abundance of very small bodies, so-called granular elements (Körnchenbildungen). The significance of these (the remains of white corpuscles?) is still doubtful. There are, finally, peculiar cells, rather large, with fat-granules. They were demonstrated by Ponfick in the venous blood, and are said to come from the spleen. We also find fatty-degenerated endothelium in the blood.

Complications are on the whole rare, and mostly secondary. Important among these are troublesome ophthalmic disturbances, especially iritis and irido-choroiditis. Sometimes parotitis, laryngitis, or pneumonia occur. Epistaxis is a not infrequent complication. It is usually profuse and persistent, and it may even be dangerous. Sometimes there has been rather severe dysenteric trouble. In one case which ended fatally we observed a very peculiar intestinal lesion, consisting of hæmorrhagic-necrotic foci in the colon and lower ileum. In severe cases acute hæmorrhagic nephritis occurs with comparative frequency. At the autopsy an important and characteristic phenomenon is the wedge-shaped white spots which occur in the spleen, like infarctions. They have a clinical interest, as they may become the starting-point of pyæmic conditions or of peritonitis. Splenic abscesses have been observed in a few cases.

Variations in the course of the disease occur in this, as in all other acute infectious diseases. First there are mild, abortive cases, in which the attacks are few and brief. Then cases have been described resembling intermittent fever. Of chief importance is that severe variety of relapsing fever first observed in Egypt by Griesinger, and described as "bilious typhoid." There is no longer any doubt about the proper classification of this disease, for spirilli have been proved to appear in it, and it has even been shown that its inoculation upon another person (!) produces an ordinary case of relapsing fever. Bilious typhoid fever occasions successive attacks, like those of relapsing fever. The type is much more severe. As a rule, there appear marked icterus, grave nervous symptoms, hæmorrhages into the skin and mucous membranes; and the termination is frequently fatal. The autopsy shows a greatly enlarged spleen, often containing infarctions and abscesses, and in some cases hepatic abscesses, septic nephritis, and similar lesions.

The **prognosis** of ordinary relapsing fever is on the whole very favorable. In the last epidemics the usual mortality was only two to four per cent. The fatal cases could some of them be laid to wretched nursing. In the remaining portion death resulted from complications, such as pneumonia and nephritis.

The **treatment** must as yet be purely symptomatic. Antipyretic treatment is generally needless, since the fever is relatively brief and often quite intermittent. Moreover, most patients can not well endure cold baths, because the muscles are so painful. As a rule, good nursing and proper food amply suffice. If the muscular pains are very violent, we may order chloroform liniment as an embrocation. Complications are to be treated on general principles.

We are not acquainted with any remedy that can influence the disease itself or avert the relapses. Large doses of quinine, salicylic acid, etc., have been frequently employed for this purpose, but never with success. Lately there has been ascribed to calomel a favorable influence upon the general course of the disease, and its use is said to diminish the number of attacks. We must await further evidence in support of this statement.

CHAPTER IV

SCARLET FEVER

(*Scarlatina*)

We now begin the consideration of those acute infectious diseases which are usually embraced under the name of the "acute exanthemata." In this group are reckoned, besides scarlet fever, measles, rötheln, small-pox, and varicella. The point which these diseases have in common is that in all of them is developed a characteristic eruption, of slight clinical significance in itself, in most cases, but of thoroughly characteristic appearance in each disease, and hence of essential importance in diagnosis. A number of the acute exanthemata have this further point of mutual resemblance that they appear chiefly in children. These diseases are scarlet fever, measles, rötheln, and varicella.

Ætiology.—Infection with the specific scarlatinal poison occurs almost always by contagion, which takes place very readily. A single approach to a patient ill with scarlet fever may suffice to communicate the disease. There is no doubt that the disease may be transferred by objects which the patient has touched, such as linen, clothing, furniture, or toys. Persons who have been with the sick may be the means of transmitting the disease, although themselves unaffected. In England it has been thought that the contagium may be carried by milk.

Numerous observations show that the scarlatinal poison is with great difficulty destroyed, and that it may keep its contagious powers for months ("tenacity"). We can thus see how difficult, how impossible, it may be in an individual case to point out the source of contagion. Scarlet-fever patients can communicate the disease certainly as late as the end of the desquamative period, or perhaps somewhat longer.

Details as to the manner of contagion, or as to the specific poison itself, are as yet unknown. All statements made in regard to finding alleged specific micro-organisms of scarlet fever are extremely doubtful. The exciting agent of scarlet fever must, however, be contained in the blood and in the contents of the miliary vesicles of scarlet-fever patients, for the disease has repeatedly been artificially produced in healthy persons by inoculation with these fluids.

Predisposition to scarlet fever is far less universal than is predisposition to measles or small-pox. In families with several children often only one or two fall sick, while the rest escape, although equally exposed. As age increases, liability to the disease greatly diminishes, although there are cases of scarlet fever among adults. The majority of patients are between two and ten years of age. Scarlet fever is rare during the first year of life. It is an interesting fact that children with fresh wounds, either accidental or surgical, are especially liable to scarlet fever. An analogous and familiar fact is that women after delivery have a strong tendency to the disease.* As a rule, a person is attacked but once, so that, after the disease is over, an immunity from contagion is enjoyed; but there are exceptions to this rule.

Scarlet fever is now spread over the entire globe. In Germany there are almost always some sporadic cases in the larger towns, while from time to time, especially in autumn, there are more or less extensive epidemics in one place or another. There is considerable variation in the different epidemics of scarlet fever, as in many other infectious diseases, in the general character of the disease, and above all in the prevailing mildness or severity of the cases and the frequency of certain complications (nephritis, diphtheria), etc.

Clinical History.—The period of incubation is not definitely known. Many observations seem certainly to indicate a short period of two to four days, but the incubation stage is perhaps often somewhat longer—four to seven days. There are hardly ever any decided prodromata. The disease begins rather suddenly, with fever, often introduced by chilliness, and sometimes by a well-marked rigor. There is almost invariably a painful, scarlatinal sore throat. A further symptom, in all cases of any severity, is cerebral disturbance, generally rather intense. There may be headache, dullness, uneasy sleep, delirium, and, in smaller children, sometimes even convulsions. A very frequent and characteristic early symptom is vomiting, which may be repeated.

The characteristic rash usually appears as soon as the close of the first day, or on the second, and begins on the neck and on the chest and face, quickly becoming almost universal. The eruption consists at first of numberless small red points, crowded thickly together and soon united into a diffuse, intense, scarlet-colored erythema. The small and somewhat elevated points almost always correspond to the swollen hair-follicles. The diffuse redness is the result of an excessive hyperæmia of the skin, and vanishes completely on pressure. The skin as a whole seems slightly swollen and thickened. The back usually presents the most vivid tint. In the face there is generally pallor of the lips and chin, presenting a very striking and characteristic contrast to the bright-red cheeks. If some object like the end of a pen-holder be drawn over the red skin, there soon arise corresponding white lines, due to contraction of the blood-vessels. It is possible thus to make letters or pictures upon the back of the patient. We should add, however, that this is not a peculiarity of the scarlatinal eruption, being seen in other erythematous eruptions.

The rash persists for some three or four days, at first even increasing somewhat in vividness. It often appears more intense by artificial light than in the daytime. Meanwhile the severe general symptoms continue—the fever, the usually excessively rapid pulse, the cerebral symptoms, and the throat trouble. The spleen is often somewhat swollen, though seldom very large. Then the eruption begins to fade, the fever gradually ceases by lysis, the general condition and the difficulty in swallowing improve. With the end of the first week or the beginning of the second, the cases which run the typical course become fully convalescent. When the rash disappears, the epidermis usually begins to peel off, in a very characteristic way, in pieces of considerable size. The exfoliation upon the hands and feet is especially pronounced, and the little convalescents often amuse themselves by peeling off the epidermis in strips. Cases which are apparently the mildest and most benign may have their convalescence interrupted by the occurrence of a secondary scarlatinal nephritis. There is no sure prophylaxis against this.

We will now pass on from this general summary to a more complete consideration in detail of general and local symptoms; and we shall see how manifold are the clinical phenomena presented by scarlet fever.

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* In puerperal cases genuine scarlet fever and septic diseases were formerly often confounded. (See Chapter XVIII.)

1. *Fever* (see Fig. 5).—Although in a few undeveloped cases there is no fever, or scarcely any, almost all cases of any severity have high fever. It is only exceptionally that severe cases are observed in which the bodily temperature is little if at all elevated. As a rule, the fever rises rapidly upon the very first day, corresponding to the sudden onset of all the symptoms, to about 104° or 105° (40°–40.5° C.). The next day it often becomes a little higher still, and then persists with but slight variations, as a rule, so long as the eruption is at its height. During this period a temperature of 105° or more (40.5°–41° C.) is not infrequently observed. When the eruption fades, and the other symptoms decline, defervescence occurs. This happens but rarely by crisis, and that in the slight attacks. It is almost always by prolonged lysis, as in typhoid, only more irregularly and more rapidly. If the fever lasts into the second week of the disease, it is almost always (though not without exceptions) caused by demonstrable complications. The most frequent causes are the persistence of a severe sore throat, the occurrence of inflammatory changes in the cervical glands, or a purulent otitis media. In closing what we have to say about the fever in this disease, we would emphasize the fact that the pulse is often very rapid (140 to 160 a minute), even in comparison with the height of the temperature.

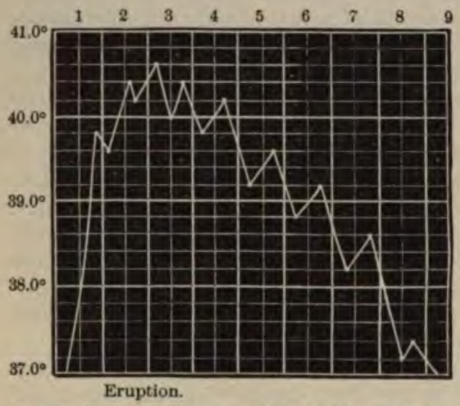

FIG. 5.—Example of a normal scarlet-fever curve.

2. *The Throat.*—The throat presents the most constant local lesion of scarlet fever. Sore throat is only in the rarest cases wholly absent;[*] but its form and intensity may vary extremely. The mildest variety is a simple, erythematous catarrh, without much swelling, but exhibiting usually a vivid and often punctate reddening of the soft palate and tonsils, the pharynx, and also the mucous membrane of the hard palate, frequently associated with enlargement of the little mucous follicles. Sometimes minute hæmorrhages take place into the mucous membrane. In other cases the scarlatinal affection of the throat is from the start associated with considerable swelling of the parts, and especially of the tonsils, justifying the term "parenchymatous sore throat." Not infrequently small abscesses form in the lacunæ of the tonsils; or superficial spots of necrosis develop which leave behind them larger or smaller ulcers, and sometimes occasion considerable hæmorrhage. There may even be a circumscribed gangrene of the tonsils.

The most important, because it is also the most dangerous, of the affections of the throat resulting from scarlet fever is the so-called scarlatinal diphtheria—that is, a diphtheritic inflammation of the tonsils and soft palate. This usually develops on the third, fourth, or fifth day of the disease, replacing a simple inflammatory condition of the parts. Whitish, dirty-colored spots develop on the tonsils, the arches of the palate, and the uvula. These rapidly increase in size, and cause a dry necrosis of the mucous membrane and subsequent ulceration. The process is a truly diphtheritic one—that is, there is an inflammation combined with an extension into the diseased tissues of a fibrinous exudation.

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[*] Absence of sore throat has been noted by others and by myself, especially in the scarlet fever of women in childbed. I suspect that the reason for this is that the infection does not enter in the usual way through the mouth, but from some wound due to parturition. On the other hand, it is to be noted that in the scarlet fever from wounds, when the infection follows an injury to the finger, etc., a sore throat occurs.

It is especially characteristic of scarlatinal diphtheria that there is almost invariably a considerable swelling of the cervical lymph-glands, except in those cases which die very quickly. It is true that the glands are usually somewhat enlarged in the milder forms of pharyngitis accompanying scarlet fever, but they seldom attain the size observed in the true diphtheritic process. In this there is an inflammatory and œdematous infiltration affecting often not only the glands themselves, but also the surrounding connective tissue, so that in severe cases the whole cervical region and the floor of the buccal cavity presents a firm and usually a very painful enlargement. It should be added that the severity of the throat symptoms and the extent of the glandular swelling are not always commensurate. Almost always the scarlatinal diphtheria is associated with a marked stomatitis, and very often also with a severe purulent or even diphtheritic rhinitis, although an invasion of the larynx (*vide infra*) is only exceptionally seen. At the alæ of the nose and the corners of the mouth there are often superficial ulcers from this cause. Otitis is also a frequent complication of scarlatina (*vide infra*).

The influence of the scarlatinal diphtheria upon the general condition of the patient is always considerable. Apart from the marked local discomfort, there is often a severe general septic condition. Grave signs of cardiac weakness (a very rapid, small pulse) are very apt to appear early. Moreover, cases of scarlet fever associated with pharyngeal diphtheria often show at the same time other severe septic complications (inflammation of several joints, simple or purulent inflammation of the serous membranes, nephritis, etc.). Many cases end fatally in a few days, while others pursue a more tedious course, lasting perhaps several weeks before death comes. These are often associated with pyæmic processes in other parts of the body.

With regard to the pathogenesis of the throat troubles seen in scarlet fever, the more simple forms are in all probability directly associated with the scarlatinal process—that is, they are direct sequences of the affection. In regard, however, to the severer forms, and especially to the diphtheritic variety, it is almost certain that these are not a direct result of the scarlatinal poison, but are due to some secondary infection which occurs on the soil furnished by the primary scarlatinal angina. The severe necrotic disease of the mucous membranes and the secondary changes in the lymph-glands and other septic complications are due chiefly to streptococci (Löffler); but we are almost compelled to suppose that there is some intimate connection between the two varieties of infection, else why should this peculiar secondary disease, the scarlatinal diphtheria, be so often conjoined with scarlet fever? It seems to us certain that "scarlatinal diphtheria" should not without further evidence be identified with true diphtheria, in spite of the similarity of the anatomical ("diphtheritic") changes. From a purely clinical standpoint the two present several important points of difference. In particular the scarlatinal diphtheria, in contrast with the primary form of the disease, seldom spreads to the larynx. Laryngeal croup is therefore but rarely seen in scarlet fever. The severe dyspnœa which sometimes develops in the course of scarlet fever is probably caused by an inflammatory œdema of the glottis. Another important clinical distinction between genuine and scarlatinal diphtheria is that paralysis of the soft palate, the ocular muscles, and other parts is scarcely ever a sequel of scarlatinal diphtheria. On the other hand, however, it is easily conceivable that many similarities between the two diseases should exist, since severe secondary septic streptococcus infection may occur both in scarlatinal angina and in true diphtheria. It is also possible that in some cases an actual true diphtheria should appear as a secondary infection in scarlatinal angina. The reported cases of laryngeal croup after scarlet fever may thus be explained. All these conditions, which have been much discussed, will be made wholly plain when the accurate bacteriological investigations of individual cases become more numerous.

3. We proceed by a natural sequence to the consideration of the affections of certain parts adjacent to the throat, troubles which must be regarded as chiefly the result of direct extension, or of a conveyance of the inflammatory process from the throat.

The stomatitis we have already mentioned, as well as the disturbance in the neighboring lymph-glands and the surrounding tissue. Parotitis is not rare in severe cases. Of especial importance is the scarlatinal inflammation of the middle ear, which often results in permanent deafness.

This inflammation usually arises at the time of desquamation, but it may occur earlier. It is either a simple catarrh, a purulent otitis media, or, in severe cases, an actual diphtheritic process. The deafness and earache are easily overlooked, as the other symptoms of the patient occupy the attention, so that the ear trouble is first recognized by the occurrence of perforation of the tympanum and subsequent purulent otorrhœa. After this declines there very often remains behind, as we have said, permanent deafness. Statistics have shown that four or five per cent. of all cases of deafness are referable to an attack of scarlet fever in childhood. The otitis is seldom immediately dangerous, but yet cases of purulent meningitis have been observed to follow it.

We have already spoken of the purulent or even diphtheritic rhinitis which almost always accompanies the scarlatinal sore throat. In rare cases there may also occur a purulent conjunctivitis, which is most probably the result of a direct conveyance of inflammatory secretions.

The tongue in scarlet fever deserves special mention. The first coating cleans off, and then the tongue usually presents a very characteristic appearance. It is diffusely reddened and covered with little elevations corresponding to swollen papillæ (strawberry tongue, scarlatinal tongue).

4. The characteristic eruption, as developed in the great majority of cases, has been described above. It remains to describe certain variations from the usual appearances.

First, the eruption may be rudimentary. It is then not pronounced, and visible only on a limited portion of the body (face, trunk, or extremities).

Variations from the type are not rare; sometimes the papules are more strongly developed (*scarlatina papulosa*); very frequently there are little vesicles (*scarlatina miliaris*). This latter form of the eruption appears by preference upon the trunk, but it may come also upon the extremities, and is often brought out by excessive perspiration, or by wrapping up the patient too warmly. Many epidemics are noticeable from the frequent appearance of this miliary form. More rarely the rash has a spotted look, resembling the eruption of measles (*scarlatina variegata*). There may be minute ecchymoses, which are not very rare and not ominous. Well-developed cases of hæmorrhagic scarlatina are, however, very dangerous, because here the general infection of the system is almost always exceedingly severe, and probably due to secondary sepsis; and there is besides, as a rule, a general hæmorrhagic diathesis. Other cutaneous lesions, especially herpes and urticaria, are not so very unusual in connection with the scarlatinal eruption. Furunculosis has been repeatedly observed after the rash fades.

Desquamation generally begins as soon as the rash has completely disappeared, but it may not occur till a few days or even one or two weeks later. Its extent corresponds in general to the severity of the eruption, although extensive desquamation may follow a rudimentary eruption. It is seldom bran-like or furfuraceous, as in measles. The rule is for it to be in lamellæ, so that, as we have stated, quite large strips of epidermis may be detached entire.

In rare cases an œdema of the skin appears after scarlet fever, which can not be shown to depend upon nephritis (*vide infra*), but which may perhaps be due to an abnormal permeability of the walls of the cutaneous blood-vessels following the eruption (*hydrops scarlatinosus sine nephritide*).

5. *Kidneys.*—Next to the severer forms of throat trouble, the most important and dangerous complications are located in the kidneys. They may appear as early as the acme of the disease, as in many other infectious diseases. The urine has a trace of albumen. In rare cases the amount of albumen may be considerable. The appearance of the urine is generally not much changed, and the microscope reveals but few abnormal constituents. There are some white and red blood-globules, a few hyaline casts, sometimes one or two renal epithelial cells. This initial albuminuria very rarely gives cause for alarm.

The genuine scarlatinal nephritis scarcely ever develops much before the end of the second or the beginning of the third week. Sometimes it comes even later. In one case under our own observation it did not begin till the thirty-third day of the disease. It may therefore be regarded to a certain degree as a localized relapse. It may be so mild as to cause no subjective symptoms whatever, so that it would be unnoticed if the urine were not carefully examined. On the other hand, it may be accompanied by the gravest symptoms, and may soon terminate fatally. It may follow either severe cases or the mildest, so that the rule should be to examine the urine in every case of convalescence from scarlet fever as often and as carefully as possible. No exact statement can be made as to the frequency of this complication, for it is much more common in some epidemics than in others.

The development of nephritis is often marked by a fresh rise of temperature. The elevation may be slight or it may reach 104° (40° C.). According to our own experience, the fever often comes a day or two earlier than the changes in the urine. As the nephritis goes on, it is very often accompanied by a moderate fever with remissions. This fever may be almost wholly absent, especially in mild cases. The pulse generally becomes harder, and is sometimes quickened; but in many cases it will be slow, and it is sometimes irregular. Among other objective symptoms, the first to excite notice is generally a slight puffiness of the face, which is usually pale. The eyelids, particularly, present an evident œdema. In the milder cases this œdema remains limited, while in others it gradually increases in extent and degree, involving first, as a rule, the dependent parts of the trunk, and later the extremities. Severe cases develop a pronounced anasarca. There are then, usually, effusions into the serous cavities, especially ascites and hydrothorax. The latter is dangerous chiefly through disturbance of the respiration, particularly if it be associated with severe bronchitis or pneumonia (*vide infra*). Although in general the development of a universal dropsy is characteristic of scarlatinal nephritis, œdema may at times be wholly absent. On the other hand, it is noteworthy that the œdema may occasionally show a peculiar localization, especially in the mucous membranes (œdema of the conjunctivæ, œdema of the ary-epiglottic folds with symptoms of laryngeal stenosis, œdema of the uvula and soft palate, etc.).

The urine exhibits the most important changes (*vide* the section on renal diseases). These may be insignificant in the milder cases, but they are very pronounced in the severe ones. The amount is much diminished. Sometimes there will be for several days almost complete anuria. In cases of any severity the urine is turbid, dark, often evidently bloody, with increased specific gravity (about 1015 to 1025), and containing a large amount of albumen. The sediment is generally abundant, and exhibits numerous hyaline casts of various lengths and diameters. To these may be attached red or white blood-corpuscles, detritus, granules of hæmatoidin, or bacteria. In cases of some duration the casts are often moderately fatty. Very frequently there are found noticeably long and broad waxy [fibrinous] casts, which are opaque and yellow. In many cases of scarlatinal nephritis the urine is peculiar in having very many white blood-corpuscles, either isolated or adhering to the casts. These undoubtedly originate for the most part in the kidneys. Red globules, some of them in the form of colorless rings, are found. They are usually present in small numbers, but may become more abundant, especially for a day at a time. We have seen a very hæmorrhagic urine after the special albuminuria had disappeared. Renal epithelium is frequently seen, but not invariably nor in very large amount.

Uræmic symptoms often develop in scarlatinal nephritis. They may be of all degrees of severity. They will be described in detail under diseases of the kidney (*vide infra*). The uræmia may be so severe as to cause convulsions, coma, and death; but it is remarkable how often children recover from what seems to be the most pronounced uræmia.

The duration of scarlatinal nephritis varies greatly according to its severity. In cases which run a favorable course, the urine is generally abnormal for two to four weeks, or even longer. There is sometimes a very slight albuminuria present for months without causing any symptoms. Death may be due to uræmia or more frequently it may come on with severe dyspnœa. The latter is due partly to dropsical conditions (hydrothorax, ascites), or often to a severe diffuse bronchitis or pneumonia accompanying the nephritis. Cardiac insufficiency also may be the cause of death. The transition of acute scarlatinal nephritis into chronic nephritis, especially into chronic contracted kidney, is rare, but it has certainly been seen both by ourselves and others. It is worthy of note that, after the nephritis has lasted some four to six weeks, beginning hypertrophy of the left ventricle can be detected by the displacement and strengthening of the apex-beat. Friedländer has demonstrated this early hypertrophy at autopsy.

The anatomical changes of scarlatinal nephritis can be mentioned only briefly here. The forms vary. We often find the "large white kidney"—that is, a diffuse acute nephritis with fatty degeneration of the epithelium—and usually also more or less numerous hæmorrhages. In other cases the kidneys macroscopically are apparently little altered, but we see in the cortex the gray, prominent, bloodless glomeruli, in which the microscope shows very marked changes ("glomerulonephritis" of Klebs and others).

6. *Joints.*—When desquamation begins, or even earlier, pain and swelling may attack a certain number of the joints. This trouble was formerly called scarlatinal rheumatism, but now it is usually known as scarlatinal synovitis. It is generally mild and quite temporary, but, exceptionally, it may be severe and even purulent. Then it is usually a part of a secondary sepsis or a general pyæmia, as evinced by such other lesions as empyema, subcutaneous abscesses, jaundice, splenic tumor, nephritis, etc.; and they all seem to be caused by a secondary infection with streptococci, etc.

We have seen a few instances of excessive pain in the muscles of the thighs, accompanied by a moderate, diffuse swelling.

7. Another important complication of scarlet fever is pneumonia. In severe cases lobular pneumonia sometimes appears as early as the first stage of the disease; but it occurs more frequently in connection with the nephritis, when it may have the significance of so-called nephritic pneumonia (*vide infra*). The respiration may be very seriously embarrassed by it. Inflammations of serous membranes in the chest—viz., endocarditis, pericarditis, and pleurisy—are more

rare. They may or may not be accompanied by disturbances in the joints (*vide supra*). Pericarditis, endocarditis, and myocarditis sometimes develop during scarlet fever, but it is usually hard to decide whether they are due to the direct action of the scarlatinal poison itself or to secondary septic complications. We refer the tendency to tachycardia or bradycardia, irregularity of the heart-beat, etc. (which sometimes last long after the scarlet fever), to myocarditic changes. Endocarditis may result in permanent valvular disease. Quite severe intestinal symptoms, such as diarrhœa, may appear. These are generally the result of a catarrhal inflammation of the intestinal follicles. Dysentery is less frequent. The enlargement of the spleen has been already mentioned. The liver is sometimes found to be considerably enlarged. A general moderate swelling of the lymph-glands (neck, axillæ, groins) is usually present in scarlet fever at the time of the eruption. We usually find in the blood a pronounced leucocytosis which sometimes lasts long after the other morbid symptoms.

Variations in the Course of the Disease.—The diversities of the clinical picture in different cases of scarlatina will be understood when we consider the variety and number of the disturbances thus far cited. It is to be added that the general course of the disease may exhibit numerous peculiarities, of which it is hardly possible to give an exhaustive presentation. We will content ourselves with a cursory statement of the most important deviations from the typical course.

1. *Rudimentary Forms.*—To this class, in which the disease does not reach a perfect development, belong first the cases of simple sore throat with no eruption, or at most an extremely faint and partial one (*scarlatina sine exanthemate*). Sometimes even the sore throat is hardly to be seen, and there is nothing but a brief and slight fever with mild symptoms of general disturbance. The recognition of these cases as scarlatinal is possible only when we consider their ætiological relation to other undoubted cases of scarlet fever. We had an excellent opportunity to observe them when the disease broke out in the children's wards of the hospital at Leipsic. The diagnosis is sometimes confirmed by a slight though evident desquamation, affecting the hands, feet, legs, and back, or by an acute nephritis, which may follow the mildest attacks of this sort. Many cases of acute nephritis, though apparently wholly spontaneous and primary, must be regarded as ætiologically scarlatinal.

2. *Rudimentary but Pernicious Forms.*—Under this head belong those attacks of scarlet fever in which the eruption is scanty or absent, while from the very start the most violent general symptoms appear. There is a very high fever, enormously rapid pulse, and delirium. Such cases must be the result of an uncommonly severe general infection. They usually end in speedy death. Other cases, ending fatally in a few days, have a well-developed rash without other localized disturbances.

3. *Severe Forms with a more Protracted Course.*—In these cases the long duration is not the exclusive result of especial complications, but is likewise due to the severity of the intoxication. One variety is the so-called typhoidal form of scarlatina, with persistent high fever and severe constitutional symptoms. Another variety is the hæmorrhagic form briefly mentioned above, in which there are extensive hæmorrhages into the skin and into the mucous and serous membranes. This form may run an extremely acute course. Further, in all pernicious forms, there may be severe local complications, particularly diphtheritic or gangrenous sore throat, inflammations of serous membranes, etc. Attacks of this sort are often not produced by the poison of scarlet fever alone, but by secondary complicating processes. While speaking of scarlatinal diphtheria it has already been pointed out that secondary infection of the system may result,

principally originating from the diseased throat, and occasioning sometimes grave septic conditions of the whole body, sometimes local disease of different parts.

4. In extremely rare cases *Relapses* do occur. After the first illness a fresh eruption breaks out with all the other symptoms of scarlet fever. In anomalous cases, running a severe course, there is sometimes, at an advanced stage, a fresh, imperfect eruption (generally in spots), which Thomas has termed a pseudo-relapse. Probably this is usually a septic eruption.

Diagnosis.—The diagnosis of scarlet fever is made in most cases from the characteristic eruption taken in connection with the other symptoms. We should, however, bear in mind that exceptionally other eruptions appear which exhibit the closest resemblance to that of scarlet fever. 1. After the use of certain drugs, especially atropine (belladonna), quinine, antipyrine, morphine, chloral; and likewise after the ingestion of crabs, fish, etc. 2. As a symptom of other infectious diseases, such as typhoid fever, small-pox; and, above all, in septic diseases (*vide infra*). In an uncertain or anomalous case, factors of importance for diagnosis are the ætiology (connection with other well-defined cases), the initial sore throat, and the eventual occurrence of desquamation or of a secondary nephritis.

The **prognosis** must in every case be guarded. From what has been said of the course of the disease, it is evident that, even in cases which are at first apparently the most favorable, dangerous complications may appear. The commonest dangerous complication during the height of the disease is scarlatinal diphtheria, the commonest dangerous sequel is nephritis.

Treatment.—The majority of those cases of scarlet fever which take a typical course will recover completely without our aid. In these the task of the physician, so far as treatment is concerned, consists in arranging the details of hygiene and the general care of the patient. The sick-room should be cool and well ventilated and the diet rather strict, consisting mainly of milk. Broths and eggs may also be allowed. We should see that the skin and the mouth are kept clean. To change the linen frequently, if done with proper precaution, is not only permissible, but very desirable. Rubbing the skin with olive-oil, vaseline, etc., has some merit, and is especially to be recommended if the skin be harsh and dry after the eruption has faded.

[From the moment that the disease is declared the patient should be thoroughly anointed daily with carbolized vaseline, lard, or the like; and this should be kept up until desquamation has ceased. Not only is the comfort of the patient promoted, but the danger of the spread of the infection is thereby greatly lessened.]

The scarlatinal disease of the throat must be treated with the greatest attention, the main duty of the physician in this regard being to prevent, if possible, the ingress of the above-mentioned secondary infection. It is therefore our opinion that in every case of scarlet fever the greatest pains should be taken from the very commencement of the disease to maintain complete disinfection of the mouth and throat. Larger children may use a gargle (two-per-cent. solution of chlorate of potash, one- or two-per-cent. solution of carbolic acid). Inhalations of carbolic-acid spray, dilute lime-water, etc., are also to be recommended where practicable. If there is prostration, or if the child be young or willful, we must cleanse the mouth and throat at short intervals, by means of a spray-apparatus, with disinfectants, such as boracic acid or permanganate of potash in solution. Sometimes it is a good plan to let the patient swallow slowly a half-teaspoonful of a solution of potassic chlorate (about 1 to 40), every half-hour or oftener, with the object of contributing to the local disinfection of the throat. If scarlatinal diphtheria nevertheless develops and the cervical lymph-glands begin to increase further in size, there is reason to hope, according to the experience of Taube and Heubner, that parenchymatous injections into the tissue of the tonsils or the palatine arches may yet check the spread of the secondary infection. About 6 minims (a Pravaz syringe half full) of a three-per-cent. solution of carbolic acid may be injected twice daily upon each side by means of a long hollow needle and a subcutaneous syringe. Catti recommends painting the affected parts with a 1-to-1,000 solution of corrosive sublimate. Of the many other remedies advised, we may add that the insufflation or dusting of the tonsils with powdered sugar is of advantage. In ordinary scarlatinal diphtheria we can expect no benefit from Behring's serum-therapy, but if there is a complication with true diphtheria a serum-injection is most advisable.

If the nose be likewise affected, the chief thing to do is frequent cleansing and syringing while the head is bent forward. We should be on the watch for the possible occurrence of otitis. In this particular the physician is often guilty of sins of omission. Much harm may be averted by a prompt syringing of the ears, or, if need be, by insufflation of air into the middle ear, or paracentesis of the membrana tympani.

Inflammation of the glands in the neck, if severe, is prone to pass on to suppuration, and it must then be treated surgically. When the swelling has just begun, or is still moderate, we may try to cure it by rubbing in iodoform ointment (1 to 15) two or three times a day. Ice is generally not so well borne as warm applications (poultices or warm bran-cushions).

If there be continuous high fever, accompanied by rather severe constitutional symptoms, a moderate employment of the cold-water treatment in scarlet fever is strongly to be recommended. The baths seldom need to be cooler than 81° to 88° (22°–25° R.), and are to be employed two or three times daily, or oftener in severe cases. If the nervous disturbance be serious, or if the respiration be impaired, the patient should be douched with cold water during the bath. Instead of baths, wet packs may be used to advantage. Internal antipyretics, such as antipyrine, may usually be dispensed with, although in private practice we may be obliged to employ them.

We must watch the condition of the heart carefully. When the pulse is very rapid we may use an ice-bag on the heart. When the heart begins to grow weak we may prescribe tincture of strophanthus, wine, etc., and the most efficient remedy for threatening collapse is injections of camphor. With mild scarlatinal arthritis salicylate of sodium and antipyrine act well as palliatives, but in severe cases they, of course, do little good.

We know of no means to avert the nephritis. In justice to himself, the physician must always at the start point out the possibility of its occurrence, and must avoid as far as possible errors in diet or exposure to cold on the part of his patient. He may thus escape blame. The treatment of the nephritis and its results is fully described in the section on renal diseases. The most important remedies are a mild diet and the use of warm or hot baths and packs. The other complications of scarlet fever must be treated symptomatically in the ordinary way.

The patient must, as a rule, keep his bed three or four weeks, even if convalescence be uninterrupted.

[This injunction is rather extreme. Nephritis is as likely to follow a mild as a severe case, and occurs sometimes in spite of every precaution. The physician should use his discretion as to the length of time the patient is kept in bed, carefully guarding against exposure to cold and imprudence in diet.]

After desquamation is completed the skin should be thoroughly cleaned by warm baths. The disease is so dangerous that, whenever a case occurs in a family, isolation is absolutely demanded, and, if possible, all the other children should be sent away. If this advice be disregarded, we can reject all responsibility for any further cases and their results.

[Scarlet fever is a disease at once so highly contagious and so common that it may be taken as the type of its class. Its hygienic treatment and the measures needful to prevent its spread consequently deserve more minute detail.

The sick-room should be at the top of the house, if possible, and exposed to the south; every unnecessary article of furniture and all ornaments should be removed beforehand, carpets, curtains, and stuffed or upholstered furniture being included. A window should be kept open constantly, top and bottom; in cool weather a fire should be burning; in warm weather ventilation is furthered by placing a gas-burner or large kerosene lamp near the throat of the chimney. Outside the door of the sick-room a sheet moistened with a disinfectant solution should be carefully hung. Only those whose presence is absolutely necessary are to be allowed in the sick-room, and the physician, when his visit is completed, should pass directly out of the house.

A convalescent should be kept away from all who are liable to contract or convey the disease until desquamation has entirely ceased. Several warm soap-baths should be given before the child emerges into every-day life, and it should finally be dressed in uncontaminated clothing.

For further directions as to the disinfection of the room, the clothing, and the excreta, see pages 28–30.]

CHAPTER V

MEASLES

(Morbilli)

Ætiology.—In contrast with the malignancy of scarlet fever is the comparatively benign nature of measles, a disease of childhood which is but little feared even by mothers. It is so widespread, and the susceptibility to it is so universal, that measles passes for an almost unavoidable but comparatively insignificant annoyance. Indeed, few escape it; and probably the reason why adults have measles so much less frequently than children is simply that most adults have already suffered from it in childhood. A second attack of measles in the same individual may occur, but it is certainly extremely rare.

[In highly civilized countries measles has prevailed so long that it would seem that a relative resistance against the poison has been acquired. The frightful ravages of the disease when it was planted in virgin soil, as among the Fiji Islanders not many years ago, apparently bear out this view. The susceptibility to measles is greater and more widespread than is that to scarlet fever—that is to say, fewer individuals reach adult life without having experienced an attack of the former than of the latter.]

Measles generally comes in epidemics. Sporadic cases are exceptional. In this respect measles differs decidedly from scarlet fever. The rapid spread of the disease when it has once broken out is a result of its great contagiousness. If one child in a family is attacked, the others almost always take the disease. The infection may be transmitted even by well persons and by means of articles with which the sick have come in contact, although this is not often the case. We are not yet acquainted with the specific poison of measles—although its existence is to be taken for granted—nor with the details of its transmission. Still it seems most probable that the poison is inhaled through the mouth and nose, and that this is the reason why its effects are usually first developed in the respiratory passages (*vide infra*). The disease can be artificially produced by inoculation of healthy children with the blood or liquid secretions of those suffering from it.

Clinical History.—The length of the stage of incubation is tolerably uniform. It is ten days to the beginning of the first symptoms, and thirteen or fourteen days to the breaking out of the eruption. These figures have been established by the observations of Panum, the opportunity having been afforded upon the first introduction of the disease into the Faroe Islands. As a rule, there are no especial prodromata during the period of incubation except some slight elevations of temperature. At the end of ten days the initial stage * begins, generally suddenly, and with an abrupt rise of temperature to 102° or 104° (39°–40° C.). At the same time the characteristic catarrhal symptoms appear: nasal catarrh (coryza), to be recognized by the abundant nasal secretion, the frequent sneezing, and sometimes also by nose-bleed; more or less severe conjunctivitis, characterized by photophobia, reddening of the eyes, and increased flow of tears; and, lastly, symptoms of a catarrh of the upper part of the respiratory tract, usually moderate, but nevertheless causing hoarseness and a slight cough. With all this the general condition is disturbed, the children are restless, have headache, and eat little. Symptoms of a mild sore throat are not infrequent, but are very far from being so prominent as in scarlet fever.

These initial symptoms last, as we have said, three or four days. Then the eruption begins (stage of eruption). This is very often preceded for a day or two by a peculiar, usually spotted, reddening of the hard and soft palates, termed "eruption upon the mucous membrane." [Koplik, in 1898, called attention to an eruption of bluish-white specks, surrounded by a reddish area, upon the mucous membrane of the cheek and lips, occurring twenty-four to seventy-two hours before the cutaneous exanthem. Others confirm his observations and regard "Koplik's spots" as a great aid in early diagnosis.—V.] The true eruption of measles begins almost always in the face, on the cheeks, forehead, and around the mouth (contrasting with the characteristic pallor of the chin in scarlet fever), and spreads from there rapidly downward over all the rest of the body. The eruption consists at first of little papillæ, corresponding to the follicles. These are soon surrounded by a pale-red, slightly elevated border, and in many cases become confluent. Perfectly flat elevations, of various sizes and of extremely irregular, dentated, roundish, or angular shape, develop. They are often so thickly crowded together as to touch one another, but usually limited portions of normal skin intervene between them. Within each raised spot the little follicular papillæ remain visible.

With the beginning of the eruption the fever rises, having been, as a rule, slight during the last days of the initial stage. It attains about 104° or 105° (40°–40.5° C.). In thirty-six to forty-eight hours the eruption reaches its full development and its greatest extent. The fever and the catarrhal symptoms also persist for the same length of time. Sometimes we find a slight swelling of all the lymph-glands. Then follows a decline in the fever, usually rapid, and indeed almost by crisis, while the eruption after a short period of full development begins gradually to fade during the two or three days following. At the same time the catarrhal symptoms diminish. A more or less extensive desquamation of the epidermis begins, scarcely ever in large pieces as in scarlet fever, but in little scales, like bran. In eight or ten days, if the disease runs a normal course, the patient is fully convalescent.

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* We consider the term "initial stage" more correct than "prodromal stage." The "prodromal symptoms" are the first slight symptoms which occur during the time of incubation of an infectious disease, while the symptoms presented by measles before the breaking out of the eruption are a part of the already developed disease.

After this brief description of the usual course, we must consider more closely some of the symptoms and possible complications.

The fever (see Fig. 6) of measles exhibits, as has been already implied, a tolerably typical course. It begins with a rather marked and rapid rise upon the commencement of the disease. On the morning of the second day there is usually a marked remission, often to normal. In the last two days of the initial stage the fever is moderate, very rarely being so high as at the beginning. With the eruption there is a new, rapid rise, usually higher than the initial one, so that we may well divide the fever into two periods—the prodromal fever and the eruptive fever. This latter is but brief and does not persist, as in scarlet fever, during the entire duration of the eruption. It falls by crisis when the rash has attained full development. There may, to be sure, be slight elevations of temperature during the next day or two; but, if the fever is considerable and persistent, it is always a sign that complications have arisen, probably in the respiratory apparatus.

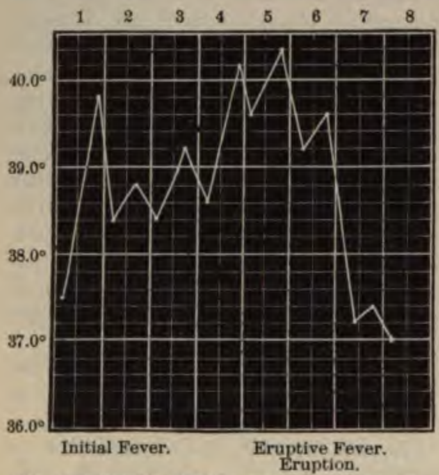

FIG. 6.—Example of the temperature curve in measles.

The eruption usually assumes the form described above, but may present manifold varieties. Sometimes its development is rudimentary. Sometimes it does not begin on the face, but on some other part of the body. This is generally regarded as a sign that the case will be anomalous in other ways as well. The individual spots may be smaller than usual, and may remain entirely separate from each other (*morbilli papulosi*). In other cases the eruption is so confluent (*morbilli confluentes*) that it resembles the eruption of scarlatina. The formation of vesicles (*morbilli vesiculosi*) also occurs, but much more rarely than in scarlet fever. Hæmorrhagic measles is also observed, but usually only in the form of small, capillary bleeding, and in cases that otherwise run a perfectly favorable course. Very rare cases have indeed been described, with a general hæmorrhagic diathesis and severe symptoms, resembling hæmorrhagic scarlatina. It is doubtful whether the "black measles" of the old writers was actually measles at all. In addition to the proper eruption of measles, other eruptions sometimes develop—such as vesicles, wheals, and pustules.

The pulse is generally not so rapid in measles as in scarlet fever. Enlargement of the spleen can be made out only to a slight degree, if at all. As a rule, we find no pronounced leucocytosis in the blood.

The **complications of measles** are for the most part exaggerations or abnormal varieties and extensions of those troubles which are observed during the usual mild course of the fever. As in scarlet fever (*vide supra*), we often have to deal with the effects not of the original, but of secondary infection. Compared with the great majority of mild attacks taking the typical course, cases presenting complications of any severity are rare, and much less frequent than in scarlet fever. Epidemics are only now and then distinguished by unusual severity.

Often quite serious eye-diseases are developed, particularly blennorrhagic conjunctivitis, keratitis, and iritis.

Marked inflammation of the mucous membrane of the nose, throat, and larynx may prolong the course of the disease. This is often merely an exaggeration of the usual catarrh. Otitis media likewise sometimes occurs. A laryngitis of marked intensity, with considerable swelling of the parts involved, may produce much discomfort, or even symptoms of stenosis ("false croup"). Actual croupous and diphtheritic lesions of the throat and larynx also occur (diphtheria of measles). This last is indeed much rarer than scarlatinal diphtheria, but it may have the same unhappy termination. It is worth mentioning that sometimes genuine laryngeal croup is observed in measles, unaccompanied by lesions of the pharynx. In all these affections secondary infection plays the chief part, as it does in scarlet fever (*vide supra*, page 41).

It is, however, in the lungs that the most frequent and important of all complications in measles occur. The usual mild bronchitis becomes very intense, extends into the bronchioles (capillary bronchitis), and then results, for the most part, in a more or less extensive, lobular, catarrhal pneumonia (*q. v.*). According to Köster, measles pneumonia rises from an inflammatory proliferation of nuclei about the small bronchi, and it usually invades the neighboring alveoli by continuity along the small branches of the pulmonary artery. It is then a "small nodular," but not strictly a lobular pneumonia. Measles pneumonia is to be diagnosticated when moist râles are heard in abundance over a large part of the chest, and when there is at the same time persistent fever and pronounced difficulty in respiration, with cough or dyspnœa. We get decided dullness on percussion only when the separate centers of infiltration are more than usually confluent. Genuine lobar, croupous pneumonia appears much less often than the lobular variety. It attacks one lobe, or several, is attended by high fever, and may end with a well-marked crisis.

The foregoing pulmonary symptoms usually appear at the height of the disease, and persist after the eruption fades. They may delay convalescence for weeks. In other cases measles will be seen at the start to run a normal course, the temperature will have already fallen, and then come new fever and the appearance of decided pulmonary disturbance. This is always to be regarded as a grave complication; and especially in feeble children it may lead to death, with the symptoms of impaired respiration or of constitutional exhaustion. At present we can not definitely state how far in measles pneumonia we have to do with the direct action of the measles poison itself or with secondary infection (streptococci, diplococci, etc.).

Marked intestinal symptoms sometimes appear, particularly an excessive diarrhœa, due to intestinal catarrh. It is characteristic of measles that in severe cases such a diarrhœa may assume a pronounced dysenteric character, indicated by blood and slime in the dejections, symptoms which usually depend upon the development of follicular colitis with ulcerations.

Now and then still other complications may present themselves, of which a full enumeration is impossible. Nephritis does occur, but far less often than in scarlet fever. A simple albuminuria during the acme of the disease is not infrequent, but as a rule it has no especial clinical significance. We should mention gangrene of the cheek, the so-called noma, as a complication, which is very rare but apparently characteristic.

Peculiarities in the course of the disease are much rarer in measles than in scarlet fever. Yet we see, on the one hand, unusually mild or rudimentary cases, in which either the rash or the other local symptoms are remarkably slight, and on the other hand, abnormally severe cases. These latter are distinguished by the unusual height or persistence of the fever, by the severe constitutional and nervous symptoms, and further by the early appearance of complications. Such cases have been termed "typhoid measles." We have already mentioned the severe form of hæmorrhagic measles.

We should notice the clinical relation which measles bears to two other infectious diseases—to whooping-cough and tuberculosis. Measles and pertussis (*q. v.*) may follow each other in the same individual at a short interval, sometimes one and sometimes the other taking the initiative; epidemics of the two diseases prevail with comparative frequency at the same time. Tuberculosis is likewise to be mentioned as an important sequela of measles. In these cases it is probable that the children have a tubercular focus smoldering somewhere in the body (lymph-glands, lungs) before the attack of measles which kindles it and aids its further extension. The measles may also, of course, excite a predisposition to infection with tubercle bacilli.

The **diagnosis** of measles, as of the other acute exanthematous diseases, is based chiefly upon the eruption, but we must also regard, of course, other symptoms which may be present (fever, catarrhal symptoms). Personal experience does more to sharpen the perception than can the fullest descriptions. We can merely suspect the disease during the initial stage unless an epidemic prevails. If, beside the characteristic catarrhal symptoms, the above-mentioned eruption on the mucous membrane of the palate exists, the diagnosis becomes tolerably certain. We should consider that eruptions similar to that of measles appear in other diseases, more especially in rötheln, scarlet fever, typhus fever, in the beginning of small-pox, and sometimes in secondary syphilis. Furthermore, we need to exclude eruptions due to drugs, especially antipyrine, and also turpentine, balsam of copaiba, etc. In doubtful cases we shall be enabled to form a decided opinion by the other symptoms, and, above all, by the further course of the disease.

Prognosis.—We have already remarked how favorable in general the prognosis is, but we must here repeat that all epidemics do not exhibit the same benign character, and that in every case the physician must bear in mind the possibility of complications, and particularly the danger of severe pulmonary disturbances.

Treatment.—The patient should in general be kept somewhat warmer than in scarlet fever. Even in what seem to be the mildest cases the child should be kept in bed till desquamation is over. The sick-chamber is to be somewhat darkened, on account of the photophobia which usually exists at first. In this way, normal cases run on favorably without any especial therapeutic interposition. The catarrhal symptoms, however, should always be heeded, since to disregard them may lead to their becoming aggravated. The chief requisite is cleanliness. At regular intervals the eyes, the nasal cavity, and the mouth should be washed out with lukewarm water.

If, despite all this, certain disturbances appear in a worse form than usual, or if complications develop, these must receive especial attention. Severe eye troubles should be treated according to the usual ophthalmological practice. The treatment of croupous trouble in the throat or larynx will be fully described in a later chapter. For the pulmonary complications, lukewarm baths, combined if need be with rather cool douches, constitute the most effectual remedy. Cold packs are also very efficient. These we shall speak of later in more detail in the chapter on catarrhal pneumonia. The chief task of the physician in the treatment of measles is, however, to hinder as far as possible the appearance of complications. We are not acquainted with any internal remedies for the lung troubles which are at all trustworthy. In rare instances the excessive accumulation of mucus in the bronchi requires the administration of an emetic. As expectorants we may try ipecac, liquor ammonii anisatus, etc. If considerable intestinal disturbance arises, we must employ small doses of opium, calomel, tannalbin, or subnitrate of bismuth. We hardly need to say that, whatever else is done, the strength of the patient should be kept up as much as possible by giving wine, broths, milk, eggs, etc. For at least two or three weeks after the disease has ended, the child must be very carefully watched.

As the disease is usually so mild, prophylaxis is not very strenuously attempted. If one child in a family is attacked, it is probably already too late to isolate the others, and it is even an advantage to the family to have all the children finish at once what they will hardly be able eventually to avoid. We would make an exception in favor of isolation if the disease prevailed in a severe form.

[It is not customary with us to insist so strongly upon isolation and thorough disinfection as in scarlet fever, but the tendency of the present day is toward a wide application of the principles of preventive medicine. It is certainly of no advantage to a child to contract measles. Delicate children, especially those with tubercular predisposition, should be carefully guarded against it; and, even if it is decided that it is not worth while to attempt to confine the disease to one member of a family, every precaution should be taken against infecting other families. Under suspicious circumstances, consequently, children are to be kept away from school and from contact with others.

If there is any reason to fear the development of tuberculosis, every possible hygienic means should be employed in order that full vigor may be regained.]

CHAPTER VI

RÖTHELN

(German Measles)

Rötheln is a disease similar to measles, but distinct from it, although formerly often confounded with it, and perhaps with scarlet fever as well. The observations of Steiner, Thomas, and others leave now no room to doubt that these diseases are distinct, for epidemics occur in which all cases present the characteristic peculiarities ascribed to rötheln. But the best proof is that children who have had rötheln are not infrequently attacked by genuine measles later. It may indeed be very difficult in an individual case to decide which disease is present; but that rötheln does exist, as an independent form of disease, can be denied by those alone who have never seen it.

After an incubation of about two or three weeks the disease begins with the appearance of the eruption, which is usually noticed first in the face. Initial symptoms (cough, sneezing, etc.), preceding the eruption, either are wholly absent or at most last for half a day. The eruption is decidedly like that of measles, but its individual spots are smaller. They are seldom larger than small peas and circular, being only exceptionally as dentated and irregular in outline as are the maculæ of measles. They appear on the whole face, the head, the trunk, and the extremities, are pale red (sometimes deep red), only slightly elevated, and are not apt to become confluent. In rare instances, small vesicles develop upon the macules. The soft palate sometimes exhibits, as in measles, a faint macular eruption at the beginning of the disease. After two to four days the eruption fades. There is usually no decided desquamation.

Other symptoms of disease than this eruption are slight. Fever in many cases appears to be entirely absent. As a rule, there is for a day or two a little elevation of temperature, reaching 102° (39° C.) at most. Tokens of a moderate catarrh of the conjunctiva, the nasal mucous membrane, the throat, and the larynx are also observed—viz., photophobia, nasal discharge, and cough. Often, the cervical lymph-glands are more or less swollen. The constitutional disturbance is generally so slight that the child can hardly be kept in bed. Important complications hardly ever occur.

The prognosis of rötheln is therefore perfectly favorable, and the employment of any special treatment is needless.

CHAPTER VII

SMALL-POX

(*Variola. Varioloid*)

Ætiology.—Small-pox has been known for centuries, although formerly often confounded with other diseases.* It is one of the most dreaded acute infectious diseases, and in earlier times it has destroyed thousands in its pestilential progress. It was the discovery of the possibility of prophylactic inoculation, and the ever-increasing spread of this precautionary measure, which first robbed the disease of some portion of its terrors.

Numerous statements have been made about the occurrence of micro-organisms in the variolous eruptions on the skin and mucous membranes, but we are compelled to say that we are not yet acquainted with the specific, organized poison of small-pox, however strongly justified we may be in assuming its existence. Bacteria can in fact easily be demonstrated in the eruption of variola, but most of them come from the surrounding atmosphere, and have no relation to the specific variolous processes. The foci of bacteria found in the internal organs (liver, spleen, kidneys) are also due, as their discoverer, Weigert, himself supposed, to the secondary ingress of other varieties of micro-organisms, and are not directly associated with the variolous process, the diseased condition of the skin furnishing a ready entrance for infectious matter.

Predisposition to variola, except as diminished by vaccination (*vide infra*), is universal. The disease may appear at any age, even *in utero*. Women are believed to be especially liable to it during pregnancy and child-bed. It is said that persons ill with another acute infectious disease, such as scarlet fever, measles, or typhoid fever, are, for the time being, tolerably secure from infection with small-pox; but this rule certainly has exceptions. The same individual rarely takes the disease a second time.

A case of variola is always the result of transmission of the poison to a healthy person from one who is already ill with it. The specific poison is most abundant in the diseased portions of the body and in the pus of the suppurating pocks, as well as the crusts and scales which are left when these have dried up; but the disease is also contagious in its earlier stages, before the pustules develop, and even, according to a few observations, during the stage of incubation. Certainly the variolous poison is very volatile—that is, it is prone to disseminate itself through the air in the neighborhood of the patient. In order to catch the disease it is not necessary to touch the patient, but merely to remain in his vicinity. In many cases we can not, however, determine with exactness the mode of transmission, since the contagion may either be direct or by means of objects and utensils with which a patient has come in contact—for example, the soiled linen. The dead body also is capable of transmitting the disease. In general, numerous instances point to a considerable "tenacity" in the poison. The precise manner of infection is not yet known. It is most probable that the poison is drawn into the lungs with the inspired air.

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* The very name small-pox (*petite vérole*) is significant of its confusion with syphilis, which was termed the "great pox."

It has been demonstrated that the disease can be transmitted to healthy persons by direct inoculation of the contents of the variolous pustules. It is stated that monkeys and other animals may be successfuly inoculated in the same way. Whether inoculations with the blood of the sick will reproduce the disease is not yet settled. The secretions (saliva, sweat, urine, and milk) do not apparently contain the infectious matter.

Course of the Disease. Variola and Varioloid.—The stage of incubation lasts as a rule thirteen to fourteen days, sometimes a somewhat shorter time. During this period prodromal symptoms are absent or insignificant.

The disease itself begins suddenly with what are usually very characteristic initial symptoms—rigor, fever, headache, and intense pain in the loins. It is only in comparatively few cases that one or another of these symptoms is slight or wanting. The constitutional symptoms may be very severe—a dry tongue, stupor, wakefulness, delirium. The fever continues intense for some days. The pulse is much quickened. There is almost total anorexia, and often there is vomiting. There is constipation, or, more rarely, diarrhœa. Frequently there is a slight sore throat, and sometimes a slight bronchitis. The spleen is enlarged in most of the severe cases, and the urine often has a trace of albumen. In women, menstruation occurs in a remarkably large number of cases. The proper variolous eruption does not at once appear, but from the second day other characteristic efflorescences are not rare. These are termed the initial rash of small-pox. We may find either a diffuse or macular erythema, extending in varying degree over the trunk and extremities (especially on the extensor surface), or a hæmorrhagic eruption with small spots, appearing by preference upon the hypogastrium and the inner surface of the thighs (in the so-called femoral triangle of Simon) or on the lateral surface of the trunk and on the upper arm. The erythema soon disappears, but the hæmorrhagic spots can be seen longer. Both forms of the initial eruption may be combined.

The initial stage, just pictured, lasts usually three days. Severe symptoms occurring at this time do not exclude the possibility that the further course of the disease may prove favorable, while mild symptoms are of good omen.

At the end of the third or on the fourth day the temperature makes a decided fall, and the regular variolous eruption begins to be developed upon the skin—the *stadium eruptionis*. During this period an evident difference among the separate cases becomes manifest. This distinction can not indeed be always drawn with a narrow line, but it is noticeable enough to justify the establishment of two types of variolous disease. We refer to the division into a severe form (variola vera), and another, mild form (varioloid). The variola proper has a well-developed eruption with many pustules, and, as a result of this, a second stage of fever (*stadium suppurationis*). Varioloid has a much more scanty eruption, and little or no suppurative fever. We must now discuss these two forms separately.

VARIOLA VERA

The eruption almost always begins in the face and upon the hairy scalp, appearing somewhat later on the trunk and arms, and last of all upon the legs. It begins in the form of little red dots and spots, which develop in about two days to small papules (*stadium floritionis*). If the hand be passed over thickly set and well-developed papules of variola, a peculiar soft, satin-like feeling is perceived. On the points of these papillæ a little vesicle forms. This keeps growing larger and larger, its contents becoming turbid and purulent, till at last, on the sixth day of the eruption and the ninth of the disease, the development of the genuine pustule of variola is complete (*stadium suppurationis*). The pustule usually presents upon its summit a little dimple (umbilication), and is surrounded by a red border or "halo." Where the pocks are especially close together, as in the face, the skin between them is diffusely swollen, and the consequent burning and pain are excessive. The countenance becomes much disfigured. Often the eyes can not be opened because of the œdema. The hands also are apt to be intensely affected, especially the back of the hands, and also all parts which have previously been injured in any way (pressure or friction of clothing, etc.). On the trunk the pustules are very rarely as close together as on the face and hands. It is worthy of note that often new eruptions of pustules keep appearing for two or three days.

At the same time with the eruption upon the skin, or even somewhat earlier, a perfectly analogous efflorescence develops upon the mucous membranes. The chief places for its appearance are the mouth and throat, the tongue, the soft palate, the nasal cavity, also the larynx, the trachea, and the upper part of the œsophagus. In the vagina and rectum it is rare and scanty. In this mucous efflorescence, however, there are no proper pustules, but small, superficial ulcers. These result from the maceration of the uppermost layers of the mucous membrane. They sometimes become confluent. The annoyance produced by this eruption in the mouth and throat is, of course, very great. The pocks in the larynx manifest themselves by hoarseness, and occasionally by symptoms of stenosis.

As we have said, the beginning of the eruption is the signal for a noticeable fall in the temperature; but in true variola the fall does not reach the normal, or only temporarily. The other symptoms likewise remit, especially the headache and lumbar pain. When, however, the suppuration begins, the fever rises once more, and there are fresh symptoms of constitutional disturbance. This is the time for the dreaded attacks of delirium, during which the patient must be vigilantly watched, lest some untoward event happen. Now, too, complications may arise (*vide infra*).

On the twelfth or thirteenth day of the disease the pustules begin to dry up (*stadium exsiccationis*). The purulent contents of the pustules, part of which have burst, form yellow crusts, the swelling of the skin subsides, and, a few days later, the crusts and scabs begin to fall off. With the beginning of desiccation, the fever declines; the local as well as the constitutional symptoms become daily slighter, and convalescence follows. The healing of the pustules is frequently accompanied by an extremely troublesome itching. After the scabs have been cast off, the skin presents pigmented spots, which persist for months. Wherever the cutis vera has itself been destroyed by the suppuration, a scar is inevitable. Thus arise the familiar scars (pitting) of small-pox, which continue visible through life. Very often, after the end of the disease, there is almost complete alopecia. The hair often grows again, but not always.

VARIOLOID

The distinction between varioloid and variola vera is not in kind, but in degree. Varioloid is only a milder form of variola. There is, as we have already said, no sharp boundary-line between the two. Varioloid is most often observed in those whose susceptibility to the variolous poison has been diminished by vaccination (*vide infra*).

As above mentioned, the behavior of the disease during its initial stage will not permit us to decide positively whether variola or varioloid will be developed. It is true that if the symptoms be especially mild, we may guess that it will be varioloid; and, likewise, the appearance of the initial erythema already spoken of is regarded as a favorable omen.

Shortly after the pocks begin to appear, the decision can almost always be made with certainty. In varioloid the eruption is rather scanty. It is often irregular, and does not by any means always begin, like that of variola, in the face, but often on the trunk. The individual pocks are in no way different from those of variola; but it often happens that they do not pass through all the regular stages to full suppuration, but undergo resolution before this occurs. Such cases, in which there is nothing beyond papillæ or vesicles, are sometimes spoken of as *variolois verrucosa seu miliaris.* The scantiness of the eruption and the limited amount of suppuration have for their corollary an absence, or at least a very slight development, of the suppurative fever.

When the eruption appears the temperature usually falls by crisis to the normal level and remains there. The desiccation may begin as early as the eighth or tenth day of the disease, so that the whole duration of varioloid is considerably shorter than that of variola. Grave complications are very exceptional. The pocks may develop upon the mucous membranes, but here, too, they are scanty and not very vigorous.

COURSE OF THE FEVER, SYMPTOMS PRESENTED BY SEPARATE ORGANS, AND COMPLICATIONS

1. *Fever* (*vide* Fig. 7).—In the initial stage, as we have said, the temperature rises rapidly as a rule, with a pronounced rigor; and during the first days it very often reaches 104° to 106° (40°–41° C.). It sinks on the third to the sixth day, when the first papillæ develop, and then, in the case of varioloid, falls rapidly to normal, and remains there. In variola the decline is slower and less complete; and with the beginning of suppuration the temperature begins to rise again. The violence of this suppurative fever is usually in direct proportion to the severity of the eruption. It has manifold fluctuations, but seldom lasts, in severe cases, less than a week. Temperatures of 104° (40° C.) and higher are common. The fever declines by lysis. In case of approaching death, the temperature may be extremely high, even reaching 108° or 109° (42°–43° C.).

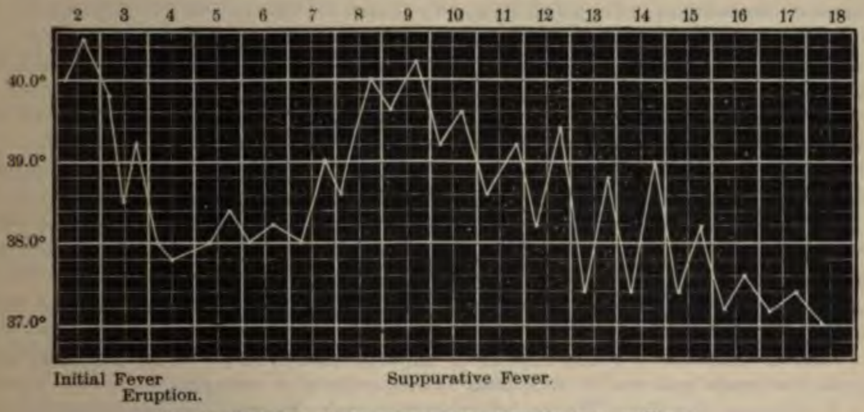

FIG. 7.—Example of the temperature curve in true small-pox.

2. *Skin.*—We have already described the macroscopic appearance of the eruption. It remains to mention briefly the histological phenomena. The first demonstrable changes are in the cells of the deeper layers of the rete Malpighii. As a result of the variolous infection, the cells perish, are swollen by the lymph which escapes from the papillary blood-vessels, and transformed into flaky, homogeneous masses without nuclei ("coagulation necrosis" of Weigert). The lymph becomes more and more abundant, and crowds the cells farther and farther apart. These are thereby finally changed into threads and membranes,

forming a distinct net-work in the vesicle. This explains why, if such a vesicle be pricked, its entire contents are never discharged at once. Great numbers of white corpuscles escape, along with the lymph, from the blood-vessels, and finally render the contents of the original vesicle purulent. Proliferative processes occur in the surrounding epithelial cells, which are still intact, and thus the margin of the vesicle becomes elevated, while the dead portion in the center sinks in. Thus the pock becomes umbilicated. If a portion of the papilla itself suppurates, a scar must be left on healing. If the process remains limited to the epithelium, complete regeneration takes place, and the skin reassumes its normal appearance.

Certain secondary complications, which sometimes attack the skin, remain to be mentioned: abscess, phlegmon, erysipelas, gangrene, and bed-sores. None of these are due directly to the specific variolous intoxication.

3. *Respiratory Organs.*—The disturbances here are in part symptoms of the specific process of the disease, and in part secondary. The frequent occurrence of secondary symptoms in small-pox is easy to understand (compare the chapter on lobular pneumonia). Of the primary symptoms, we should mention genuine pocks in the larynx, the trachea, and the larger bronchi. As sequels to these, more or less severe secondary disorders are very frequent: laryngeal ulcerations, which may even lead to laryngeal perichondritis and œdema of the glottis; diffuse bronchitis; lobular pneumonia, often of great extent, due to the inhalation of solid matter into the lungs, and frequently accompanied by pleurisy. It should be especially noticed that lobar, croupous pneumonia is not rare. Whether this be likewise secondary, or a direct result of the variolous poison, is not yet known.

4. *Digestive System.*—The genuine pocks often develop, as stated, in the mouth and pharynx, and likewise in the upper part of the œsophagus. They are not observed in the mucous membrane of the stomach or intestines. The active diarrhœa sometimes seen depends upon catarrh of the intestine. Dysentery is rare. The eruption in the mouth and throat may result in severe secondary troubles, purulent otitis, parotitis, pharyngeal diphtheria, etc. The spleen is almost always considerably enlarged, and often the liver also, but in a less degree.

5. *Circulatory System.*—Pathological changes in the heart are rare, if we except the slight parenchymatous degeneration of its muscular fibers, common to almost all severe infectious diseases. Sometimes there is a slight endocarditis (*q. v.*), which is probably secondary. Pericarditis is rather more frequent.

6. *Organs of Special Sense.*—Genuine variolous pustules occur upon the eyelids and the conjunctiva. Later in the disease there may be keratitis, iritis, or choroiditis.

We have already mentioned the relative frequency of aural disturbances, particularly purulent otitis media.

7. *Articular swelling* may appear in the suppurative stage. The shoulders and knees are most apt to be attacked. Periostitis also occurs.

8. *Nervous System.*—We find no pathological changes corresponding to the severe nervous derangements manifested during the disease. After the small-pox is over, spinal diseases sometimes occur, with either paralysis or ataxia. Westphal has shown that they are caused, in some cases, by numerous disseminated centers of inflammation in the spinal cord. Encephalitic processes have also been observed in small-pox, and in some cases neuritic paralysis.

9. *Albuminuria* is quite common in severe attacks, but genuine nephritis is a very rare complication. In pregnant women attacked by small-pox abortion or premature labor is very apt to occur. If a living child be born, it usually dies soon after birth.

Anomalies in the course of the disease are manifold. We will not speak of the two typical forms already considered. There are abnormally mild cases, with scarcely any initial symptoms, or with an obscure eruption, or with no eruption at all (*febris variolosa sine exanthemate*). In such cases a correct diagnosis is possible only at the time an epidemic prevails, and by the aid of the attendant ætiological circumstances. There are also abortive cases in which the first symptoms are severe, but which recover with remarkable rapidity.

The abnormally severe cases are more important. First, there is the confluent variety. This is merely the typical process in its completest development. The initial symptoms are themselves generally very severe, and are followed, without any considerable remission of the fever, by the eruption of hundreds of pustules. The skin of the face and hands is one continuous area of suppuration. The local discomfort is extreme, as is also the intensity of the fever and of the constitutional symptoms. The nervous system suffers most. There is at the same time an unusually abundant eruption upon the mucous membranes. The occurrence of the above-mentioned complications affecting the various organs of the body is frequent. Death is a common result; or, if recovery takes place, it may be delayed by tedious sequelæ.

Hæmorrhagic small-pox is the worst anomalous form. The name is applied to several different varieties. In the first place, any variolous eruption may become more or less hæmorrhagic, and yet the general course of the disease not be essentially altered. Such cases are more common among elderly people, cachectic persons, and drunkards. Secondly, there is a very severe form of small-pox, which is generally quickly fatal. The initial stage is marked by the unusual severity of the symptoms. The abundant eruption soon becomes hæmorrhagic, and there are also ecchymoses in the mucous membranes and the internal organs. This has been called black small-pox, and by Curschmann *variola hæmorrhagica pustulosa.*

There is another form of hæmorrhagic variola, different from these but linked to them by transitional varieties. In it the acute hæmorrhagic diathesis develops during the initial stage. Death almost always occurs before the regular variolous eruption. This most frightful form is usually termed *purpura variolosa.* That it is small-pox is proved by its ætiological relations alone. Otherwise it would be impossible to distinguish it from certain other acute septic disorders. It is prone to attack the youthful and vigorous. Chills, headache, and pain in the loins are the first symptoms, just as in ordinary cases. Cutaneous ecchymoses appear as early as the second or third day. They increase in area so rapidly that one can almost see them grow. They are most extensive in the hypogastric region. There are also ecchymoses in the eyelids, the conjunctiva, the mouth and pharynx, and, as the autopsy discloses, many in the internal viscera. The constitutional symptoms are most severe, and the patient seldom survives the fifth or sixth day of the disease.

Diagnosis.—The certainty with which we can make the diagnosis of small-pox in any well-developed case is equaled by the difficulty of deciding about it during the beginning of the disease, or even during the beginning of the eruption. At this period diagnosis may be impossible. When the variolous eruption is in process of development, it may be confounded with typhus fever, with that form of measles in which the papillæ are prominent, with syphilitic eruptions, and with certain forms of erythema exsudativum, just breaking out. We can not here fully discuss all the factors which should be considered in making this diagnosis. It is important not to regard the cutaneous appearances alone, but to note all the other symptoms besides. But it is often necessary to watch a doubtful case for some time before a diagnosis can be established.

Prognosis.—The facts which are of greatest weight in prognosis have already been emphasized. We may repeat that during the initial stage the prognosis of any individual case can seldom be determined. If the first symptoms are mild, or if the initial erythema appears, the case is regarded hopefully. The abundance of the eruption has an influence upon the severity of the disease. Circumstances peculiar to the individual are also important—e. g., age, constitution, or alcoholic habits. We have already called attention to the danger of confluent small-pox, and to the almost absolutely fatal prognosis in the genuine hæmorrhagic variety. The mortality varies greatly in different epidemics; on the average it may be taken at about fifteen to thirty per cent. Beyond doubt, the introduction of vaccination has decidedly lessened the fatality of the disease by diminishing the frequency of the severe forms.

Treatment.—1. *Prophylaxis—Vaccination.*—As in all contagious diseases, isolation is of little avail unless complete. This fact has led to the erection in late years of small-pox hospitals. All utensils used by the patient, and his clothing, bedding, and the like, should be most carefully disinfected. The best method is to employ a high degree of heat—viz., 240° to 250° (115°–120° C.).

These precautionary measures are employed in many other diseases as well, but for small-pox we are acquainted with a special method of prophylaxis. It is founded upon a fact which is the most remarkable within the domain of the infectious diseases. We refer to prophylactic vaccination. It must long ago have been remarked that a person who has had the disease once, enjoys, to a large degree, immunity from any fresh infection. This suggested the idea of exposing children purposely to contagion, so as to insure them from small-pox for the rest of their lives. The actual inoculation of small-pox is said to have been long practiced in India and China. In the year 1717 it was employed by Lady Mary Wortley Montagu, of England, upon her own son, and with success. Unfortunately, however, the inoculated small-pox proves fatal in many instances; and, being itself contagious, it serves to spread the disease still further. Then appeared an article written by the English surgeon, Edward Jenner, in 1798. This informed the medical profession of a fact already known to the rural population of his native place, but which Jenner first established scientifically, and recognized in all its importance. There sometimes occurs a disease similar to small-pox upon the teats and udder of the cow, called *variola vaccina.* It is apparently a local trouble, and it can easily be inoculated upon the skin of human beings. Vaccine pustules will be developed upon the spot inoculated. These almost invariably heal without any great constitutional disturbance; but the person vaccinated possesses the same immunity from small-pox as if he had had small-pox itself. This statement of Jenner's was soon confirmed upon every side. The result is the continually spreading custom of prophylactic vaccination. In some countries it is enforced by law, and it can be opposed only by ignorance or by lamentable prejudice.

To explain exactly how vaccination can protect against small-pox in this way is still beyond our powers; but vaccination has at present lost much of its former enigmatical character, since we know that a similar immunity can be produced in almost all infections by inoculation with attenuated or altered virus (see the chapters on malignant pustule, diphtheria, tetanus, pneumonia, etc.). This analogy has become the more certain since there is no longer any doubt, according to recent observations (Fischer and others), that vaccinia is, in fact, only a form of small-pox rendered milder and modified by transmission to animals. If we inoculate a calf with the contents of a fresh small-pox vesicle from man, typical vaccinia is produced. With the contents of this vaccine vesicle children can be inoculated without any danger, and they are no longer susceptible either to vaccinia or small-pox.

We can mention only the most important of the details relating to vaccination and the method of its performance. Only exceptionally do we vaccinate with the contents of a human vaccine vesicle ("humanized lymph"), but we almost invariably use animal lymph from vaccine vesicles of calves. This is now obtained in large quantities and put on the market. The process of vaccination is to make three shallow incisions, 3 or 4 centimetres apart, in the skin of the upper arm, and to introduce the vaccine-lymph into them. The surrounding tissue becomes swollen in three or four days. In seven or eight days the vaccine vesicles are well developed, if the disease takes its normal course. Next they become purulent, and then dry up, and finally, on healing, leave the familiar scar behind. The whole process occupies about three weeks. If the vaccination fails, or is but partially successful, it must be repeated after a few months. The protective power of vaccination does not last indefinitely, and therefore re-vaccination is necessary every five or six years. The first vaccination of children usually takes place when they are three or four months old. If they are feeble we wait longer, unless small-pox is prevalent.

It must be confessed that vaccination is not without its dangers. The little cutaneous wound made by it may lead, like any other, to sepsis or to erysipelas. The latter has been called vaccination-erysipelas. But such misfortunes are extremely rare. The not uncommon "vaccine roseola" deserves especial mention. It appears first upon the arm vaccinated, and spreads over the rest of the body; but it is not a serious matter. It is of course possible, by especially bad luck in the use of human lymph, that other diseases, among which syphilis is of chief importance, may be inoculated along with vaccinia; but this is a very rare occurrence—much more so than the enemies of vaccination pretend. If the physician exercise proper care in the selection of the person from whom to take humanized virus, it can be entirely avoided.

[The incubation stage of vaccinia being shorter than that of small-pox, the prompt vaccination of an unprotected individual who has been exposed to infection should always be practiced, if possible; oftentimes the severe disease may thus be prevented.]

2. The treatment of small-pox is purely symptomatic. When the disease has once begun it is too late for vaccination to have any influence upon its further course. During the initial period we may advantageously employ cool baths to diminish the fever and alleviate the constitutional symptoms. An ice-bag will relieve the headache. We must not let the lumbar pains lead us to any but a cautious use of local irritants, for the pocks come out in greater abundance upon such portions of the skin as have been in any way irritated. If the disease proves, during the stage of eruption, to be varioloid, there will be no further need of special treatment. Good nursing and proper food will suffice.

The true small-pox, on the other hand, demands the interposition of the physician. He must strive to guard the regular course of the disease in the skin and in those portions of the mucous membrane which are accessible from being disturbed by secondary inflammations; for we have no doubt that the ruptured pustules furnish a most easy ingress to septic impurities from the surrounding atmosphere, so that later, when there is extensive suppuration of the skin, or analogous and severe disturbance in the mucous membrane, it is impossible to discriminate between the effects of the small-pox itself and those due to the secondary suppuration. If we were able to have the whole process go on "antiseptically" we should certainly have made an important advance in therapeutics. Indeed, the methods of treatment which have been up to this time recommended fulfill this indication up to a certain point, e. g., painting the skin with tincture of iodine, or with a strong solution of nitrate of silver—methods formerly much in vogue. Schwimmer's suggestion seems still better. He recommends, from the beginning of the eruption, the use of a paste made as follows: Acid. carbol., parts 4 to 10; ol. olivæ, 40; cretæ præparat., 60. M. et ft. pasta mollis. This is spread on pieces of old linen and laid upon those parts where the eruption is apt to be worst—viz., the forearm, hand, and leg. The face is covered with a mask, having holes corresponding to the mouth, nose, and eyes. The applications are changed every twelve hours. Under this treatment the local distress is said to be diminished, suppuration slight, and healing comparatively rapid. The pain and sense of tension in the skin are often relieved by cold applications, or by simple ointment or oil. Under Hebra, in Vienna, continuous warm baths were employed in severe cases with great success.

The treatment of the affected mucous membrane in small-pox must also meet the indication above mentioned. The most thorough disinfection of the mouth and pharynx must be aimed at. The means to be used are careful washing and gargling with solutions of chlorate of potash (1 to 30), carbolic acid, borax, permanganate of potash, or liquor ferri chloridi. The eyes, if they need it, must also be appropriately treated. As to all other complications, cool baths are relatively the most useful remedy. They can be given without difficulty. The chief indications for them are severe pulmonary or nervous symptoms, or continuous high fever. Internal antipyretics, such as quinine or antipyrine, are also employed. Violent nervous disturbances, such as delirium, sometimes require the cautious use of narcotics. There is nothing to add as to the treatment of malignant hæmorrhagic small-pox, for, as we have said, such cases are unfortunately almost hopeless.

CHAPTER VIII

VARICELLA

(Chicken-pox)

Varicella is truly one of the diseases of childhood. Adults very rarely have it. It is contagious, and often comes in epidemics.

The stage of incubation does not last over thirteen to seventeen days. The disease begins with the appearance of vesicles, the size of a pea or a little larger, usually having a small red areola, and varying in number from ten to one hundred or more. The trunk usually bears the greater part of the vesicles, while the extremities have few. The face is frequently the seat of a considerable number, and sometimes there are a few upon the hairy scalp. There may be a vesicle here and there upon the mucous membrane of the mouth or palate. There are seldom any prodromata. Slight symptoms of fever may attend the eruption itself. The eruption is usually over in a few days, although there may be repeated crops, so that we often see fresh vesicles by the side of others which are drying up. Each individual vesicle heals quickly, and the pustulation seen in small-pox is here exceptional. The course of the disease is completed in a week or ten days. Most children feel perfectly well the whole time, although there may be in rare cases pain in the limbs, anorexia, and a slight coryza. A severe complication is hardly ever seen. An unusual event is a mild nephritis.

Exceptionally, the disease may be rudimentary, with a varicelloid roseola and no formation of vesicles. On the other hand, some cases present quite severe constitutional symptoms and a high fever, even reaching 105° (41° C.) temporarily. In most cases, however, as we have said, the child is so slightly disturbed that a physician is hardly thought necessary.

The **diagnosis** is almost always easy. Formerly varicella was often confounded with small-pox, and to this day the followers of Hebra, in Vienna, for some inconceivable reason, maintain the identity of the two. That they are essentially distinct is shown (1) by the epidemics of the two appearing entirely separate from each other, (2) by the fact that having one does not give immunity from the other, and (3) by the uniform failure of attempts to produce variola by inoculating varicella, or *vice versa*. Still, we must bear in mind, in order to avoid mistakes, that many dermatologists class the mildest cases of small-pox as varicella. Those who devote themselves to general diseases are probably all now convinced that varicella is a separate disease.

The **prognosis** is perfectly good. There is usually no special treatment necessary, but young children should be kept in bed till the eruption has dried up.

CHAPTER IX

ERYSIPELAS

(*St. Anthony's Fire*)

Ætiology.—Erysipelas is a peculiar inflammation of the skin, recognized by redness, swelling, and pain. It has the peculiarity of spreading gradually, by direct extension, from its point of origin over a larger or smaller portion of the body. The cause of this inflammation, as was first shown by Fehleisen, is a local infection by the streptococcus pyogenes and its further extension in the skin. The exciting agent of erysipelas is then bacteriologically identical with the streptococcus which is the cause of phlegmonous suppuration, severe septic infection, etc. The reason why in the individual case erysipelas develops, and not some other streptococcus disease, is the special variety of infection and the further spread of the streptococci in the deeper lymphatics of the skin, and in part perhaps the special "virulence" of the infecting germs.

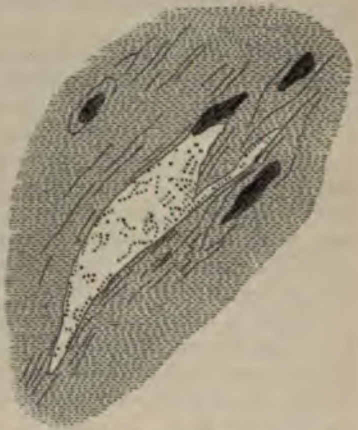

FIG. 8.—The cocci of erysipelas. ×700. Section through a lymph-vessel in the skin.

The former distinction between "traumatic" and "idiopathic" or "exanthematic" erysipelas can no longer be rigidly maintained. Every erysipelas is in a certain sense traumatic, since infection with streptococci can not occur through the unbroken skin, The so-called "idiopathic" erysipelas is distinguished from "wound erysipelas" (in which, of course, we include puerperal erysipelas, erysipelas of the newborn starting from the umbilical wound, etc.) only by the fact that in the former the infection takes place in small, insignificant wounds of the skin which are easily overlooked. Such an erysipelas occurs chiefly in the face and head, and the following description of the symptoms refers chiefly to this facial erysipelas. Most of these cases of erysipelas arise from little excoriations, cracks, and scratches about the nose or ears, or more rarely about other parts of the face or scalp. A previous coryza, by the accompanying slight erosions about the nose, sometimes gives the opportunity for an attack of erysipelas, but sometimes the infection occurs in the mucous membrane of the nose or pharynx; then we have a primary erysipelas of the mucous membrane, which extends through the nasal passages and finally appears on the external surface of the nose.

Facial erysipelas is most apt to attack the young, and seems to be somewhat more frequent in women than in men. The laity erroneously regard catching cold and getting frightened as frequent causes of the disease. If we except the predisposing causes above mentioned—viz., coryza, slight scratches, cuts, etc.—we usually find no cause of which we can feel certain. Often endemic influences are important. It has been long known that traumatic erysipelas can get so secure a footing in particular hospitals or wards that every wounded person treated in them is in danger of this disease; but the apparently idiopathic variety is sometimes remarkably frequent in particular places (barracks, etc.). Several members of one family may likewise have facial erysipelas simultaneously. In nearly all such cases the sufferers are infected from some common source, for direct contagion is certainly exceptional; but patients with open wounds should never be put near erysipelas cases, because here the risk of infection can not be wholly excluded. Direct inoculation can, however, as has been proved, convey the disease from a patient to other persons or to animals.

In contrast with the behavior of many other infectious diseases (typhoid fever, measles, scarlet fever, small-pox, etc.), erysipelas is peculiarly apt to attack the same individual over and over again. There are persons who have facial erysipelas about every one or two years, and ten to fifteen times. Often the explanation of this apparently lies in some chronic disease—e. g., chronic ozæna, chronic eczema of the nostrils or ears—which makes infection easy, but in other cases no cause can be discovered. Of course when erysipelas afflicts the same person a number of times the individual attacks gradually become milder. Marasmus seems to predispose to erysipelas. At least we have observed that erysipelas occurred with relative frequency, in the Leipsic hospital, in patients suffering from the last stages of phthisis or cancer, or similar diseases.

Clinical History.—In many cases the first subjective symptoms are simultaneous with the cutaneous swelling, and these are chiefly local. There is pain and a sense of tension in the skin. Soon subjective symptoms of fever also appear, such as general malaise, anorexia, headache, and sometimes vomiting. In other cases the disease starts with more violent constitutional symptoms: there is an initial rigor, with violent headache and great languor. The local erysipelatous swelling appears almost at the same time with these general symptoms or sometimes a few days later, either because the local inflammation is at first slight or because it is not noticed, as in the scalp when the hair is very thick. In rare instances the disease begins with sore throat. We saw three almost simultaneous cases of facial erysipelas in one family, in which a severe sore throat lasted for four or five days preceding the appearance of the cutaneous disorder. We have seen a number of similar cases since and believe that they are due to a true erysipelatous angina—that is, to an erysipelas of the soft palate which extends through the nostrils to the external skin. In cases of erysipelas which follow a coryza we may assume, as stated above, that there is an erysipelas of the nasal mucous membrane preceding the erysipelas of the skin.

The erysipelatous process in the skin is almost always circumscribed at first. It usually starts on the nose, less often upon the cheek, the ears, or the hairy scalp. The skin becomes considerably swollen, grows red, smooth, and shiny, and feels hot. The redness and swelling keep spreading. There is usually a sharp, elevated ridge, perceptible to sight and touch, separating the diseased from the still healthy portion of the skin. As long as the erysipelas is spreading, we see stretching out from its border, or somewhat removed from it, small red streaks and spots which gradually increase in area and intensity, and finally coalesce. Any decided fold in the skin may hinder for a time the extension of the disease. The naso-labial folds are particularly apt to limit it. The border of the hairy scalp frequently forms a terminal line; but the whole scalp may be attacked, the inflammation stopping only when it reaches the nape of the neck. The boundary of the erysipelas in the hairy scalp can usually be quite accurately determined by palpation (swelling and local tenderness).

It is only in a relatively small number of cases that it spreads farther yet, attacking the back, the arms, and the anterior surface of the trunk, or even extending to the feet. This is known as *erysipelas migrans*. The facial erysipelas may be healed long before the disease ceases to extend over the other parts of the body. When the spreading process is about to cease, the inflammation usually becomes decidedly milder, appears only in isolated spots, and finally stops completely. In most cases, only the face, the ears, and a part of the scalp are attacked.

It is not a rare thing for vesicles or bullæ to form in the portions of skin attacked. Such cases are called *erysipelas vesiculosum* or *erysipelas bullosum*. The serum may change to pus in these blisters, and then we have *erysipelas pustulosum*. Exceptionally the infiltration of the skin becomes so intense as to result in a localized necrosis or gangrene—*erysipelas gangrænosum*. The parts most apt to be attacked by this are the eyelids, where abscesses sometimes form.

Microscopic examination of the skin shows a marked hyperæmia of all the blood-vessels and a very considerable infiltration of both the skin and the subcutaneous connective tissue with serum and cells. In those parts where vesicles are formed there are many dead and disintegrated epithelial cells in the rete Malpighi. In the parts where most streptococci develop there may also be necrosis of the deeper layers of the skin. We have already said that there may be many streptococci in the lymphatics of the skin, but they are found only in the first fresh stages of the inflammation. When the inflammatory exudation has reached its full height, the streptococci have usually wholly disappeared. As a rule we find only a few or no streptococci in the contents of erysipelas vesicles.

The inflammation in any one spot usually ends four or five days after it has made its appearance there. There is apt to be much attendant desquamation. The face is often left with a finer complexion than it had before.

The other symptoms, of which the constitutional disturbance and the fever are chief, correspond pretty closely to the severity and extent of the cutaneous lesion. It is comparatively seldom that this correspondence does not exist.

The fever in facial erysipelas usually rises rapidly at first, and to a considerable height. We have seen but few cases where the high fever was delayed till a day or two after the skin was attacked. The temperatures observed in erysipelas are often extreme: 106° (41° C.) is not at all rare. The highest we ever saw was 107.2° (41.8° C.). While the erysipelas continues or is spreading, the fever is seldom continuous, nor are the remissions insignificant. Pronounced intermissions, even down to normal, are very frequent, but are followed again by a rapid and great rise of temperature. The fever may terminate with a genuine crisis. In intense cases of considerable extent, or in erysipelas migrans, the termination is more apt to be by a more or less gradual lysis. We have seen the cutaneous inflammation in erysipelas migrans still extend itself a little, in a rudimentary form, after the fever had completely ceased.

The headache is often intense, and seems to result not merely from the inflammation of the scalp but from disturbances of the circulation in the underlying parts, or more probably from the action of a toxine. Other severe cerebral symptoms are also relatively frequent. The patient may be very restless, excited, and wakeful. At night there may be mild or even violent delirium; or there may be decided stupor. All these symptoms are in chief part due to the general intoxication caused by the infection; but they also justify a surmise, as we have said, that there is a circulatory derangement in the meninges and the brain itself, resulting from the inflammation of the scalp. In drunkards, delirium tremens is not infrequent.

One of the most constant symptoms in facial erysipelas is gastric and intestinal disturbance. There is usually complete anorexia. The tongue is thickly coated. Vomiting is frequent, not only at the beginning but during the course of the disease. There is constipation; or there may be quite severe diarrhœa. There is no pathological lesion known corresponding to these clinical symptoms. The spleen is usually moderately swollen. In all severe cases we find in the blood a pronounced leucocytosis (16,000 to 18,000 leucocytes in a cubic millimetre). Streptococci are not found in the blood.

The entire duration of the disease varies greatly in different cases. A very light case may get well in a few days. Most cases of average severity last a week or ten days. Erysipelas migrans may continue for many weeks. We have several times seen a relapse come on after a number of days of complete apyrexia. Either the face would be once more attacked, or some portion of the skin which had previously escaped.

Local **complications** are comparatively rare and insignificant in erysipelas. The lymphatic glands of the throat and back of the neck are very frequently somewhat swollen, but seldom attain great size. Bronchitis and lobular pneumonia may develop in severe cases, but are not at all characteristic. Some observers call attention to the occurrence of pleurisy, endocarditis, and pericarditis; but these complications also are very rare. The spleen is usually slightly swollen. Sometimes there is an icteroid hue. The urine frequently contains a small amount of albumen, and in severe cases of erysipelas acute hæmorrhagic nephritis is not so very rare, but it almost always disappears entirely. The albuminuria usually disappears a few days after the fever. Swelling of the joints has been repeatedly observed. It is more frequent in the severe surgical forms of erysipelas, which are combined with universal septic and pyæmic conditions of the system. Purulent meningitis may complicate an erysipelas located in the head, but it is very rare. We should be exceedingly cautious about asserting its existence even when the cerebral symptoms are very pronounced.

Cutaneous complications are relatively frequent. We have seen herpes labialis quite often, and a number of cases of urticaria. Of much greater importance are the cutaneous abscesses which occur in severe cases. These are due to a phlegmonous or even gangrenous inflammation of the connective tissue. Their most frequent seat in the face is the eyelids, as already stated; and in that case the eye may itself be endangered. At the close of severe cases of erysipelas migrans, numerous abscesses may develop in the skin of the trunk and extremities, delaying convalescence. Since, as we have said, the streptococcus of erysipelas is absolutely identical with the streptococcus pyogenes, all purulent inflammatory complications of erysipelas are to be referred to the local or metastatic action of the original infectious germs, but erysipelatous nephritis is probably of purely toxic origin.

The **diagnosis** of erysipelas is almost always easy when once the cutaneous lesion has developed. Phlegmonous inflammation of the skin and lymphangitis are to be eliminated; but this is always possible, with proper care. After a single examination, we may confound it with acute facial eczema of great severity, or even with a marked urticaria. Chief attention should be paid to the characteristic border of erysipelas and to its manner of extension. In erysipelas beginning in the hairy scalp the local inflammation may easily be overlooked, being completely masked by the severe general symptoms.

The **prognosis** of facial erysipelas, when it attacks a healthy person, is very favorable. In drunkards a severe case may be complicated by delirium tremens, and the issue be unfavorable. We saw one case end fatally because of gangrene of the eyelids, followed by purulent inflammation of the orbital connective tissue. Erysipelas migrans may so exhaust the powers of feeble patients as to become dangerous. The prognosis of surgical erysipelas is relatively more unfavorable, but it can not be considered here.

Treatment.—The treatment of ordinary erysipelas of the head and face, which is the main question here, is purely symptomatic. The large number of remedies recommended (tincture of iodine, nitrate of silver, iodoform, ichthyol, carbolic acid, collodion, etc.) shows that they can not any of them materially influence the morbid process. It is easy to be deceived about their efficacy, as most cases are relatively benign. We therefore usually content ourselves with powdering the affected skin or covering it with oil or vaseline to relieve the tension, and also with putting on an ice-bag if the patient likes it. We have found no proof that we can limit the advance of the disease mechanically by applying firm adhesive plaster to the boundary of the erysipelas, etc. We can expect no specific action from internal remedies (Pirogoff's camphor cure, liquor ferri sesquichlorati, etc.).

Apart from alleviating the local discomfort, some other symptoms occasionally demand special consideration. Severe headache and other severe nervous symptoms may be relieved by the local use of cold or by antipyrine and similar remedies. With high temperature cool baths or antipyretics internally may be used with benefit, but in general the fever does not often demand energetic treatment, since in erysipelas, as we have said, considerable spontaneous remissions of temperature often occur. When there are marked gastro-intestinal symptoms we may prescribe hydrochloric acid, opium, etc.

Only when there is a true migrating erysipelas and the disease spreads over the body should we try more energetic methods. Heuter recommended subcutaneous injections of a two-per-cent. solution of carbolic acid a little way from the edge of the erysipelas. Although this method is certainly rational, we have usually seen no striking results from it. It seems more effective to scarify the erysipelatous skin and then wash it with a corrosive-sublimate solution, as lately recommended. In the severe cases the main point, after all, is to maintain the patient's strength by nursing and food. If cutaneous abscesses form, they should be opened promptly, when they usually soon heal.

CHAPTER X

DIPHTHERIA

(*Diphtheritis. Croup. Cynanche contagiosa*)

Ætiology and General Pathology.—Clinically, "diphtheria" means a certain well-characterized, specific, acute, infectious disease, the chief visible lesion of which is a croupous-diphtheritic inflammation of the pharynx and upper air-passages. In a purely pathological sense, however, the terms "croupous" and "diphtheritic" have a broader meaning. They denote a certain form of inflammation which may occur in the mucous membrane of almost any part of the body. It is frequent in the intestine and bladder. There is great diversity in the causes which may produce it.

The pathological characteristic of every croupous-diphtheritic inflammation consists in the formation of a fibrinous exudation. This may either be a croupous membrane, which is grayish white, rather firm and elastic, and which can be lifted off with comparative ease from the mucous membrane upon which it rests, or it may be a diphtheritic infiltration with necrosis of the tissues. Here the exudation is more or less deeply imbedded within the proper structure of the mucous membrane itself. There is no essential difference between croup and diphtheria; diphtheritic inflammation is the severer form of the disease, croupous inflammation the milder. In diphtheria the fibrinous exudation is preceded by a necrosis of the epithelium and of the underlying tissues of the mucous membrane as well, while in the case of croupous exudation the necrosis is limited to the epithelium. The croupous membrane never rests upon an intact mucous surface, but replaces the epithelium, which has already been totally or in very large part destroyed. Flaky remnants of the epithelium, no longer nucleated, are sometimes found in the meshes of the fibrine. The preceding destruction of the epithelium is essential to the occurrence of fibrinous, croupous inflammation. The fibrinous exudation can be formed in those places only where the cause which excites the inflammation kills the epithelium at the same time. It is still undecided whether or in what degree the epithelium takes part in the formation of the croupous membrane. Most pathologists hold that the material for the fibrine comes from the fibrinogen of the inflammatory matter which transudes through the walls of the vessels, and also from the disintegrated migratory white blood-globules. These last are abundant throughout the deposit itself, and still more numerous in the entire tissue of the mucous membrane beneath the croupous or diphtheritic exudation. If recovery takes place in croup, all that is needed after the exudation has been cast off is the renewal of the epithelium, which can be accomplished through the exclusive agency of the remnants of epithelium left along the borders of the diseased spot. In diphtheria, however, the entire necrotic portion of mucous membrane must slough off, a line of demarkation being formed, and cicatricial tissue replaces the necrosed portion.

The above is a bare outline of the present views about croupous and diphtheritic inflammations. They have been reached gradually through the labors of E. Wagner, Weigert, and others. We have not yet touched upon the ætiological factors; but what precedes renders it evident how manifold they may be, for many causes which destroy the epithelial layer of the mucous membrane, and at the same time promote inflammation, may excite croup. We have mechanical causes, such as impacted fæces, gall-stones, renal calculi; chemical irritants, caustics, like ammonia and the acids; and, finally, a number of specific, infectious, disease-producing poisons. Among these is the specific poison of diphtheria proper.

There has been no doubt for a considerable period that true pharyngeal diphtheria could be produced only by a specific organized cause. To demonstrate this, however, has been thus far extremely difficult, for there are in the diseased parts a great number of diverse micro-organisms, originating in the mouth and throat, and really secondary to the diphtheritic process; and, although they of course are entirely different from the specific "diphtheritic bacteria," it is very hard to separate them. Nevertheless Löffler has of late succeeded in solving the question of the origin of diphtheria in an apparently decisive way. From numerous investigations by Roux and Yersin, Heubner, and others, there can scarcely be a doubt at present that the specific diphtheria bacillus is a form of bacillus characterized by a definite and particular granular formation, a club-like swelling at the ends, and special methods of growth on blood-serum. The diphtheria bacilli are found only on the diseased portion of the mucous membrane or the point of inoculation, and never in the blood or the internal organs. This fact is of the greatest pathological significance, and it has been established not only for diphtheria in man, but also for experimental diphtheria in animals. In pharyngeal diphtheria they are found chiefly on the under surface of fresh false membrane. The severe general symptoms of diphtheria, however, except when due to secondary infection (*vide infra*), depend upon the poisonous chemical action of certain substances produced by processes of metabolism in the bacilli. Little that is definite is known, however, as to the precise chemical nature of these "toxines."

We may accept it as a fact that the diphtheria bacilli or their spores reach the pharyngeal cavity directly through the inspired air or in some other way, and there, in case they attach themselves to the mucous membrane and increase, produce diphtheria. In a few rare cases they seem to be inhaled into the larynx at once, and to excite there a "primary laryngeal croup" (*vide infra*). The source of the infection is always to be referred ultimately to another case of diphtheria, but the way in which the disease spreads can not always be traced in detail. In many cases, of course, a direct ("contagious") transmission of the poison is evident, as from coughing, or the many cases of the disease in physicians and nurses from sucking the croupous membrane out of the tracheotomy tube in children, etc. If several children in one house fall sick, as often happens, we may here suspect a direct transmission of the disease, although it is also possible that in such an event several cases may arise from the same source of infection. We do not yet know positively how far the diphtheria bacilli can lead an independent existence outside of the human body (the earth, the floors of dwellings, etc.). It seems certain that the infection can be carried from one person to another by some intermediary, by clothing, linen, toys, and other objects to which the diphtheria poison clings. The resistance of the latter to external influences—temperature, drying, etc.—is very considerable. After complete recovery from diphtheria, virulent diphtheria bacilli may be found in the oral cavity for a considerable time. This is particularly to be noted on account of its practical importance. We must mention, in conclusion, that attention has lately been called to the possibility of the transmission of diphtheria from sick animals (hens, doves, calves, [cats]) to man, since diseases resembling diphtheria undoubtedly occur in the domestic animals mentioned.

Diphtheria, as is well known, is a disease chiefly of childhood, but some cases, and even very severe ones, may occur in adults. In advanced life the disease is very rarely seen. In large cities sporadic cases occur from time to time, but the disease often takes on an epidemic character. At such times some places appear to be much more subject to the disease than others.

[While it seems in the highest degree probable that the poison is usually purely local at the start, cases occur which suggest that constitutional infection through the pulmonary blood-vessels may precede the local manifestations. Infection through the alimentary canal is not probable, though its occurrence can not be positively denied.

There are still points in the ætiology and pathology of this affection which are involved in obscurity. Much has been said and written in this country and in England about the relations of filth and diphtheria. That filthy surroundings contribute a soil favorable to the development of the poison, and at the same time diminish the resisting power of the human organism, can not be doubted; but, as long as the parasitic theory of infectious diseases prevails, sewer-gas and the like must be classed among the predisposing or accessory causes.

Some of the frightfully virulent epidemics of diphtheria in sparsely settled country districts and on the Western plains are difficult to explain under the theory that each case is mediately or immediately the result of a previous case; these difficulties will, however, doubtless be cleared away in time.]

Clinical History.—The incubation is rather brief, seldom exceeding two to five days. The disease itself almost always begins with general malaise, headache, fever, and pain on swallowing. Little children, however, often do not complain of this last symptom, and in older children the sore throat may not be very troublesome at first. It is therefore a very important rule for the physician to examine the throat carefully in every child who presents ill-defined general symptoms. If diphtheria is beginning, we find redness of the soft palate, and more or less swelling of the tonsils. Upon the inner surface of the latter, and perhaps upon the arch of the palate and the uvula also, are spots covered with a grayish-white coating, which is quite firmly adherent to the mucous membrane. They are less frequent at first upon the posterior wall of the pharynx and the hard palate. Their extent varies greatly in different cases. In the mildest they are chiefly confined to the tonsils, attacking the soft palate or the tonsillar surface of the uvula but little if at all. In severer attacks the spread of the false membrane during the first days of illness is rapid. Almost invariably there is a very early and considerable swelling of the lymph-glands at the angle of the jaw. The constitutional symptoms persist. The children are restless. There is complete anorexia, and frequently vomiting. The temperature-curve is not typical. It is irregular, but is often rather elevated. In true diphtheria such high temperature, 104°–105° (40°–41° C.), as is common in follicular tonsillitis, is certainly exceptional. On the other hand, fever may be slight or almost absent, even in the worst cases. The pulse is very rapid. The urine may have a trace of albumen.

In mild cases the local and constitutional symptoms remain moderate; and at the end of a week or ten days there is decided improvement, with rapid convalescence. In severe cases, however, dangerous symptoms appear, perhaps early; the croupous inflammation involves neighboring organs, or a severe constitutional condition is developed.

The diphtheria very frequently extends into the nose. This "diphtheritic coryza," though not in itself dangerous, is usually a sign that the case is a severe one. The inflammation of the nasal mucous membrane may be simply mucopurulent (from secondary infection with streptococci?), but it may also be croupous. It is betrayed by the abundant purulent discharge. Excoriations and superficial ulcers are usually soon produced at the edge of the nostrils. There may be nose-bleed.

A much more dangerous complication is the extension of the croupous inflammation into the larynx. This creates a mechanical hindrance to respiration, which proves fatal in a great many cases, as the child's larynx is so small. Formerly "croup"—i. e., croupous inflammation of the larynx—was regarded as a different disease from diphtheria. Many specialists in children's diseases still maintain this view; but it is in direct opposition to all anatomical, clinical, and ætiological facts. We grant that there are cases where the pharynx is slightly affected, while the croupous inflammation of the larynx is extreme; and once in a great while the diphtheritic infection results in croupous laryngitis and tracheitis alone, the pharynx escaping disease. Still the proposition that there are two distinct diseases, "croup" and "diphtheria," is absolutely untenable. In the overwhelming majority of cases the throat is first affected and then the larynx. We should also consider how easily slight lesions in the pharynx may be overlooked, especially if located upon the posterior surface of the soft palate or upon the epiglottis. Cases of what is called "ascending croup," in which the laryngeal affection precedes the appearance of the disease in the pharynx, are, to say the least, very exceptional.

Hoarseness is usually the first indication that the diphtheria has attacked the larynx. Then follows the peculiar, harsh, ringing, "croupy cough," so dreaded by the parents, and, finally, there are signs of beginning laryngeal stenosis. Respiration is not much accelerated, but is labored, and the accessory muscles of respiration are called more and more into action. The child becomes more restless and anxious. Its face grows pale and livid. Even in children who can make no definite statement as to their subjective sensations these symptoms which we have mentioned are very noticeable. The chief cause of the dyspnœa is undoubtedly the mechanical stenosis due to the croupous deposit. Paralysis of the laryngeal muscles may perhaps be a factor. If portions of the false membrane become partially detached, they may act like valves, being sucked in at each inspiration, and pushed aside by the current of expired air. If greater stenosis occurs, respiration becomes noisy, resembling snoring. Inspiration, particularly, is prolonged and "sawing," and is attended by marked depression of the larynx toward the sternum. An important diagnostic point is the drawing in during inspiration of the supra-sternal region, the epigastrium, and the lower part of the sides of the thorax. This is the direct result of the obstructed flow of air into the lungs. As the lungs do not expand enough to correspond to the inspiratory dilatation of the thorax, the parts mentioned are forced in by atmospheric pressure. The degree of dyspnœa may vary at different times. The false membrane may be loosened and coughed up, rendering respiration easier for a time, till fresh exudations or displacements of membrane cause renewed distress. Recovery is still possible. The membrane may be expectorated and no more be formed. Unfortunately, this happy termination rarely occurs. In most cases the symptoms of suffocation increase more and more, respiration grows quicker and more superficial, and the child becomes more and more stupefied by the excess of carbonic dioxide in the blood. The pulse gets very small, rapid, and irregular. There are mild convulsions and then death.

The autopsy in these cases usually discloses that the croupous inflammation has extended into the larger bronchi or even into the smaller. The lumen of the bronchioles may be almost completely occluded by false membrane. This croupous bronchitis gives rise to no especial auscultatory phenomena during life, so that often we suspect it rather than diagnosticate it. Its clinical significance, however, is very great, since, even in adults, it may cause death by suffocation without any especial stenosis of the larynx. If it is present, respiration may not be materially relieved in spite of tracheotomy.

Beside the dangers attendant upon the local extension of the diphtheritic inflammation, the general intoxication of the body must be regarded as the second most important circumstance in our general estimate of the disease. As mentioned above, the diphtheria bacilli produce an extremely poisonous toxine, which enters the circulation and which may give rise to the severest symptoms. Our knowledge of human diphtheria is not yet so far advanced that we can state positively what particular symptoms are due to this intoxication, but special attention must be called to the fact that the general symptoms are often very slight, in spite of extensive local croupous inflammation. On the other hand, where the local disease is relatively slight, severe general symptoms sometimes occur which may justly be referred to this diphtheritic intoxication. Among these severe general symptoms are somnolence, which may increase to complete sopor, an increase in the pulse to 120 or 140, progressive cardiac weakness, and collapse. The conditions are different in those cases which are termed septic diphtheria. Here the local disease in the pharynx usually has a special appearance: instead of the croupous deposits we see a deeper-seated necrotic destruction of mucous membrane covered with a foul-smelling, greasy, purulent deposit ("gangrenous diphtheria"). In such cases the lymph-glands in the neck are almost always much swollen. The tongue is dry and fissured, and a stinking secretion runs from the nose. The temperature is usually not very high, but the pulse is very rapid and small. In these cases there is certainly a secondary septic infection, probably in most cases from streptococci, as in scarlatinal diphtheria (*vide supra*). General septic intoxication and secondary septic inflammation are now mixed with the original disease, and, as we have said, it is often hardly possible to trace in an individual case the precise origin of the morbid condition.

These considerations must also be borne in mind in our study of the symptoms manifested in other organs in diphtheria. We must first mention the direct local extension of the original specific diphtheritic process to the neighboring parts. We have already said that the nose, larynx, trachea, and bronchi are often involved, but the dense squamous epithelium of the œsophagus and the mucous membrane of the stomach, which is protected by its secretion, are very rarely affected. We must add, however, that diphtheria often extends to the Eustachian tube and middle ear, and also to the anterior part of the oral cavity (gums, lips), and through the nose to the lachrymal canal and the conjunctiva. The latter may also be affected by the direct conveyance of the infectious matter by the finger, etc. In a similar way diphtheria may sometimes involve the external genitals (in girls) or any accidental wounds or injuries of the skin, such as blistered surfaces, etc.

Of the internal organs, the lungs, heart, and kidneys demand chief attention. In the lungs, beside croupous bronchitis, pneumonia often develops in severe cases. This is usually a lobular catarrhal pneumonia, more rarely lobar croupous pneumonia. Although we can not absolutely deny that the pneumonia may sometimes be of a true diphtheritic nature, the majority of cases of pneumonia developing in diphtheria must certainly be classed as secondary affections, due to the inhalation and development of secondary agents (probably streptococci), which may readily take place. After the original diphtheria has disappeared the secondary lobular pneumonia may long prevent recovery or even cause a fatal termination of the illness. The condition of the heart in diphtheria is of especial importance, because severe functional impairment, probably of toxic origin, is common. Even when the fever is slight the pulse may sometimes be very rapid, small, and often irregular. An abnormal slowness of beat is much rarer. It is especially to be borne in mind that even in apparently mild cases there may be very dangerous cardiac weakness. Even during convalescence we repeatedly see sudden death which must be regarded solely as "heart-failure." The autopsy shows that gross disease of the heart is very exceptional in diphtheria, but degeneration of the muscular fibers and little interstitial myocarditic foci of secondary origin are often found in severe cases. It is at present quite impossible to estimate the actual clinical significance of these anatomical changes, since it is at least probable that disturbances of the innervation of the heart cause the cardiac weakness as much as these lesions of the cardiac muscle.

Since nephritis is especially common in septic diphtheria, it is hard to decide whether it is due to the primary disease or is to be considered a secondary complication. Its clinical significance is not very great, since it neither materially modifies the general picture nor has any definite bearing on the result of the disease. Œdema, uræmia, etc., are only rarely seen in diphtheritic nephritis. The macroscopic changes in the kidney are usually very slight. Microscopically we find the various degenerative conditions of acute nephritis (*vide infra*).

The Nervous Sequelæ of Diphtheria.—The convalescent from diphtheria is liable to be attacked by certain nervous sequelæ. Of these, diphtheritic paralysis is the most important. It appears about one or two weeks after the throat trouble ceases, or perhaps earlier, and it is quite as likely to follow mild cases as severe ones. It attacks the soft palate by preference. The voice becomes nasal and deglutition difficult. The naso-pharynx is imperfectly cut off during the act of swallowing, and with each attempt liquid regurgitates through the nose. Usually the pharyngeal mucous membrane is hypoæsthetic at the same time, and deprived of its reflex excitability. There may also be paralysis of the vocal cords upon one or both sides, and this again is frequently combined with anæsthesia of the mucous membrane of the throat. There may be paralysis of the ocular muscles, of which those controlling accommodation are most apt to be affected, rendering the vision for near objects imperfect. Paralysis of the muscles of the trunk and extremities is least frequent, but it may be very extensive. Sometimes several of these parts are paralyzed simultaneously. Thus we see quite often paralysis of the soft palate and of the muscles of accommodation combined. In some cases there is well-marked ataxia of the lower limbs with or without paresis. This renders the gait very uncertain and tottering, the tendon reflex is almost always abolished, while sensation is affected slightly if at all. Very rarely diphtheria is followed by contracture of the hands or other parts, by difficulty in articulation and paresis of the bladder. A paralysis of the pharynx is sometimes left behind, so that the children have to be fed for weeks through an œsophageal tube. It is a remarkable fact that not only in almost every case of the nervous disorders which we have mentioned, but often also in individuals who have entirely escaped them, there may be no patellar reflex after diphtheria for weeks or even months. With regard to the pathological state, it is probably a degenerative condition of the corresponding peripheral nerves, not only in the post-diphtheritic paralysis, but also in the cases of post-diphtheritic ataxia (see the chapter on multiple neuritis). These degenerations are probably due to the poisonous chemical action of certain toxines which are produced directly by the vital processes of the diphtheria bacilli. It is therefore of great interest to note that paralysis has been produced experimentally in animals by the action of diphtheritic toxine by Roux and Yersin and others. The prognosis of all the nervous sequelæ of diphtheria is very favorable, and even in severe cases complete recovery usually takes place in the course of a few weeks or months. This is in harmony with the peripheral nature of the disease. But there is one paralysis which is highly dangerous—that of the heart, as already mentioned. It may occur suddenly during convalescence. Probably it is analogous to the other nervous derangements, and the result of degeneration in the [nuclei or] fibers of the pneumogastric.

Diagnosis.—The physician will seldom fail to recognize a case of actual diphtheria if he pays proper attention. The characteristic patches and the severe general and local symptoms make the diagnosis certain. It is much more common to mistake other forms of sore throat, particularly in adults, for diphtheria. The most deceptive are follicular and necrotic tonsillitis (*vide infra*). We must not suppose that every white spot upon the tonsils is diphtheritic, and we must speak of " diphtheria " only when there is an actual croupous inflammation in the pharynx with a true membranous deposit. The practiced eye can usually distinguish at the first glance the plugs of pus limited to the follicles of the tonsils, which are usually multiple, and also the slight and superficial necrosis of mucous membrane limited to the tonsils in necrotic angina. Of late, it has often been proved by bacteriological examination that in these tonsillar affections there are no diphtheria bacilli, and that they therefore are not true diphtheria.

We must add, however, that there are light forms of true diphtheria in which the pathological changes assume a milder form, resembling the tonsillar affections just mentioned. In such cases we are therefore certainly justified, especially in dealing with children, in bearing in mind the necessary precautions, but in our opinion the physician should never alarm the family by the justly dreaded word " diphtheria " without sufficient reason.

An absolutely definite diagnosis between diphtheria and the other tonsillar diseases can be made only by bacteriological examination. This is at present still too difficult a matter in diphtheria to be within the scope of every physician, but the constantly increasing number of good bacteriological laboratories renders it possible for almost every physician to obtain bacteriological examination of the suspected deposit in doubtful or important cases. An experienced clinician can usually make a correct diagnosis and prognosis even without a " culture."

[When the membranes are confined to the nose, the diagnosis may be more or less difficult; but it is especially in cases in which the nasal mucous membrane is involved that we encounter great swelling of the glands at the angles of the jaw. There is also apt to be a thin, acrid, bloody, or sero-purulent discharge.

Jacobi states that while diffuse pharyngeal injection may or may not point to imminent diphtheria, marked local congestion is either traumatic or diphtheritic. An examination of the urine should never be neglected in doubtful cases: in diphtheria a trace of albumen is very common; in simple or follicular sore throat albumen is very rare, if indeed it occurs at all.]

Prognosis.—The dubious prognosis of true diphtheria is universally known, even by the laity. The very fact that the best-developed and healthiest children so often fall victims to it associates the name diphtheria with the saddest memories. There are indeed many mild cases which recover in a week or two, and severer ones which end happily in three or four weeks; but in most cases, where the process extends into the larynx, or the symptoms of a severe constitutional infection occur, the prognosis must be regarded as very serious. Only recently, however, since the introduction of the serum treatment, has the prognosis even of severe diphtheria become considerably more favorable. Formerly, before the serum treatment, it was regarded as a relatively favorable result if one half of the children with diphtheria in the hospitals, on whom tracheotomy was done, recovered! Sixty or seventy per cent. of the children operated on often died. At present, however, the mortality of the tracheotomy cases, treated at the same time with serum, has fallen to about twenty-five per cent., and these are only the severe cases in which the serum treatment was begun late. The improved prognosis under the serum treatment appears much more favorable if we compare the whole number of cases and the death-rate of diphtheria before and after the introduction of this treatment (Heubner, Widerhofer, Baginsky, etc.).

In general, diphtheria is to be regarded as the more serious the younger the child is. In later childhood, after the eighth or tenth year, the number of dangerous cases is much less. The description of the symptoms shows sufficiently what the dangers of the disease are, and how they are to be recognized. We would state once more that, even in apparently mild cases, the danger of sudden paralysis of the heart, though fortunately rare, always demands the greatest caution.

Treatment.—Up to a short time ago it was impossible to say anything in regard to any generally recognized treatment of diphtheria which was really effective, but very recently a method of treatment has been discovered, chiefly by the important and interesting investigations of Behring and Roux, which deserves to be called an actual specific. If further observations confirm the present results, this treatment is one of the most brilliant and triumphant medical acquisitions.

Behring found, in continuing the important discoveries of Pasteur, Buchner, and others, that certain protective matter ("antitoxine") was formed in the blood-serum of those animals (sheep, goats, horses, etc.) which had been infected with artificially weakened cultures of diphtheria bacilli. If the animal survive the milder infection, it is less sensitive to a severer infection, and, finally, by continued inoculation, it may even become completely immune to the severest infection. The blood-serum of such immune animals can now be used in the treatment of diphtheria in man. The treatment is simple, and consists in injecting the serum under the skin of the child who has diphtheria by means of a carefully disinfected syringe. The best places for injection are the anterior chest wall or the thigh. Since the amount of serum to be injected is usually about five cubic centimetres, we should use a special syringe which is not too small. The best are made like a Pravaz syringe with an asbestos packing. It is to be understood that the injections must be made with the strictest aseptic precautions. There are three different preparations of serum in the market, varying according to the strength of their immunizing action. In fresh and milder cases we inject Serum I ("600 units of antitoxine"), in severer and more advanced cases, especially if there be any signs of disease in the larynx, we use at once Serum II ("1,000 units of antitoxine"), or even Serum III. According to the severity of the case, or according to its course, we repeat the same injection or a weaker dose of the same once in the next twelve to twenty-four hours. In many cases of moderate severity a single injection of Serum II suffices. The favorable action of the serum is shown chiefly by the fact that the affection does not extend after the injection. The existing croupous deposit is thrown off in the course of the next four or five days.

The serum injections have no injurious action. Urticaria sometimes occurs after an injection, but it has no serious significance. There is no proof that albuminuria and post-diphtheritic paralysis are seen oftener in cases treated with serum than in other cases. We need not explain further that the serum must be ineffectual in severe septic diphtheria. Taking everything into consideration, it is therefore the duty of the physician, in accordance with our present knowledge, to use the serum in every case of true diphtheria in children. At present it is, of course, impossible to pronounce a final judgment on the whole question; but it seems beyond a doubt that we physicians are standing on the threshold of an entirely new era in the treatment of infectious diseases.

It is of the greatest practical importance that the serum treatment be begun as early as possible. The most favorable results are seen in children treated on the very first days of the disease. [In merely suspicious cases, therefore, it would seem wise to administer the antitoxine at once, and thus give the patient the benefit of the doubt.—V.] Local treatment of the affected places in the pharynx at the same time is unnecessary. We would, however, emphasize the importance of a general cleansing and disinfection of the whole oral cavity by gargling and rinsing with dilute solutions of potassic chlorate, salicylic acid, corrosive sublimate (1 to 3,000), etc. Inhalations may also be used, with caution, for the same purpose. It is also very important to watch the child's nutrition carefully in order to keep up the strength. Milk, eggs, meat juice, small quantities of wine, etc., are most advisable.

Since the introduction of serum-therapy all the former methods of treatment have been quite superseded. This is especially true of the many previous attempts to limit the morbid process and cause it to disappear by local disinfection. The practical difficulties in carrying out any really effective local treatment have always prevented the general recognition of the value of caustics, painting, etc. Nevertheless some remedies, even up to the present time, are still recommended as efficient, especially liquor ferri sesquichlorati (pure or diluted), dabbing with a 1-to-1,000 sublimate solution, etc.

Of the many internal remedies which were formerly tried we can mention only chlorate of potassium and iodide of potassium. The former remedy may still be recommended and may be given in teaspoonful doses of a solution containing 1 part to 30 or 40 of water.

[The tincture of the chloride of iron is much used in this country in the treatment of diphtheria, and appears to be of real service; but it must be given in large doses. The following prescription is recommended by Jacobi, whose experience has been very large, for a child of two years:

℞ Tinct. ferri chloridi	ʒij;
Potas. chlorat.	gr. xx;
Glycerin. pur.	℥j;
Aquæ	℥v.

M. S.: Teaspoonful every fifteen, twenty, or thirty minutes.

Turpentine is better as an inhalation than by the stomach; a teaspoonful or two of the oil can be poured in water kept at the boiling point by an alcohol-lamp. The whole air of the room is thus charged with the remedy. No drug should be used with disorders the stomach. Tablet triturates containing one one-thousandth of a grain of corrosive sublimate can be allowed to melt in the mouth with the greatest freedom, and seem sometimes to exert a distinctly beneficial local action. The dose is, however, too small to secure the systemic effects of the drug unless the case is a mild one and free from notable dysphagia.]

If the larynx is attacked, and if, in spite of the serum treatment, the consequent laryngeal stenosis threatens to cause suffocation, tracheotomy is our only resort. It is never indicated by the disease itself nor by the severity of the case, but only by persistent obstruction of the larynx. It is therefore not invariably easy to decide whether tracheotomy is called for in any particular case. If the general condition be bad and respiration already impaired, it is often very difficult to determine whether laryngeal stenosis exists. Tracheotomy will be of no avail if the croup has already extended to the bronchi, or if the dangerous condition of the patient is due to the severity of the constitutional infection or to incipient paralysis of the heart. We have already said that, since the introduction of the serum treatment, the prognosis of diphtheria has become better even in the tracheotomy cases. How tracheotomy is performed, and in what the after-treatment consists, must be learned in the text-books on surgery, where we will also find the necessary description of the so-called intubation of the larynx.

Intubation, as devised by Dr. O'Dwyer, is a procedure which makes a distinct advance in the treatment of laryngeal stenosis. In permitting the free access of air to the trachea, intubation may save life without resort to the serious operation of tracheotomy; or it may tide over a time until tracheotomy becomes absolutely necessary or until the consent of the parents can be obtained to the use of the knife. In hopeless cases it may promote euthanasia.

If severe pulmonary symptoms occur in diphtheria, we should use first of all wet packs of the whole body or tepid baths with cool affusions. Such cutaneous stimulation refreshes and enlivens the whole nervous system, and we should therefore try it also in the severe cases with secondary septic infection. In septic diphtheria we must also pay especial attention to the condition of the heart. So far as is possible we should try to avert the threatening cardiac paralysis by stimulants, such as wine, camphor, and strophanthus.

The nervous sequelæ of diphtheria are best treated with the constant current. As an internal remedy, iron is good, and also nux vomica or strychnine. The last may be given subcutaneously, if desired, in doses of gr. $\frac{1}{60}-\frac{1}{30}$ (grm. 0.001–0.002).

The prophylaxis of diphtheria demands that every child with diphtheria be absolutely isolated from healthy children. Behring [and many others] believe that a healthy child may be positively protected from infection by diphtheria for two or three weeks by the injection of a small dose of serum—the whole or one half of Dose I—that is, about three hundred units of antitoxine. It is, of course, very hard to judge of this, and for the present, therefore, it must be left to the individual judgment of the physician in what cases he will try prophylactic injections of serum.

[Diphtheria is a disease which involves commonly much exhaustion, and too much stress can hardly be laid on the importance of administering the maximum amount of nourishment in the most assimilable and easily swallowed forms from the start.

It is also important to give stimulants early in most cases, not waiting for signs of exhaustion. Enormous quantities of brandy can often be given to small children without the slightest toxic effect. No general rule can be laid down; the requirements of each case must be studied and met.

When painful deglutition interferes with nutrition, peptonized milk, eggs, brandy, and the like, must be given by the rectum. Rectal alimentation and stimulation and feeding with the œsophageal tube are also to be resorted to in cases of post-diphtheritic paralysis of the œsophagus.]

CHAPTER XI

INFLUENZA

(*La Grippe*)

INFLUENZA is a specific, acute, infectious disease which is especially distinguished by the occasional enormous extent of its epidemics. While often years and decades pass without any especial attention being called to the disease, suddenly cases of it will appear with such frequency that the largest part of the population is attacked, and the disease may better be described as pandemic than epidemic. Pandemics of influenza can be traced back with certainty into the sixteenth century. In the present century the influenza during the years 1830–'33 traversed almost all of Asia and of Europe, then later there appeared numerous smaller epidemics, but these aroused general attention so little that the disease, upon its last pandemic appearance in the winter of 1889–'90, was almost unknown to many physicians. Since that time the disease seems never to have wholly disappeared, as a few cases, and occasionally small groups of cases, are constantly seen.

Ætiology.—The organized agents of influenza are still not positively known, but R. Pfeiffer has invariably found in the bronchial secretion of patients with the "catarrhal form" of influenza (*vide infra*) a definite form of very small bacilli with rounded ends which stain darker than the central portion. These bacilli, which are either free in the mucus or in the pus-cells, are very probably of significance as a cause of the disease. The influenza bacilli very soon die if dried, and also if put in water.

In all probability the infection is usually due to inhaling the germs. The bacilli seem to appear at certain times over an immense territory, so that they or their spores are scattered everywhere through a large extent of country. Many observations upon the appearance of the disease in isolated institutions (convents and the like) render it very probable that the poison may also be carried by a person suffering from the influenza to another in regions previously unaffected. Nevertheless, this contagious manner of spreading plays no great *rôle* in comparison with the direct infection from the outer world, this latter mode being everywhere possible during an epidemic of influenza.

There is scarcely any reason for speaking of especial predisposing causes of influenza, inasmuch as at the time of a well-marked epidemic the overwhelming majority of the population are attacked, both the healthy and the diseased, the vigorous and the feeble. Sex certainly makes no difference, and age only to this extent, that the disease is seen more rarely in little children under one year of age than in older children and adults. That catching cold has no special ætiological significance is evident from the fact that influenza often appears in patients who are already sick in bed.

It should finally be mentioned that animals also, and in particular horses, may be attacked by the influenza; but, nevertheless, it is as yet a doubtful question whether all the diseases in animals which are described under this name are actually identical with genuine influenza.

Symptoms and Clinical History.—The best general idea of the extremely manifold symptoms of the disease will be obtained if we bear in mind that the influenza causes both a marked infectious (or toxic) general constitutional disturbance of the body, and also certain local lesions with local symptoms. The clinical picture therefore varies greatly according to the predominance of one or the other group of symptoms, and also according to the special form of the local disease.

The onset of influenza is generally rather sudden. As a rule the marked cases begin with rather high fever, ushered in with a chill, violent headache, marked constitutional depression, and usually considerable pain in the back and loins. The weakness of the patient may be so great that, even if a vigorous individual, he will at once take to his bed. Severe nervous symptoms, such as stupor and delirium, are exceptional. Sometimes, but not very often, there is initial vomiting. The backache is often associated with pains in the muscles and joints. Oppressive pain in the eyes is quite characteristic also. This is particularly felt upon moving the eyeballs, and therefore is probably located in the external muscles. The spleen is occasionally somewhat swollen, but any great increase in its size is exceptional.

If the clinical symptoms as the case progresses are mainly limited to the above-named constitutional symptoms—fever, languor, headache, pain in the muscles—we may speak of a "typhoidal form" of the disease. Usually, however, certain local symptoms put in an early appearance, and it is especially the respiratory apparatus which is attacked. The precise symptoms vary considerably in different cases. Sometimes the upper portion of the respiratory tract, the nose, larynx, and trachea are involved; sometimes, from the start, the smaller bronchi. In the first instance there is marked coryza or hoarseness, in the other case there is cough, due to a dry bronchitis, which can be easily detected upon auscultation, and which involves especially the lower portion of the lungs. If these local symptoms outweigh the constitutional, the case is described as belonging to the "catarrhal form" of influenza.

Sometimes the influenza is localized in the digestive apparatus. This "gastro-intestinal form" is much rarer than the catarrhal. In this case, in addition to the more or less strongly characterized constitutional symptoms, there is marked disturbance of the stomach and intestines, as shown particularly by nausea with persistent vomiting, diarrhœa, abdominal pain, etc. In one case we observed jaundice. We may also mention in this connection the appearance of an initial pharyngitis.

The pains in the back, loins, and extremities already mentioned may persist with unusual violence, and this peculiar form of the disease is known as the "rheumatoid." The muscles and the muscular attachments are probably the chief seat of these pains, which may be so violent that the patient is unable to lie comfortably, and sometimes keeps up a continual moaning. The loins, in particular, may be the seat of most acute pain, also the upper arms, the knees, the thighs, and the eyes. Objective changes in the painful parts, such as swelling of the joints, are scarcely ever seen, nor are the nerve-trunks as a rule especially sensitive to pressure. The painful muscles are usually weaker than normal.

The grouping of the clinical varieties of influenza under the four forms already named affords a general idea of the manifold symptoms of the disease, but this division into separate forms must not be carried out too strenuously, for in reality many cases of the disease occur which present transition forms and combinations of the various groups of symptoms. Moreover, in all the forms a distinction must be made between mild and severe attacks, for in influenza, just as in most other infectious diseases, there are many rudimentary and mild cases as well as the fully developed ones, and some could not be properly interpreted but for the presence of the epidemic.

The duration of the disease is best determined by the duration of the fever.

In the very mildest cases there may be no fever whatever, or simply a slight evening rise of temperature. As a rule, there is a moderate fever, between 101.5° and 103° F. (38.5° and 39.5° C.), although higher temperatures even to 104° F. (40° C.) and more are not infrequent. In the beginning of every severe attack the fever rises abruptly. After a duration of several days (four to seven) it may fall again in a manner approaching a crisis. More frequently, especially when there exists diffuse, catarrhal trouble in the lungs, the fever ends by lysis. With comparative frequency there are found to be noticeable deviations in the temperature curve; thus, for example, the high fever of the onset sinks on the second or third day, to be followed by an almost afebrile period of one or two days, whereupon a marked rise of temperature ensues. With this change in the temperature there are usually also corresponding variations in other symptoms.

We see, then, that the duration of simple, uncomplicated influenza is in the mild cases about three or four days, in the severer cases about seven to ten days. To be sure, we should also consider in this connection that convalescence is often surprisingly slow, so that the after-pains (as it were) of the disease are felt for weeks. These consist, for instance, in a certain degree of debility, and in painfulness of the muscles. Sometimes also there are complete relapses, so that directly or a short time after the disease has ended the symptoms begin anew. The special form of the disease may change in this case, so that, for example, the relapse of an influenza with predominant constitutional symptoms assumes the pronounced catarrhal form. Again, during the same epidemic it is not very rare for a patient to suffer from two attacks of influenza separated by a considerable interval of time.

Complications and Sequelæ.—While all the symptoms of influenza which we have thus far described are the direct effects of the original pathogenic cause, the majority of the frequent complications are undoubtedly dependent upon the ingress of secondary infection. The system when attacked by influenza is greatly exposed to these secondary influences, and almost all the dangerous and tedious cases of influenza become such only because of a mixed infection of this sort. This is particularly true of the lungs, in which secondary disease occurs most frequently—sometimes even in the first days of illness, but also in other cases later. The conditions here are similar to those seen in measles and whooping-cough. The simple, mild catarrh belongs to the original disease; the severe pulmonary affections are, however, invariably secondary complications occasioned by new pathogenic influences. These influences are not always the same. According to the investigations of Ribbert, Finkler, and others it is chiefly the pneumonia diplococcus and the streptococcus which are the true excitants of the secondary pneumonia seen in influenza. These cases of pneumonia are either extensive catarrhal pneumonia especially affecting the lower lobes, or more rarely croupous pneumonia with its characteristic sputum. We must also add that the influenza bacilli themselves may excite a lobular pneumonia, which shows a marked purulent character. If pneumonia be added to influenza, the former overshadows the whole picture. The patient is oppressed for breath, has a severe cough with profuse expectoration, looks pale or cyanotic, and suffers from high fever. These symptoms persist for two or three weeks, and then gradually abate. It is in this way that influenza becomes dangerous for elderly and feeble or sickly persons. With noticeable frequency, pleurisy with effusion is conjoined with the influenza pneumonia. The exudation is generally serous, but exceptionally it is purulent.

Complications in other organs are less frequent. We would make especial mention of purulent otitis media and keratitis and other severe diseases of the eye. We have several times observed acute nephritis, but this has always pursued a mild course. Among cutaneous eruptions herpes labialis is a frequent phenomenon in all forms of influenza, even the milder. Other exanthems, such as urticaria and roseola, are much less frequent. Many of the complications named may continue even after the fever and all other symptoms have ceased, so that they must be regarded as sequelæ. This is particularly true of the diseases of the ear and eye and of persistent bronchitis, but only rarely of a pneumonia assuming the chronic form. An important and for the patient a troublesome and painful sequel is furunculosis, especially if some of the boils are located in the axilla or near the anus. Very often neuralgic pains in the distribution of the trigeminus or in the course of the sciatic or other nerves will persist for a considerable time after the influenza has ceased; but these pains may sometimes be located in the muscles; thus, for example, the frequent, persistent, and troublesome pain in the eyes. In a few cases more severe nervous sequelæ, such as spinal symptoms and psychoses, have been seen after influenza. A special form of acute hæmorrhagic encephalitis (*vide infra*) has been thought to have a relation to influenza.

Diagnosis.—The diagnosis of influenza is in general not difficult if one has to deal with a well-marked case at the time of an influenza epidemic. The characteristic initial symptoms of fever, headache, and pain in the loins are to be considered first of all. Their onset is much more rapid than, for example, in typhoid fever. Later on the pain in the various muscles as well as the catarrhal symptoms are the most characteristic phenomena.

Uncertainties and errors in diagnosis may arise from the fact that, on the one hand, we are disposed at the time of an epidemic to call almost all the catarrhal and indefinite mild affections we see " influenza," while, on the other hand, when there is no epidemic, we can not positively distinguish the sporadic " cases like influenza " from the ordinary acute febrile bronchitis, etc. It is indeed very possible, *a priori*, that different morbid agents may produce similar mild acute diseases of the accessible mucous membranes. In such cases, with pronounced general symptoms, high fever, etc., we must often be content with the diagnosis of " infectious " laryngitis, bronchitis, etc., without being able to decide definitely whether the case is to be classed ætiologically with influenza or not. At any rate in practice we should not abuse the diagnosis of influenza.

Prognosis.—For an individual who is healthy and vigorous, influenza is not a dangerous disease, even in its severer forms; for elderly persons or invalids it may, however, be a serious affection. Patients with heart disease or pulmonary disease, or those suffering from chronic nervous troubles, sometimes succumb to it; so that the general mortality at the time of a great epidemic of influenza is always considerably increased. The above-enumerated pulmonary complications are by far the most dangerous; less often is a fatal termination caused by general or cardiac weakness. The above-mentioned nervous sequelæ are also to be considered in prognosis.

Treatment.—No specific remedy for the disease is known. Many physicians maintain that the exhibition of calomel at the beginning of the attack decidedly shortens its course, but confirmatory experience is wanting. In general we must, therefore, pursue a purely symptomatic method of treatment. For the initiatory fever, the headache, and the pain in the loins, antipyrine is sometimes a good remedy, and the same may be said also of phenacetine, antefebrine, and especially salipyrine. These drugs are also prescribed for the persistent pains in the muscles which come on later. Soothing liniments and ointments may be employed, with friction, for the same purpose. The treatment of the pulmonary complications is according to the established methods. Morphine is mainly used for the troublesome cough. Apomorphine, senega, and other expectorants may be employed, and, if indicated, external remedies such as an ice-bag or dry cupping. If the patient becomes very feeble, stimulants such as [strychnine], champagne, and strophanthus are demanded.

CHAPTER XII

DYSENTERY

Ætiology.—By "dysentery" is meant a disease of the colon, which appears sporadically, but more often in epidemics; it is excited by infection with an organized pathogenic poison, about which we have as yet no definite knowledge; and the infection is probably at first a local one. From the investigations of various men, especially Kartulis, it seems very probable that a certain form of amœba must be regarded as the cause of "endemic tropical dysentery." At least in endemic dysentery we almost invariably find, both in the stools and in the walls of the dysenteric intestinal ulcers and in the dysenteric abscesses of the liver, many amœbæ (amœba coli), which are never found in any other intestinal affection. Dysentery may also be artificially produced in cats by means of fæces containing amœbæ. In dysentery as it occurs in Germany, however, we do not apparently find amœbæ in the stools as a rule, although even in Germany Quincke and others have described certain cases of "acute enteritis" ("amœbic enteritis") in which there were a large number of amœbæ. The question of the excitants of dysentery therefore needs further investigation, and it is not impossible that several intestinal affections with different ætiology have thus far been included under the name of dysentery, especially in the tropics. The true home of dysentery is in warmer and tropical countries, where the disease is much more violent and widespread than here. For example, the mortality among the soldiers of the Anglo-Indian army due to dysentery is said to be at times thirty per cent. of the entire number of deaths. In our climate most of the epidemics occur at the end of summer and in autumn. Endemic influences are certainly important. The special conditions in some places are evidently very favorable for the development and dissemination of dysenteric germs, and there are other places equally unfavorable. There can be no other explanation of the immunity of some localities contrasting with the great prevalence of the disease in others. How infection occurs we do not yet know. Many observations, especially in southern countries, support the idea that the germs may be taken into the system by drinking water. Dysentery does not seem to be directly contagious; but that it can be spread through the medium of the fæcal dejections of the sick—e. g., from privies, chamber-vessels, and bed-linen—is very probable. Many cases were formerly referred to catching cold or to some error in diet; but we must, of course, regard these merely as predisposing influences.

The objective **pathological lesion** of the colon, in all severe cases, consists in a pronounced croupous-diphtheritic inflammation. The remarks as to the general pathology of such inflammations made in the preceding chapter are equally applicable to the analogous dysenteric inflammation. In this case, too, there is first a destruction of the epithelium and then the formation of a fibrinous exudation occupying its place, and penetrating down into the tissue of the mucous membrane itself. At the same time there is an intense purulent infiltration of the mucous and submucous tissue, accompanied by extensive ecchymoses. In the most virulent cases the macroscopic appearances are marked thickening of the whole wall of the intestine, congestion of the serous layer, and the conversion of the inner surface into a mottled, dark-red, irregularly roughened area of ulceration. The disease may be confined to the rectum and the sigmoid flexure, but in severer cases it involves the entire colon as far as the ileo-cæcal valve, or even extends to the lower portion of the ileum. Besides this severe form of diphtheritic or even gangrenous dysentery, there is a milder variety, termed catarrhal dysentery. In this the mucous membrane is found in a state of intense purulent inflammation, with ecchymoses. Even here little masses of croupous exudation, which can be torn off, have replaced the epithelium; but they never form continuous layers of great extent. There is no sharp boundary-line between the two forms, the milder catarrhal-croupous and the severer diphtheritic dysentery. Numerous transitional and combined varieties exist.

We must remark, in conclusion, that precisely the same anatomical changes as are presented in true dysentery may result from other causes. Important among these is persistent fæcal impaction in the rectum, which, by a purely mechanical effect upon the epithelium, may excite a diphtheritic inflammation in the mucous membrane. And any severe constitutional disease whatsoever, such as typhoid fever, measles, small-pox, septicæmia, or phthisis, may be attended by a so-called "secondary dysentery." This is most frequent in hospitals. Whether it has the same ætiology as genuine dysentery is uncertain.

Clinical History.—Throughout the entire illness the most prominent symptoms are intestinal. There may be first of all some slight irregularity of the bowels for a few days, and then appears a moderate diarrhœa. The stools are at first feculent, although thin, and number two to six daily. After a few days the discharges increase in frequency, and become extremely characteristic.

The stools are very frequent, occurring ten to twenty, and even sixty or more, times, in twenty-four hours. In severe cases there may be a distressing and almost constant desire to evacuate the bowels. After every operation, and to some extent during it, there is tenesmus attended by intense burning pain in the anus. The stools soon lose their usual feculent character in great part if not entirely. They become scanty, so that not more than about half an ounce is evacuated each time. For the most part they usually consist of a sero-mucous fluid, in which are suspended numerous shreds and particles of varying size. These are blood-stained bits of mucus, little coagula of blood, and necrosed pieces of mucous membrane. One or another of these constituent parts may predominate, so that there may be slimy, purulent, or bloody stools, or all sorts of combinations of these varieties. We often find, besides, a few small masses of fæces, usually covered with mucus. We sometimes see numerous clumps of mucus, resembling sago or frog's spawn; they are probably mucous casts of the follicles. Under the microscope the greater part of the dysenteric discharge is seen to consist of pus-corpuscles and blood. There are also cylinder epithelium and an enormous amount of detritus, and the bacteria of putrefaction. A purely dysenteric stool has no bad odor, except that in the worst cases of gangrenous dysentery the discharges become blackish and extremely offensive.

The rectal tenesmus may be accompanied by a cramp-like pain during micturition. There are often violent attacks of colic. The abdomen is usually rather tense, and tender on pressure along the line of the colon, but without tympanites. The anus may be red, inflamed, and excoriated. Gastric symptoms are on the whole infrequent, if we except the complete anorexia which exists in all severe cases. Sometimes there is repeated vomiting. Occasionally hiccoughs prove distressing. The tongue usually has a dry, greasy coating.

The symptoms just depicted last about a week or ten days. If the case is of much intensity, the general condition is also greatly affected. The patient seems much collapsed, and is very languid and feeble, with a small and rapid pulse. The skin becomes cool and rough, the voice weak and hoarse. There is pain in the muscles. The patient wastes away. The temperature has little that is characteristic or typical. In many cases there is no fever at all, and the temperature may even be subnormal. In most cases, however, there is an irregular fever seldom exceeding 104° (40° C.), and having remissions.

In the worst cases the general weakness may increase more and more, and death occur; but with us a favorable termination is much more frequent. The distress gradually diminishes, the stools assume more and more of a feculent character, the patient becomes stronger, and after one and a half to three weeks convalescence is established. It may be a long while, however, before a patient completely recovers from a severe attack. A third possibility is the transition of the acute into a chronic dysentery. In this the symptoms of a chronic colitis, usually attended with cachexia, may persist for months and years.

Mild, rudimentary forms of dysentery also occur, presenting no severe intestinal symptoms, and recovering at the end of a few days. In these cases, too, great sensitiveness of the intestine to disturbing influences frequently persists for quite a long time after the illness. There may be exacerbations of the disease, and relapses.

Complications of dysentery, localized in other organs, are rare, at least in epidemics here. In tropical dysentery abscess of the liver is comparatively common. It is dependent upon metastatic processes from the portal radicals. In some cases abscesses of the lungs and of the brain may follow abscess of the liver. Inflammation of the serous membranes or of one or more joints may also occur. If paraplegia follows it may be referred either to secondary myelitis or to polyneuritis. Dysentery is also said to occur in connection with a "general scorbutic diathesis," but to all appearances this is usually a "septic" complication. The dysenteric ulcers rarely lead to perforation and consequent peritonitis.

The **diagnosis** is seldom very difficult. It is based exclusively upon the intestinal symptoms and the character of the stools. It is only the cases of secondary dysentery which occur in the course of other severe diseases that are likely to escape observation.

The **prognosis** is mainly influenced by the character of the epidemic, which, as we have said, is in our climate usually benign. There may be danger, particularly to elderly people, from bodily weakness and collapse.

Treatment.—Prophylaxis demands that the isolation of the patient and the disinfection of the stools be as complete as possible. The healthy must be very careful during an epidemic not to catch cold, and to avoid errors in diet, for experience shows that an opposite course predisposes to the disease.

The patient must be kept warm, and must not leave his bed, even if the attack be mild. The diet must be rigorous. If the strength is fair, thin porridge, milk, and broths suffice for some days. To a feebler person we should give somewhat stronger nourishment from the start, e. g., eggs, peptonized meat, and wine. Most patients bear liquids that are lukewarm better than those which are cold.

As to drugs, the habit of almost all experienced physicians is to give at first a mild laxative. Although opium does not usually control the diarrhœa and tenesmus at all, it is the rule for decided improvement to follow the exhibition of the laxative. During the first days, or, if need be, later, we give two to four tablespoonfuls of castor-oil daily. If this medicine is very disagreeable to the patient, we can replace it by a strong infusion of rhubarb (10–100). In southern countries large doses of calomel (gr. x to xv, grm. 0.5–1) are customary, and are highly praised by the physicians there. Further on in the disease we may content ourselves with giving mistura amygdalæ; or we may administer bismuth in the following mixture: Bismuthi subnit. vel salicylat., grm. 5; mucilaginis acaciæ, syrupi simpl., āā 15; aquæ destil., 120—to be shaken before taking. If the disease should get worse again, however, we should always try a laxative.

Emetics at the beginning of the disease are often employed in the tropics, but seldom with us. Ipecacuanha (*radix antidysenterica*), given in large doses of fifteen to thirty grains (grm. 1–2), is even regarded by many as a specific. Among antiparasitic remedies, naphthalin (gr. viij, grm. 0.5, thrice daily) and salol (one to two drachms, grm. 4.0–8.0, a day) have been especially recommended. Numerous attempts have been made at local treatment by enemata. No brilliant results, however, can be claimed for any of these methods or medicines. A decided palliative effect can be obtained from the injection of thin starch to which twenty or thirty drops of laudanum have been added. Suppositories of cocoa butter containing extract of opium often mitigate the tenesmus. Other injections are recommended, each to measure ℥ij to iijss. (grm. 60–100), and to contain either argenti nitrat., gr. j to vj (grm. 0.05–0.30), or plumbi acetat., gr. ij to viij (grm. 0.1–0.5), or potassii chlorat., gr. xv to xx (grm. 1–1.5), and especially tannin (three injections a day of a warm 0.5-per-cent. solution). In severe cases we may also try high injections of solutions of tannin as in cholera (*vide infra*). Many other solutions are used. The success of this treatment is, however, dubious.[1] In all cases the margins of the anus must be protected from inflammation by frequently washing and anointing the skin.

The treatment of weakness and collapse is by the usual stimulants—wine, ether, camphor, and the like. In chronic dysentery the main point is to persevere in a strict control of the diet. We may exhibit astringents, such as tannin and columbo. Subnitrate of bismuth is also given, and nitrate of silver and acetate of lead. And in these chronic cases a long-continued and thorough use of rectal irrigation with fluids containing some mild astringent or disinfectant may have a good effect.

[Sporadic dysentery is a self-limited disease, and, as has been shown by Flint, runs its course within ten days without medication. Treatment, however, adds to the comfort of the patient and shortens the course. It is not customary with us to use daily laxatives. If there is any doubt as to whether the intestines have been emptied, a saline should be given, the action of which should be followed by opium in sufficient doses to allay pain and tenesmus. Subsequent action of the bowels is best obtained by simple large enemata. In weak persons castor-oil is to be preferred to salines.

In epidemic dysentery active treatment is much more important. Laxatives are contra-indicated by sero-sanguinolent dejections or by asthenia, but enemata can be freely used. Stimulation is often required; nutrition must be carefully looked after, such articles being chosen as are digested and absorbed by the upper portions of the intestinal tract, leaving as little residue as possible to pass on to the inflamed colon. Opium is often demanded and tolerated in large doses, and astringents, such as the acetate of lead, gallic acid, and the pernitrate of iron, are of service.

In acute dysentery the patient should be instructed not to yield to the desire to go to stool if he can help it, and tenesmus can often be much diminished by simple irrigation of the lower bowel with water, which may be warm or cold, whichever the patient finds more agreeable.

Chronic dysentery is one of the most difficult maladies with which we have to deal. In its treatment a sea voyage, or removal for at least some months to a climate other than that in which the disease originated, is of far more value than drugs.

Amœbic Dysentery

The amœba coli, first found by Lösch in the stools of a dysenteric patient, has received very careful study at the Johns Hopkins Hospital, and forms the subject of a most exhaustive and valuable monograph by Councilman and Lafleur.

The living organism is readily seen, and recognized especially by its active amœboid movements on the warm stage of the microscope. If the fæces contain small gelatinous masses, these will be found to provide the most fruitful field for search. The numbers of the organisms vary widely in different cases, and even in the same case, from day to day. They are said to be present in this form of dysentery alone, and have been found in secondary abscesses of the liver and the lung, alike after death and during life. They should be sought for in all obstinate cases with dysenteric symptoms.

The prognosis is uncertain, and the cases are apt to drag along with exacerbations and remissions of the symptoms; but recovery does take place. There is notable danger of the formation of secondary abscesses, in which event recovery is hardly to be hoped for. The only treatment which seems to have been of much service—beyond a general hygienic and supportive regimen—consists in the use of copious injections into the intestinal canal of solutions of quinine, in the strength of one to one thousand or even five thousand. Lösch found that contact with a solution of the latter strength for one minute suffices to kill the amœbæ.]

CHAPTER XIII

CHOLERA

(Asiatic Cholera)

Historical Remarks.—The home of genuine Asiatic cholera is India. The first epidemic in that country with which we are accurately acquainted occurred in 1817. This was very widespread. The disease was probably endemic there at an earlier period. In the next few years the cholera extended in all directions, and reached Astrakhan by way of Persia. Between 1830 and 1832 the disease made its first great epidemic progress over Europe. Invading all European Russia, it reached Germany in 1831, and France and England in 1832. Then came many smaller epidemics up to 1838, when there was a complete cessation till 1846, in which year the disease, again starting from Asia, overspread Europe. There have in later years been epidemics in many places, but we can not here enter into the particulars of them. During the war of 1866 there were many cases of cholera in Germany, and from 1883 to 1886 Italy, France, and Spain were visited by the disease. In August, 1892, cholera broke out suddenly and very unexpectedly in Hamburg, where within three months about 18,000 persons were attacked by the disease, and over 7,600 died of it.

Ætiology.—Some time ago it had become evident that the real cause of cholera consists in the infection of the system by a specific micro-organism. Koch was, however, the first to succeed in the search for the poisonous agent. He was in charge of the scientific expedition sent out by the German Government in 1883 to Egypt and India for the purpose of investigating the disease. Koch found in the intestines of all the victims of cholera whose bodies he examined a certain kind of micro-organism which he named the comma bacillus. It is shorter than the bacillus of tuberculosis, but somewhat thicker, and it is usually bent in the shape of a comma, or even like a semicircle (see Fig. 9). In pure cultures the comma bacilli grow into long spiral threads, resembling the spirilli of recurrent fever. Examined in a liquid, the individual bacilli are seen to make vigorous movements. This mobility is probably dependent upon the thin, filiform fibers at the ends of the comma bacilli discovered by Löffler.

The comma bacilli flourish best at a temperature between 86° and 104° (30° and 40° C.). Below 61° (16° C.) they cease to grow, but they are not killed even by a greater degree of cold. The free access of oxygen is desirable but not absolutely indispensable to their growth. They multiply very rapidly in liquids—e. g., broth or milk—and they may, under favorable circumstances, retain their vitality for many weeks, while they can be readily destroyed by desiccation. In this again they resemble the genuine spirilli, which can maintain their existence only in fluids. It is necessary that the nutrient material have an alkaline reaction. On neutral or acid material the growth of the comma bacilli ceases entirely. The characteristic features of pure cultures can not be described in detail here, but we may observe that the nutrient gelatin is slowly liquefied by the bacilli. Nothing definite is yet known as to the occurrence and the formation of permanent spores.

There can now be no doubt that the sole cause of cholera is infection by the comma bacilli. It has been shown that in every case of genuine Asiatic cholera the comma bacilli are present in the intestine, and that they are never found under any other circumstances. Even the last postulate which was needed to show their pathogenic significance has been fulfilled. Rietsch and Nicati, followed by Koch himself and others, have succeeded in producing cholera in a guinea-pig by introducing into its duodenum or into the contents of the stomach previously made alkaline, pure comma bacilli.

Investigation as to the origin of cholera must, therefore, now meet this culminating question: Under what circumstances and through what channel do the comma bacilli penetrate into the human system, and in what manner do they there excite the characteristic processes of the disease? There can be no doubt that among Europeans, and probably everywhere except in India, the cholera is invariably imported. It is equally certain that the dejections of cholera patients, which are rich in comma bacilli, are the chief if not the only agent by which the disease is spread. The bacilli which escape into the outer world with the stools find abundant means to prolong their existence. They continue their growth upon moistened bed-clothes, or in water which contains a sufficient amount of organic substances, or in food, such as fruit or milk, or in moist earth; and the ways by which they can in turn enter the system of a healthy human being are infinite in number. It is easy to understand why certain persons—e. g., laundresses and nurses—are more liable to infection than others; and it is equally intelligible that the spread of the disease should often bear a relation to certain outward circumstances. The fact has long been a familiar one, that the cholera almost always progresses along the world's most frequented highways, and that it never travels faster than the means of human intercommunication render possible. This is important, because it shows plainly that the germs of the disease are not disseminated by currents of air. It is easy to understand that the distribution of the disease should sometimes correspond with that of water destined for personal use. The experience of the last few years has again proved most clearly that the drinking-water is the chief, although of course not the only, source for the dissemination of cholera. If an extensive water-supply is contaminated by comma bacilli, as was the case in Hamburg, the disease may suddenly break out in a place with great severity. Sporadic cases usually arise from the use of a contaminated stream which is used for drinking only by a small number of men (sailors, etc.).

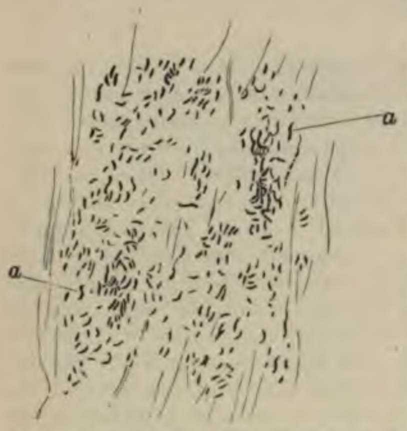

FIG. 9.—(From KOCH.) Comma bacilli from a cholera dejection which had lain for two days on a wet cloth. The S-shaped bacilli are at *a*. 600 diameters.

A man falls sick with cholera, of course, not simply from swallowing the cholera bacilli, but because the bacilli remain in the intestine and multiply. It is safe to assume that many men swallow comma bacilli at the time of a cholera epidemic without falling sick at all, or without any but the slightest disturbance, because the comma bacilli are at once destroyed by the acid gastric juice or they develop in the intestine only in small numbers or perhaps in an attenuated form. In the last epidemic at Hamburg comma bacilli were sometimes found in the solid stools of healthy men, who were in close contact with cases of cholera, and also in the stools of men who had a very mild "cholera diarrhœa." These facts are extremely important. They lead to a correct estimate of the infection experiments repeatedly practiced of late on human beings, and, on the other hand, they have a great practical significance in regard to the danger of transmitting cholera.

These theories may be brought into harmony with the observations collected by Pettenkofer, which go to show a connection between the dissemination of cholera and the character of the soil. The character of the soil can evidently influence the dissemination of the cholera bacilli in various ways. The conditions of the water in springs, etc., may depend very materially upon the character of the surrounding soil. The epidemiological facts especially emphasized by Pettenkofer are: 1. Certain places are immune, especially those lying in a rocky terrain. 2. The frequency of cholera (as of typhoid fever, *vide supra*) corresponds with variations in the ground-water.

Most cholera epidemics happen in the summer months. Liability to the disease is very widespread, although some remarkable exceptions are seen. Sex is unimportant. Age has more influence. The disease occurs in sucklings, but, as a rule, is more rare among children than among adults. Elderly people are very apt to take the disease, while of typhoid fever the opposite is true. Most authors lay great stress upon predisposing causes. Among these, taking cold is not so important as are errors in diet and mild attacks of gastro-intestinal catarrh, which are shown by numerous observations to predispose strongly to the disease, because the acidity of the gastric contents is thus diminished, and the retention of comma bacilli in the intestine is thus facilitated. The stage of incubation seldom lasts over one to three days at most.

Clinical History.—In cholera, as in most acute infectious diseases, the intensity of the illness varies between the extremes of mildness and severity, so that usually a correct interpretation of the mildest cases is rendered possible only by the fact that an epidemic exists and by the discovery of comma bacilli. These insignificant cases are called simple choleraic diarrhœa. The symptoms are those of a violent acute intestinal catarrh; the dejections are watery, rather large, painless, and number about three to eight in twenty-four hours. There is considerable malaise, complete anorexia, and thirst, and there may already be indications of severer choleraic symptoms—vomiting, slight pains in the calves of the legs, and diminished secretion of urine. Many cases recover after a few days or a week, but in others the first mild diarrhœa is succeeded, at the end of about one to three days, or rarely later still, by a severe attack of cholera. In such cases we speak of a "premonitory diarrhœa of cholera."

The mild form is succeeded in a gradual transition by the cases designated as "cholerine." Cholerine exhibits the symptoms of a violent, rather sudden cholera morbus. It often begins at night. To the diarrhœa, which now and then displays even at this time the characteristics of pronounced cholera, vomiting is soon added. The accompanying constitutional symptoms are rather severe. There is great languor and depression. The voice grows weak, the extremities are cool, the pulse is small and accelerated, painful cramps occur in the calves of the legs, the urine grows scanty and perhaps albuminous. The whole attack lasts about a week or two, before recovery is complete. The course of the disease is not infrequently varied by repeated improvements and relapses.

From these cases of medium severity there is again a continuous line of transition to the pronounced severe form of cholera. Statistics as to the frequency of the separate forms can not be given, since many of the milder cases escape observation.

The true attack of cholera may begin suddenly with the severest symptoms. As a rule, however, it is preceded, as already stated, by a first stage of brief premonitory diarrhœa. This, after one to three days, is replaced with equal suddenness by the severe symptoms of the second or "algid stage," or "cholera asphyxia." Its first symptoms are the abrupt appearance of great bodily weakness, chilliness, and vertigo. The characteristic gastro-intestinal symptoms soon declare themselves.

The diarrhœa grows very violent. At short intervals there are copious painless dejections, which at first retain somewhat of a feculent character, but very soon present a characteristic resemblance to rice-water, gruel, or whey. A single stool will measure a little less than half a pint (grammes 200). The stools have no color and almost no odor. They are watery, and usually deposit a finely granular, grayish-white sediment upon standing. Their reaction is neutral or alkaline. Only one or two per cent. is solid matter, with a little albumen and a relatively large amount of sodic chloride. In many severe cases the dejections contain more or less blood. The microscope reveals epithelium, triple phosphate, and numerous micro-organisms. Of these last a part are the comma bacilli, and a part are bacteria of putrefaction, etc.

These excessive evacuations are but very rarely absent. They are more apt to fail if death occurs at the end of a few hours—*cholera sicca.*

[In cholera sicca the intestines after death contain the characteristic rice-water material which, perhaps owing to paralysis of the muscular coat, was not expelled during life.]

The appearance of the diarrhœa is soon followed by frequent though rarely distressing vomiting. The vomitus consists in part of ingested liquids and in part of an actual transudation through the mucous membrane of the stomach and intestine. Hiccoughs may accompany and follow the emesis.

In addition to these prominent digestive symptoms of vomiting and profuse diarrhœa there are complete anorexia and excessive thirst. The tongue has a thick, dry coat. The abdomen is usually flat and soft, or it may be concave and hard. Sometimes we may feel fluctuation in the intestines, due to their being filled with fluid. There is not much real abdominal pain; what there is, is described as a "feeling of heat and pressure" around the umbilicus.

At the same time very severe symptoms develop in other organs. The circulatory system is chiefly affected.

The action of the heart may be stimulated at the beginning of the attack. The patient complains of palpitation and great precordial anxiety. After a brief time, however, cardiac weakness appears, and continually increases. The action of the heart becomes very weak, and the heart-sounds feebler and feebler. The pulse at the wrist grows very small, and is usually somewhat accelerated. In a severe case the pulse vanishes completely after a few hours.

This collapse of circulation makes itself quickly evident in the appearance of the patient. The face and extremities grow cool, and then ice-cold; the complexion becomes partly livid and partly a bluish gray; the lips are almost black. The surface temperature may fall below 95° (35° C.), while in the rectum febrile temperatures may often be observed, reaching 102° (39° C.) and higher. The eye and cheek grow very hollow, the skin becomes wrinkled, and loses all its elasticity. The voice grows hoarse and feeble (voice of cholera). Respiration is laborious and superficial. The mind may remain unclouded to the end, but usually there is great apathy, and all acuteness of perception is destroyed. Only a few patients are restless and excited. Reflex action is much impaired.

One characteristic symptom is the cramps in the muscles. These are usually very painful, and consist in tonic contractions of the muscles, particularly those of the calf of the leg, but also those of the toes, thighs, arms, and hands. The cramps occur spontaneously or upon the least provocation, last a few minutes, and recur at short intervals. The precise reason of their occurrence is not yet known. It may be the effect of poison (*vide infra*). They can be observed in other severe acute diseases, although most marked in cholera. They sometimes occur in cholera morbus.

In a well-developed attack of cholera there is almost invariably oliguria or anuria. The urine, if any be secreted, is concentrated, with abundant sediment, and it very often contains albumen. In many cases not one drop of urine reaches the bladder for days, and this condition persists till death or recovery.

The symptoms thus far depicted, if taken as a whole, represent the algid stage, which seldom lasts more than one or two days. In many cases death occurs during this period. It is ushered in by the tokens of extreme general prostration, and may take place after a few hours, or more frequently in the second half of the first day. But in other cases the "stage of reaction" succeeds. This may be a true compensatory period, leading directly to convalescence. The evacuations become less frequent and more feculent, and the vomiting ceases. The pulse becomes stronger, the cyanosis and coolness of the extremities diminish, and an abundant perspiration is not infrequent. After a few days urine is again excreted. This is almost invariably quite albuminous, and usually contains casts and red blood-globules. If convalescence be uninterrupted, however, the urine very soon becomes perfectly normal, and after a week or two recovery is to be regarded as complete.

Departures from this favorable course of the stage of reaction are frequent. Recovery may be interrupted by repeated relapses into the previous condition, sometimes with a fatal result; instead of convalescence, there is developed a severe third stage, usually with fever. This stage ordinarily bears the generic name of cholera typhoid, although it is subject to manifold variations in its clinical symptoms as well as its exciting causes.

Cholera typhoid may present an actually typhoidal general condition with severe fever. There is a considerable elevation of temperature, headache, and dullness. The pulse is full and rapid, the face flushed. The skin, particularly that of the extremities, sometimes presents the so-called choleraic eruption, in the form of an erythema, roseola, urticaria, or the like. This variety of cholera typhoid ends after a few days in recovery, or else passes into one of the following conditions.

A second form of cholera typhoid is distinguished by the development of the most diverse local inflammations. Thus, there may be a severe dysenteric or diphtheritic inflammation of the small and large intestine, attended by offensive purulent and bloody stools. Pneumonia is also possible, as well as purulent bronchitis, diphtheritic inflammation of the larynx, pharynx, bladder, and female genitals, parotitis, and sometimes erysipelas and pyæmia. And when we consider that, besides all these conditions, the usual intestinal symptoms, or those of choleraic nephritis, may exist also, it is evident how varied the clinical picture may be. The development of these local affections frequently lays the foundation for numerous sequelæ.

Choleraic nephritis gives rise to the third or uræmic variety of cholera typhoid. The secretion of urine is almost suspended. The region of the kidneys is sometimes sensitive on pressure. The little urine that is still passed contains numerous casts, albumen, and frequently renal epithelium and white and red blood-globules. Somewhere toward the end of the first week, or possibly earlier, there are grave nervous symptoms, to be regarded as uræmic: first there is headache and vomiting, then sopor and coma, or delirium and convulsions. Most of these cases are fatal.

Pathology and Pathogenesis.—We are now acquainted with the manifold symptoms and varieties of the disease. If we seek for the pathological changes which control the process, and endeavor to find some correspondence between them and the symptoms, we shall be disappointed. At least, in its early stages, cholera is merely a severe local disease of the intestine. We find the serous layer of the coils of the small intestine rose-red from congestion. The mucous membrane is in a state of catarrhal inflammation: it is swollen, reddened, and at first covered with a layer of tough, transparent mucus; but very soon an abundant transudation flows into the canal, so that the intestinal coils are filled with a large amount of clear fluid, looking like rice-water or gruel, and so devoid of bile as to indicate the suspension of its secretion. The signs of inflammation of the mucous membrane now grow more pronounced. The solitary follicles and Peyer's patches become swollen, with edges of a vivid red, and frequently there are many small ecchymoses in the mucous membrane. The extensive desquamation of the epithelial lining of the intestine has also been regarded as important, because it was regarded as in part the cause of the copious transudation. Still it may be questioned whether the desquamation is not, at least to some extent, a post-mortem change. In yet later stages of the disease the intestinal trouble very frequently assumes a croupous-diphtheritic character. The surface is necrosed and ulcerated in many places, and the contents of the intestine are no longer colorless, but sanious and bloody, with a foul odor.

Otherwise most of the post-mortem lesions correspond to what was obvious at the bedside. The muscles exhibit an early and persistent rigor mortis, and frequently contract in such a way as to throw the corpse into some unusual posture. All the internal organs are remarkably dry, pale, and anæmic. The left ventricle is contracted. The blood lies mostly in the large veins, the right side of the heart, and the cerebral sinuses. It is thickened, is but little clotted, and is said to resemble the juice of bilberries or huckleberries. The spleen is not enlarged—an exception to the rule in infectious diseases. The kidneys present marked passive congestion, most pronounced in the cortex. The microscope reveals a greater or less degree of parenchymatous nephritis, with great destruction of the epithelium. If death takes place at a rather advanced stage of the disease, the tissues have lost their characteristic dryness, and the most diverse local lesions, including nephritis, may be found to have occasioned death.

If we search for the connection between the pathological changes just described and the cause of the disease, or again between these lesions and the clinical symptoms, the first point to guide us is that the comma bacilli are found only in the lumen of the intestine, and never in the blood or in other parts of the body. The intestinal symptoms are satisfactorily explained by this abnormal state of the intestine, but for all the other grave symptoms we have to seek some special cause. The desiccation which the body undergoes as a result of the excessive liquid dejections can not fail to affect the tissues, but can not fully explain the symptoms, for the circulatory disturbances and the cardiac failure at least may develop before large evacuations have occurred. It has also been settled beyond question by means of the newer investigations that precisely the worst symptoms of cholera—namely, the muscular cramps, the subnormal temperature, and the changes in the blood—are occasioned by the chemical results of tissue metamorphosis in the comma bacilli, that is, by the so-called toxines. Some of these have been already isolated chemically by Brieger and others. It is an interesting circumstance that the amount and virulence of the toxines formed by the comma bacilli seem to depend on the physical characteristics of the nutrient material on which the culture grows.

As to the complications which occur in the later stages of the disease and which are embraced under the generic name of cholera typhoid, we regard them as mainly secondary. The choleraic process itself does not cause them, but is merely the occasion for their appearance. The examination of the intestine in such cases shows that numerous other varieties of bacteria follow closely upon the comma bacillus, gaining entrance to the system by treading in its footsteps. Cholera nephritis is probably due to various causes. Some forms of nephritis seem to be caused by specific cholera toxines (in analogy with scarlatinal nephritis), while others are probably of a secondary septic nature.

Diagnosis.—A positive diagnosis of cholera can be made only by finding the comma bacilli in the stools. When an epidemic prevails it is often neither necessary nor possible to make the search in each individual case. Sporadic cases or the first cases of a beginning epidemic can and must be diagnosticated only in this way, which gives absolute certainty. We can not give here a detailed account of the bacteriological diagnosis, but we may state briefly that, when cholera exists, the simple microscopical examination of a smear preparation sometimes makes the diagnosis extremely probable. For this purpose a small clump of mucus is taken from a stool as fresh as possible, spread on a cover-glass as thinly as possible, fixed by passing it carefully through the flame several times, and stained with an aqueous solution of methylene blue or carbol-fuchsine. In actual cholera we then find the comma bacilli sometimes in large amount (almost a pure culture) or especially arranged in characteristic small groups. To render our diagnosis unassailable we must employ gelatine or agar plate cultures, etc., the details of which are to be found in the text-books of bacteriology.

All those affections whose symptoms are like those of cholera may be confounded with true cholera, especially cholera morbus (*vide infra*); and also certain cases of poisoning, especially acute arsenical poisoning, may give rise to symptoms wonderfully like cholera.

The **prognosis** should always be guarded at the beginning, even if the symptoms be mild, for, as already mentioned, a simple diarrhœa may prove to be "premonitory" of a severe attack of cholera. During the real attack the prognosis grows graver in proportion as the case presents the characteristics of asphyxia and cyanosis. The mortality in many epidemics is frightful. All the inhabitants of a house or street may in a brief period be swept away. Accurate statistics are difficult to give. If we count the typical cases alone, the mortality is not infrequently fifty to seventy per cent. In about two thirds of the fatal cases death occurs during the first days of the stage of asphyxia, and in about one third during the second period, known as "cholera typhoid." The influence of the diet and the hygienic surroundings of the patient before his illness is important. A greater proportion of children and old people perish than of the middle-aged.

Prophylaxis and Treatment.—The measures to be taken to prevent the spread of the disease, when it has once started in a place, can be only briefly discussed here. The chief things are the quickest isolation possible of the first cases that occur, the disinfection of the stools and all linen, objects, etc., soiled by the stools, and finally the determination of the source of infection (drinking-water, etc.), in order to prevent further infection. The evacuations (stools and vomitus) can be best disinfected with a five-per-cent. solution of carbolic acid or with milk of lime, chloride of lime, etc.; the linen and other objects should be disinfected in a special disinfecting apparatus. It is very important to keep a strict oversight of the drinking-water, milk, and all articles of food eaten in an uncooked condition.

In regard to individual prophylaxis, we must remember especially that any slight gastric or intestinal catarrh increases the liability to the disease. Hence, at the time of a cholera epidemic, careful dietetic restrictions are imperatively necessary and every disturbance of the stomach or the intestines, even the slightest, needs the promptest and most careful treatment. It is best wholly to avoid the use of water that has not been boiled, raw vegetables, etc.

[Various efforts have been made to render human beings immune to choleraic infection. The best results seem to have been attained by inoculating Haffkine's attenuated virus.—V.]

In the treatment of the cholera attack itself many physicians even now use opium (laudanum or pure opium in powder, gr. ss.–j, grm. 0.03–0.05), while others, especially at the beginning of the disease, prefer large doses of calomel (gr. v–x, grm. 0.3–0.5, several times), and during the subsequent course they give continued small doses of calomel (gr. ss.–j, grm. 0.03–0.05, every two hours). In the algid stage warmth is especially advisable. Hot baths, hot packs, sweat-boxes with dry or moist hot air, embrocations of hot oil, an abundant supply of hot drinks (coffee, tea, mulled wine, broths) are praised by all physicians, and they must certainly be used. The remaining treatment is symptomatic; morphine or ice for the vomiting and embrocations of chloroform oil or subcutaneous injections of morphine for the painful cramps in the calves. The weaker the heart becomes, the more energetically must we give stimulants (injections of camphor or ether, champagne).

Among the newer methods of treatment tried during the last epidemic we may mention subcutaneous infusions of warm "normal" (0.6 per cent.) salt solution in the infraclavicular region or under the skin of the abdomen. By uninterrupted infusion by means of an irrigator or a funnel two or three quarts (litres) of salt solution can be infused in twenty-four hours. Intravenous injections have given still better results, and injections into the abdominal cavity have also been tried. Cantani strongly recommends intestinal injections ("enteroclysis") with warm tannin solutions at a temperature of 100° to 104° F. (38°–40° C.). The fluid contains 5 to 10 parts of tannic acid, 50 parts of gum arabic, and 2 or 3 parts of laudanum to 2,000 parts of water. All these methods are believed to have shown some favorable results, but none of them has been able to attain general recognition and use. After many experiments most physicians have finally gone back to the old forms of treatment.

[The vital importance of the serious treatment of a beginning diarrhœa during a cholera epidemic can not be too strongly insisted on. Rest, simple diet, and a little medication will, in the vast majority of instances, entirely prevent grave consequences. The apparently trifling character of the symptoms is apt to lead people into a false security. Those who can leave an infected district should do so without delay.

With reference to the prevention of an epidemic, a pure water supply and strict cleanliness in its broad sense possess far more virtue than cordons of troops or measures of quarantine. It is more practicable to destroy the soil than to keep out the seed in these days of constant and rapid international communication. The systematic disinfection of all cholera discharges or articles soiled by them should be a matter of course.]

Great caution must be exercised about the diet, not merely during the attack itself, but for a considerable time afterward. At first we can allow only thin porridge, milk, broths, and toast or rusks. It is advisable to administer dilute hydrochloric acid with the food.

The treatment of cholera typhoid varies greatly, of course, according to the kind of attack. The separate complications should receive their customary treatment.

[In the first stage, absolute rest, opium, and lumps of ice by the mouth *ad libitum* are the chief measures on which reliance is to be placed. It should be remembered that the entire function of the intestinal tract is reversed; thus, instead of an absorbing, it has become an excreting surface.

In the stage of collapse the nervous system is more or less paralyzed, the blood is damaged by the loss of its watery constitutents, and the circulation of that fluid is greatly impeded. The subcutaneous or gastric absorption of drugs is consequently delayed or suspended. The utility of any active internal treatment during this stage is very questionable. Certainly narcotism by opium is highly undesirable. Mild external stimulation and the tentative administration of ice and small quantities of champagne or food are, at all events, not likely to do harm. Nature sometimes reasserts herself when the conditions are seemingly desperate, and the third stage, or that of reaction, comes on. In this stage, careful nursing and a sensible symptomatic but in no way meddlesome treatment are most likely to be followed by good results.]

CHAPTER XIV

MALARIAL DISEASES

(Intermittent Fever. Fever and Ague. Swamp Fever)

Ætiology and Pathological Anatomy.—Malarial poisoning is the best example of a purely "miasmatic" affection. The poison which produces the disease is without doubt localized in certain places, in which every human being is liable to become its victim; but if an infected person comes to a place free from malaria and not naturally favorable to its development, there is no danger that he will cause the disease in others. The disease is never caught through contact with the patient. It is not at all contagious; the malarial poison, after it has once penetrated into the body, has practically no opportunity to escape again in an efficient form from the diseased system into the outer world; but the blood of a patient injected into a healthy person may transfer the disease (Gerhardt and others).

If we except the polar zones, there are few regions where malaria is not endemic in certain parts, at least from time to time, if not constantly. There is, however, great variation in the virulence as well as in the number of cases. While the common forms of intermittent fever are very frequent in Germany, in numerous places, especially on the shores of the North and Baltic Seas, and also in the alluvial lands of the Vistula, Oder, Elbe, etc., yet the grave forms of the disease are very rare. Other lands are notorious for the severe malarial diseases, e.g., Hungary, the lands lying on the lower Danube, the Roman Campagna, the Pontine marshes, Sicily, and numerous districts in other parts of the world, chiefly tropical. Numerous observations have only served to confirm the opinion that the soil is the true home and cradle of the malarial poison, and that the virus, escaping thence into the lower strata of the atmosphere, may be taken into the system, probably during inspiration. Permanent dampness of the soil is essential to the development of the malarial poison. This explains why marshy districts are so often malarial. The ground must not be covered by a great amount of water, but must during the dry season lie exposed to the atmospheric air. The access of air to the moist soil seems to be a second essential condition for the development of the malarial germs. A third influential factor is the temperature of the air, as proven by the great prevalence of the disease in southern countries and in the summer season.

The special agent which causes malaria was first described by Laveran in 1881, and soon after by Marchiafava and Celli. Since then many other observers, especially in Italy, have investigated the subject and have given us very interesting information about these peculiar parasites. Besides the writers named we may refer to the studies of Golgi, Guarnieri, Canalis, etc. The parasite belongs to the protozoa (whether to the sporozoa or rhizopoda is still uncertain), and is now generally called the *plasmodium malariæ*. The organisms are found in the blood of men sick with malaria, in unstained specimens or in specimens stained with methylene blue, and are at first inclosed in the red blood-corpuscles. Here they appear as delicate protoplasmic bodies with an indication of a nucleus; at rest they have a rounded form, but they may assume various shapes through their amœboid movements (see Fig. 10). These movements probably have some relation to the nutrition of the parasite, which takes place at the expense of the red blood-corpuscle that shelters it. The hæmoglobine taken up by the plasmodium is thus changed to melanine, which probably contains no iron, and the black granules of this melanine can easily be seen in the body of the adult parasite. The second phase in the life of the plasmodium now begins and serves for its propagation and increase. The amœboid movements cease, the pigment granules collect in a ring about the periphery, in radiating stripes, or in the center of the parasite, while the rest of the mass in a few hours breaks up into a number of rounded spores. Their inclosing capsule disappears, the spores are set free in the blood and penetrate other red blood-corpuscles.

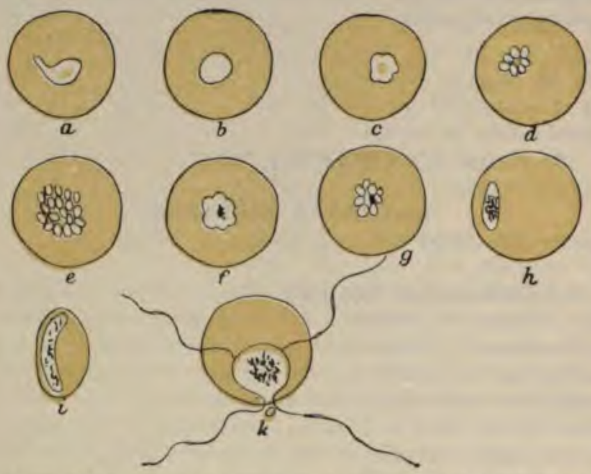

FIG. 10.—Different forms of the malarial plasmodium according to Marchiafava and Celli. *a*, *b*, *c*, ordinary plasmodia without pigment. *d*, *e*, spore formation in plasmodia. *f*, *g*, pigmented plasmodia with spore formation. *h*, *i*, oval and crescentic forms of plasmodia. *k*, pigmented plasmodium with flagellæ.

Besides these chief forms of the malarial plasmodium just described, there are also other forms (crescentic form, flagellated form) whose relations to one another and to the ordinary type are still obscure. This fact alone seems to be certain that the different clinical forms of malaria—tertian, quartan, pernicious, etc.—correspond to different biological forms of the plasmodium. By inoculation of the blood into another human being we can therefore produce nothing but a tertian from a tertian or a quartan from a quartan. The quotidian fever is due to two tertians co-existing in the same person, each individual tertian owing its origin to a special generation of the plasmodium. The continuous and pernicious malarial fevers are characterized by the presence of many of the crescentic forms of development.

Although no one has yet been successful in establishing this view of the plasmodia by means of pure cultures and inoculation of the same, yet the relation of the plasmodium to malaria seems evident from the fact that in every patient with malaria the plasmodia can be easily demonstrated in the blood, while these same forms are never detected in the blood except in malaria. The way in which the plasmodia enter the human body is not yet known. It may be by inhalation or by the stings of insects.

[It has now been proved that malarial organisms can be communicated by certain varieties of mosquito (*anopheles claviger* and *a. pictus*) to man, and also by man to the mosquito. The obvious deduction is that a healthy man should guard himself from mosquitoes for his own sake and an already infected man for the sake of others.—V.]

The investigations with regard to the plasmodium of malaria have also explained the long-recognized fact that in chronic malaria large amounts of pigment collect in the internal viscera. These deposits are most abundant in the spleen, which in the chronic varieties of the disease develops into a firm, hard tumor; but they are also found in the bone-marrow, liver, brain, and kidneys, leading finally in the liver and the kidneys, in frequent instances, to processes of chronic degeneration and inflammation. It is especially noteworthy that in those patients who present the most marked cerebral disturbances (pernicious comatose fever, *vide infra*), the cerebral capillaries are found to be completely occluded with pigmented plasmodia.

[Periodical fever is very widely distributed in the United States, and in the southern portions occurs in severe though not in the severest forms. Some regions which were formerly free from it are no longer so, and, *vice versa*, some regions which were greatly subject to it are now exempt; these changes are closely connected with the clearing and upturning of virgin soil largely impregnated with decaying vegetable matter, and with the subsequent cultivation of the same for considerable periods of time. The poison does not extend far above the surface of the ground, as is shown by the relative safety of sleeping in the upper as compared with the lower story of a house; during the night the poison seems to exist in greater intensity than during the day. Attacks are more liable to occur during the spring and autumn than at other seasons.

The hopes which have been entertained in some quarters that malarial regions might be rendered healthy by large plantations of the Australian eucalyptus globulus, a rapidly growing tree which absorbs immense quantities of water, do not seem likely to be realized in the light of French experience in Africa, and in that of the Trappist monks in Italy.]

Klebs and Tommasi-Crudeli have made extensive investigations as to the nature of the malarial poison. We must regard it as organic. The authorities just named state that the true cause of malaria is a specific variety of bacillus. They found peculiar bacilli and their spores both in the earth of malarial regions and in the adjacent strata of the atmosphere; and, by infecting rabbits with these, they were able to induce attacks of fever, swelling of the spleen, and the characteristic formation of pigment matter (*vide infra*). Before this, bacilli and spores had been found in the blood and spleen of patients suffering from malaria. It can not yet be said just how much significance these discoveries have.

[To Laveran really belongs the credit for the discovery and accurate description of the plasmodium. His work in Algiers has been substantiated by observers in widely separated malarial regions, and, at the same time, no very important additions have been made to it.

It is difficult to overrate the diagnostic and therapeutic importance of this discovery. The ordinary intermittents are usually easy of diagnosis, but the remittent forms often simulate typhoid fever, while the pernicious form may resemble uremia or intracranial disease. Especially in pernicious cases life may be saved by prompt and appropriate treatment, which must be based, of course, on accurate diagnosis. The following details may be of use to those who are not familiar with the method of blood examination. The finger tips should first be scrubbed with a nail-brush, and soap and water, and then washed in alcohol or ether, to obviate the danger of mistaking minute particles of dirt for the pigment-granules derived from the destruction of red blood-corpuscles. A portion of a drop of blood is then to be received on a scrupulously clean object- or cover-glass, and squeezed out between the two, so that a thin layer of blood with separation of the individual corpuscles is secured. The amœboid forms of the plasmodium retain their movements for a number of minutes in a warm room, much longer, of course, with a warm stage. The presence of pigment-granules in the red corpuscles may be the readiest indication of the presence of the organism. The crescentic forms are found more especially in chronic cases which have been under treatment by quinine, as is stated by Osler. Many of the other forms can be found in the blood only during a paroxysm. The editor has seen them in abundance in a cachectic malarial patient from Panama, and failed to find them again after a single large dose of quinine, which terminated the chills.

In dried and stained specimens it is also possible to detect the organisms, but amœboid movement is, of course, lost.]

Liability to the disease is very widespread. No race, no age, no sex, enjoys immunity. It is a noticeable fact that those who have had the disease once are all the more apt to have it again. Former patients, although they feel perfectly well in a non-malarious region, are very liable to fresh attacks, or at least much discomfort, as soon as they re-enter an infected district. The time of incubation does not seem to be constant. It is put at from six to twenty days, but may be shorter. We shall consider below chiefly the common forms of intermittent, such as appear in Germany, contenting ourselves with a very brief description of the severer forms.

VARIETIES OF MALARIAL DISEASE

1. **Intermittent Fever.**—This is the simplest form, and has for its especial characteristic the relative brevity of the febrile attacks, which almost always exhibit a remarkably uniform type. A febrile attack of this kind is frequently the very first symptom of the disease. In other cases the paroxysm of fever is preceded by a prodromal stage lasting several days, during which the patient feels languid, has no real appetite, complains of headache and pain in the back of the neck and in the limbs, and often even thus early presents a slightly yellowish complexion and an enlarged spleen.

In the typical attack of intermittent fever there are three stages. The attack begins with a chill. There is pronounced malaise, attended by intense chilliness and more or less shivering. The skin is cool and pale, the face may be somewhat livid. The temperature of the interior of the body is elevated, and rapidly rises higher. In by far the greater number of cases the attack occurs in the morning, or at least before noon, and but seldom later in the day. This cold stage varies greatly in length, usually lasting an hour or two.

The chilliness is followed by the hot stage. The skin grows burning hot, the face flushes, the pulse, which was before small, becomes full, and the action of the heart is excited. At first the temperature continues to increase, and reaches in this stage its maximum for the attack. It is exceptional for it to remain under 104° (40° C.), and by no means rare for it to touch 106°, or even 107° (41°–41.5° C.). This stage almost always lasts longer than the preceding, generally about three to five hours. The temperature may begin to fall as early as the latter part of the hot stage, but may persist till the beginning of the third stage.

In this sweating stage the skin grows moist, and there is soon a profuse general perspiration. The patient begins to feel much better. In a few hours the temperature usually becomes normal, and, after lasting in all about eight to twelve hours, the attack is over. It may be shorter or rarely longer. Usually, however, the temperature keeps on sinking slowly, so as to be still subnormal even on the next morning, perhaps not above 97° (36° C.).

There are certain peculiarities in the temperature-curve which we have ourselves observed. The elevation of temperature is almost invariably more rapid than its decline. The rise is most abrupt during the first hours of the cold stage, and slower during the first portion of the hot stage. The ascent is but very seldom interrupted. During the hot stage, when the fever is highest, in the neighborhood of 106° F. (41° C.), there are not infrequently two little summits to the fever-curve, if the temperature be taken at short intervals. But the temperature may for hours remain the same. The temperature generally begins to fall some little time before the perspiration is evident. The decline is slow. It may be perfectly continuous, or it may be interrupted by fresh elevations, which are sometimes slight and sometimes considerable. In many cases the descent is by steps, the temperature remaining the same for half an hour or an hour, and then abruptly falling a couple of degrees and remaining for a time at this new level.

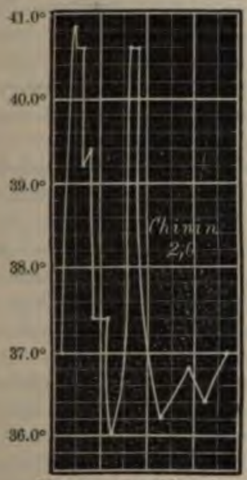

FIG. 11.—Quotidian intermittent fever.

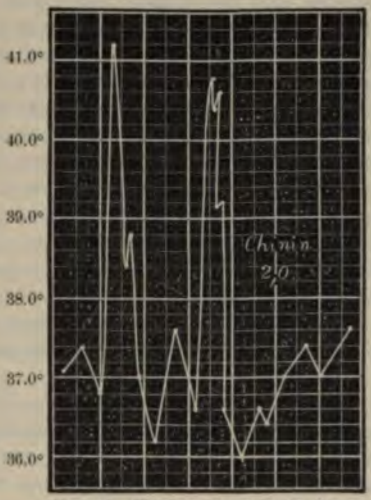

FIG. 12.—Tertian intermittent fever.

[Chinin 2.0 = Quinine 30 grains.]

The chief characteristic is not, however, the nature of the single attacks, but the peculiar manner of their repetition. If the case is not under treatment, the single attacks keep recurring for a time, either daily, as in the quotidian variety, or every second day. This latter type of tertian intermittent fever (cf. Figs. 11 and 12) is probably the most frequent. There may exceptionally be still longer afebrile intervals. Thus we have *quartans*, *quintans*, etc. If there are two attacks in one day, a rare event among us, we have a double quotidian. If there is a violent attack every second day, and on the intervening days there are milder attacks, it is a case of double tertian. Very often the attacks do not recur at just the same time of day, but a few hours earlier each time. Less frequently they are later. This peculiarity is expressed by the term "anticipating" or "retarding," as the case may be—e. g., a retarding tertian ague. In cases of long standing, the paroxysms may finally lose all regularity, so that the fever is described as "erratic."

Next to the febrile attacks, the swelling of the spleen is the most constant and important symptom. It is usually considerable and capable of demonstration by percussion and palpation. At first the tumor increases with every fresh attack, and diminishes but little during the intervals. After the patient is freed from his attacks of fever the spleen may continue enlarged for some time. It is tender on pressure. [Certainly in many cases tenderness is absent.—V.] The liver may likewise be swollen, but this is less constant and also less important.

Certain changes in the skin are very characteristic, chief among which is a peculiar yellowish-brown discoloration. This is due to an abnormal deposition of pigment in the skin. Herpes on the lips or nose is seen very frequently during the attacks. We have seen one case of herpes on the cornea. Mention has also been made of urticaria, purpura, and other eruptions.

Other internal organs than those already spoken of are rarely much disturbed. One symptom should be mentioned, which we have ourselves seen several times, viz., a quite marked acute dilatation of the heart during the attack. There were no bad results, and the normal condition was soon re-established. We may hear during the attack functional cardiac murmurs of a blowing character. Thoracic examination, particularly if made during the attack, may afford the signs of a dry bronchitis. Sometimes there is considerable diarrhœa, or other evidence of intestinal derangement. Catarrhal jaundice is confined to the severer cases. Sometimes the urine has a moderate amount of albumen. Genuine nephritis is met with only in the graver varieties of the disease. The increased excretion of urea on the days of the fever results, as in any fever, from the increased destruction of albumen. Severe pain in the cervical and upper dorsal vertebræ is regarded as characteristic of intermittent.

Besides the typical attacks, rudimentary and modified ones are not rare, in which the separate stages are ill defined, or in part wanting. We are most apt to see this in cases which have been already treated with quinine. Children do not have a true rigor. They merely become pale or livid. They may present marked nervous symptoms.

2. **Pernicious Intermittent Fever.**—This dangerous form occurs only in the true malarial districts, and is often preceded by a few attacks of a milder character. Then appear, in addition to the more or less perfectly marked stages of the febrile attack, other graver symptoms which not infrequently end in death. Severe nervous symptoms are most frequent. There may be unconsciousness, coma, delirium, or epileptiform or tetanic convulsions. None of these symptoms persist longer, as a rule, than does the common sort of an attack, and in a favorable case vanish completely when the sweating, which is usually profuse, begins. The great danger comes from the recurrence of the attacks. A second form of pernicious intermittent fever causes violent gastro-intestinal symptoms, which may almost exactly imitate the algid stage of cholera, with vomiting, diarrhœa, and collapse; or there may be severe cardialgia, dysentery, and the like. In the so-called pernicious intermittent with jaundice, intense jaundice appears during the attack, with vomiting and diarrhœa, and sometimes the gravest nervous symptoms. There are certain very peculiar forms, in which local disorders, such as pleurisy or pneumonia, can be demonstrated during every attack, but vanish wholly or in part when the temperature declines, only to appear again during the next attack.

[The pernicious form occurs in isolated cases wherever the ordinary variety of the disease prevails, but is much more common in the Southern and Western States, and there varies in frequency in different years. Periodicity in the attack is not always observed. The pernicious character is not always manifested in the first attack, one or more mild paroxysms being often precedent. In this country the algid form of pernicious periodic fever is often called "congestive chills," and this form is more common than the comatose or another form mentioned below by the author—the hæmorrhagic. In the latter the blood escapes from the kidneys, and less constantly from the mucous membrane. During the late civil war the mortality among the white soldiers of the United States army from pernicious malarial fever was 23.91 per cent.]

3. **Remittent and Continuous Forms of Malarial Fever.**—These are generally severe, and are seen, like the preceding, only in the worst haunts of malaria. The proof that they have the same ætiology as intermittent fever lies in the fact that they are sometimes developed out of the milder forms; but it is to be noticed that many types of disease which physicians in the tropics describe as malarial affections have not yet been proved to our satisfaction to have an actual identity of origin with the common intermittent fever. A positive diagnosis can be made only by examining the blood for the plasmodia. The symptoms of this variety are likewise those of a severe constitutional infection. Gastro-intestinal symptoms may predominate; or there may be such grave nervous symptoms as coma, delirium, and convulsions; or there may be jaundice, hæmaturia, and even a general hæmorrhagic diathesis; or various local disorders may exist, such as pneumonia, nephritis, and hepatic and splenic abscesses. The fever is high, but without any sort of regular intermissions, maintaining for one or two weeks a remittent or a tolerably continuous type. Milder forms may end in recovery after eight to fourteen days, but often death ensues at this time, or even earlier.

[The remittent form apparently shows a greater intensity of the poison or a greater susceptibility of the individual. In the United States army, from 1861 to 1866, its mortality was twelve times as great as that from the intermittent form.]

4. **Chronic Malarial Cachexia.**—This occurs in the true malarial regions, and affects not only persons who have had frequent attacks of pronounced intermittent or remittent fever, but also those who have never had acute attacks. The condition is chronic. It may exhibit a genuine intermittent character. The patient usually has a decidedly yellowish, malarial complexion, and almost always the spleen is evidently enlarged. There are no regular febrile attacks, but merely symptoms of general debility, anorexia, tendency to diarrhœa, or, more rarely, constipation, vertigo, wakefulness, frequent perspiration, pain in the muscles and joints, dyspnœa, and palpitation. There may be such nervous symptoms as trembling, paralysis, and mental disturbance [or multiple neuritis, *q. v.*]; or we may see intestinal symptoms and jaundice. Dropsy occurs; also epistaxis, cutaneous ecchymoses, and other signs of a scorbutic condition. The spleen and liver gradually become greatly hypertrophied and melanotic. At the same time there may be an irregular fever, approaching either an intermittent or a remittent in type. Finally, secondary diseases are possible—e. g., tuberculosis, amyloid, or dysentery —and these may prove the immediate cause of death. The milder forms may be cured, but seldom unless the patient abandons forever the malarial district.

5. **Masked Intermittent.**—This is the designation of cases in which, although there is no fever, certain other disturbances arise in regular intermittent attacks. Chief among these is neuralgia. Its favorite seat is the supra-orbital branch of the trigeminus. It may occur in the other branches of the same nerve, or in the sciatic, the anterior crural, the nerves of the brachial plexus, and elsewhere. Cardialgia and enteralgia may occur in the same way. These attacks last from thirty minutes to several hours, and are frequently associated with all sorts of constitutional symptoms, but, as we have said, are afebrile. There may be a splenic tumor, which aids diagnosis; but this sign may be wanting.

Numerous other intermittent disorders besides neuralgia have been described as masked intermittent. The list includes anæsthesia, convulsions, and paralysis; also intermittent hæmorrhage, œdema, cutaneous affections, and intestinal symptoms. We must add, however, that those who have described diseases of this sort, some of which seem strange enough, have not always been as critical as they ought, and that the connection between many cases of "masked intermittent" and true malaria is more than doubtful.

[6. **Typho-Malarial Fever.**—This is a term which was formerly in use, but it is not a distinct disease. It expresses a combination in the same individual at the same time of the effects of the special poison causative of each affection. Typhoid being a continued fever, its complication with malaria results in a pyrexia of a remittent type. The combination of the two occurs in malarial regions, especially in the Southern States, and may be seen in non-malarial regions in the persons of those in whom malaria, contracted elsewhere, is in a more or less active state.

The characteristic symptoms of the two diseases are intermingled, those of typhoid, the graver disease, usually predominating. The history of the case and careful observation of the symptoms will generally clear up any doubts felt as to the diagnosis in the early stages. It would naturally be supposed that the combined affections must produce an illness more severe in character and more unfavorable as regards prognosis than belongs to simple typhoid. Such does not, however, seem to be the case. Woodward's statistics show that the mortality of uncomplicated typhoid was far greater among the white and colored troops alike during our late civil war than was the mortality of typho-malarial fever.

The treatment as regards the typhoid fever differs in no way from that suitable for cases of the ordinary affection; the periodic element demands the management appropriate to simple intermittent or remittent fever.]

Diagnosis.—It is often very difficult to diagnosticate a case of intermittent fever at the first visit, particularly in a place where malarial poisoning is infrequent. The history of the case is by no means always enough to put one on the right track; and a single examination of the patient may prove equally negative in its practical results, whether it is made during the febrile stage or in the interval. Continued observation, however, will almost always disclose the regularity of the febrile attacks, the splenic tumor, the characteristic complexion, and the herpes; and our diagnosis becomes evident. Still it is not very exceptional for an intermitting fever to be taken at first for an intermittent malarial one, while eventually some quite different disease is found to produce the symptom. Pyæmia may give rise to mistakes of this kind; also purulent phlebitis, acute ulcerative endocarditis, and even tuberculosis. We should be very cautious in making a hasty diagnosis of "irregular intermittent fever." Our own experience has taught us that almost invariably the case turns out to be something else. Where there is doubt we may, in addition to a careful consideration of all the symptoms and a thorough physical examination, be aided by the therapeutic action of quinine (*vide infra*). If a high fever of intermitting type is affected by large doses of quinine but temporarily if at all, then a diagnosis of malarial intermittent fever is rendered doubtful. An absolutely certain diagnosis can be made only by finding the plasmodia in the blood under the microscope. We have already said that the greatest skepticism is requisite in the diagnosis of "masked intermittent."

Treatment.—Malarial poisoning is one of the few diseases upon which we can make a direct attack with assured success. In quinine we possess a remedy which probably acts upon the very cause of the disease, and its therapeutic efficacy is undisputed. Quinine is therefore the sovereign remedy in all forms of malarial poisoning, and is often the only drug employed. In the mild cases, which are the only kind that occur in Germany, we do not usually give the remedy upon the instant that the patient comes under treatment. It is best to wait for one or two attacks, partly to make sure of our diagnosis, and partly to learn what the type of the fever is, whether quotidian, tertian, anticipating, or recurring at the same hour; and in most cases this delay works no harm to the patient. During the attack itself there is seldom any use in special treatment. Of course, the patient must stay in bed and be kept warm during the cold stage, and have lighter coverings during the hot stage. During the afebrile interval the patient may be up if he feels strong enough and is careful. Quinine (hydrochlorate or sulphate) is given about five or six hours before the next attack is due. It is best to administer one large dose of twenty to thirty grains (1.5–2 grm.), either in solution or in capsules of seven grains (0.50 grm.) each. If the quinine be given in powder or capsules, it is a good way to follow it with a few drops of muriatic acid, to promote its solution in the stomach. Often one large dose prevents the next attack. In other cases it does occur, but with less subjective disturbance, no chill, and more moderate fever. We must then give another large dose before the second attack is expected. If the attack does not take place, then we may give for several days quinine to the amount of eight grains (0.5 grm.) *per diem*. After all, relapses are possible, even at the end of several weeks; but they yield readily to quinine.

In the treatment of pernicious intermittent fever, of the masked forms, of the remittent and continued fevers, and of malarial cachexia, the chief remedy is likewise quinine, given in sufficient doses. Baccelli has shown that sometimes in pernicious fever the direct injection of quinine into a vein may save life. In all cases of considerable duration it is also of the greatest importance to remove the patient from the malarial region, if it can possibly be done. This often proves to be the only way to avoid relapses and attain a perfect cure.

In cases of longer standing, quinine sometimes loses its power. Then we resort to arsenic. It is frequently employed in malarial cachexia and in intermittent neuralgia, either alone or combined with iron. We give gtt. v to viij of Fowler's solution two or three times a day in water. It is still better to give pills of arsenious acid containing gr. $\frac{1}{30}-\frac{1}{20}$ (grm. 0.002–0.003) and gradually increasing to a daily dose of gr. $\frac{1}{6}-\frac{1}{5}$ (grm. 0.010–0.012). It may be added that arsenic is also said to have a prophylactic virtue: a long-continued use of it is stated to render a person proof against malarial poisoning. We will not speak of the numerous other remedies recommended, such as eucalyptus, piperine, pilocarpine, berberine, and many others, for they can be entirely dispensed with.

The management of the severe varieties of malarial disease involves symptomatic treatment as well as the administration of quinine. We can not enter into the particulars. In combating the grave, nervous, intestinal, pulmonary, and renal symptoms, the dropsy and the anæmia, the physician must conform to the general rules of treatment.

[There is nothing to be gained by allowing a patient to have an unnecessary chill. If there is a reasonable probability that his paroxysms are due to malaria, a prompt effort should be made to cut them short. Four hours is the shortest time that it is safe to allow for quinine by the stomach with probability that the expected chill will be prevented. The drug acts much more promptly when given hypodermically. The hydrobromate is preferred by some to the sulphate for subcutaneous use on account of the necessity of using acid to dissolve the latter, and the consequent risk of abscess. Such a risk should have no weight if the physician has any suspicion that he has to deal with the pernicious form of the disease. If the stomach is irritable, the remedy can be given by enema. Quinine can also be given by suppository, though it may thus produce some irritability of the rectum; but the impossibility of disguising the bitter taste of the remedy or of making children swallow capsules renders this a valuable means of treatment sometimes in infants and young children.

Warburg's tincture is an antiperiodic which does good service in cases which do not yield to the ordinary methods of treatment.

The hypodermic injection of pilocarpine is reported to abort an impending chill.

Some prefer divided and smaller to the single and large dose of quinine or one of its substitutes, a difference of view which is of minor importance.

In the remittent forms boldness in the use of quinine is required. Cinchonism should be induced as promptly as may be, and maintained to a mild degree for several days; the quantity of the drug can then be gradually diminished.

The treatment of the pernicious forms of periodic fever presents itself under three main heads:

1. The prevention of pernicious paroxysms.
2. The treatment of the paroxysm when present.
3. The prevention of a recurrence.

1. We have seen that very frequently the pernicious character is manifested after the occurrence of one or more mild attacks; consequently, in localities and seasons marked by the occurrence of grave cases it is an imperative duty to treat every mild case promptly and energetically, a course which unquestionably saves many a life.

2. The management of the paroxysm differs according to the form which it assumes; in other words, is largely symptomatic. Bemiss (Pepper's "System of Medicine") says: "The cure of a congestive chill is one of the most difficult problems the physician can possibly encounter." Heat externally, opium and chloroform by the mouth, and morphine and atropine subcutaneously, nutrition by the stomach or rectum, according to circumstances, and alcoholics if the action of the heart is feeble, are the measures of widest application.

Whatever the type of the attack, a weak heart calls for alcoholic stimulation. Cinchonism is always to be induced as rapidly as possible.

In the comatose form it is to be remembered that the cerebral and other nervous symptoms are not due to congestion, but probably to a combination of the malarial and secondary blood-poisons. To quote Bemiss again: "Efforts to nourish the patient must never be relaxed. One must see many of these cases before he can realize how often they recover from conditions apparently hopeless when promptly treated and properly nourished."

The hæmorrhagic form, like the others, demands cinchonism and careful nutrition, but also hæmostatics. Purgative doses of calomel are indicated in some cases of each form, but should not be given in a routine manner.

3. Prompt cinchonism is the chief means of attaining the third aim of treatment. Removal to a healthy locality should be secured if possible, and the general condition of the patient requires careful attention.

It remains to add that those going to a malarial region can often avoid contracting the disease by taking advice of a local physician as to hygienic precautions, and by moderate divided doses of quinine.]

CHAPTER XV

DENGUE

[This affection has never appeared in Germany, and hence, doubtless, was omitted by the author. The name "dengue" is supposed to be a Spanish corruption of dandy, the term dandy fever having been applied to the disease by the West India negroes on account of the stiff carriage of those affected by it. Another name is "break-bone fever."

The disease generally appears in epidemics, and is almost exclusively confined to tropical and semi-tropical countries. In 1870 an epidemic supposed to be dengue prevailed in Philadelphia, and outbreaks have occurred repeatedly in the Southern States in the last hundred years. In 1880 Charleston, Savannah, New Orleans, and other Southern cities were visited by it. There are those who maintain that dengue and epidemic influenza are identical, a view which the facts do not seem to the editor to bear out.

Ætiology.—As to the causation but little is known. Those who have had opportunities of studying the disease consider it both contagious and infectious, and the inference that it depends on a specific germ is readily suggested. It seems to prefer low lands along the sea-shore, and to be influenced by meteorological conditions in that it generally prevails during the summer and disappears as cold weather comes on. Neither age, sex, nor condition affords any protection from the disease; it was thought by Dickson that one attack generally confers immunity for life.

Pathology.—The disease is so rarely fatal that few opportunities have been afforded for its post-mortem study. So far as is known, it has no peculiar lesions. The prominence and the character of the muscle and joint pains have led some observers to think the affection related in some way to rheumatism.

Symptoms and Course.—The onset is usually sudden, but a pronounced chill is said never to occur. Prodromata similar to those of other infectious diseases are sometimes observed, but the first symptom is very often pain and stiffness in the muscles and joints, with frequent swelling of the latter. The large and small joints are equally affected, and the pain is increased by motion. With the pain there is a rise in temperature and in the frequency of the pulse. The pain is apt to increase during the first two or three days and disappear on the fifth, but irregular remissions are liable to occur. As the thermometer falls the pain and other symptoms diminish, but reappear in full force with any subsequent rise. During the later days of the disease a skin eruption appears on the upper part of the body, and in severe cases becomes general in about two days. This eruption is very variable in character; it may appear simply as an erythema, or simulate the eruptions of scarlet fever, rubeola, lichen, or urticaria; it is apt to be associated with well-marked heat and itching of the skin. In mild cases the eruption is evanescent or absent. Swelling of the lymphatic glands is not uncommon; in severe cases the mucous membrane of the mouth, throat, and nose is reddened, and hæmorrhage from the outlets of the body has been observed. Pregnant women are apt to miscarry. Delirium is rare in adults, but common in children; the face is generally flushed, and the eyes are injected; the tongue becomes increasingly coated as the disease progresses, the appetite is lost, nausea is not uncommon, vomiting is rare. The bowels and the kidneys present no constant or distinctive symptoms.

In mild cases recovery is sometimes rapid; sometimes, and especially after severe cases, convalescence is very tedious, the muscular and articular pain and stiffness passing off gradually, and the glandular swelling lasting for weeks. Copious skin eruptions are followed by desquamation.

Diagnosis and Prognosis.—As to diagnosis there can seldom be any difficulty during the prevalence of an epidemic. The first cases are the only ones which are liable to be mistaken, and even their nature can not remain long in doubt. The prognosis is uniformly good.

Treatment.—We are acquainted with no agent capable of aborting or cutting short the disease; nor is there any known measure of prophylaxis except for an individual to keep away from those places in which the affection is known to prevail.

The treatment of the attack is simply symptomatic; notable pain calls for opium in some form. Quinine has not seemed to be of service. Debility following the attack demands suitable alimentation and tonics.]

CHAPTER XVI

YELLOW FEVER

[THIS disease is not a visitant of Germany, but its consideration can not be omitted from a text-book on the practice of medicine for use in America. In the following description the aim will be to bring out the more important features of the disease, while for fuller details the reader is referred to larger works and monographs.

Ætiology.—Yellow fever is an acute infectious disease, confined within certain geographical limits, and occurring chiefly in epidemics of greater or less extent. In certain localities, notably Havana and New Orleans, a season rarely passes without some sporadic cases. The influence of temperature is well established; the disease prevails, namely, during the summer or the warm season, and is abruptly arrested by one or two decided frosts; dampness is favorable to it. That it depends ultimately on a special cause and does not originate *de novo* are undisputed facts. [Sanarelli claims to have found the specific germ in a minute bacterium, occurring in pairs and with rounded ends, which he calls the *bacillus icteroides.* Other authorities do not as yet admit that this bacillus is certainly the cause of yellow fever.—V.] The poison appears to be more active at night than during the day, prefers low-lying districts, and in them hugs the ground to a certain extent. Bad sanitary conditions are most important accessory causes of the disease, furnishing the soil for the multiplication of the poison. There can be little doubt that, under the observance of strict personal and public cleanliness, yellow fever visitations might be made simply a matter of history. The transmission of the poison may take place through the atmospheric air, thus finding its way to the blood through the lungs; conclusive evidence is lacking that it gains entrance to the system through the alimentary canal. [Recent investigations seem to indicate that it may be transmitted by mosquitoes.—V.] While the air may be the medium of transmission, the distance to which the poison can be carried by the air alone is very short; it can, however, be transported to an indefinite distance by fomites, especially if inclosed in trunks, packing-cases, letters, and the like. Its vitality may thus be maintained for very long periods. It is a remarkable fact that in large cities the infection may be of great virulence, but confined to a limited district or districts, by carefully shunning which unprotected persons are comparatively safe. An infected area is apt to extend itself, but the progress is slow, and is interrupted by streams of water, high walls, or simply streets.

That the disease is not, strictly speaking, contagious is the nearly unanimous opinion of those competent to form one. In other words, the poison is not thrown off in a matured state from the body of an individual suffering from the disease; but, after being so thrown off, remains in the atmosphere or lodges on solid bodies, and then undergoes changes which render it active for evil. One attack of the disease renders the system of that person insusceptible forever after; the natives of a yellow-fever district are far less liable to contract the disease than are those who move into the district from elsewhere; until these have passed through an attack they can not consider themselves as acclimated. The negro race is susceptible to the disease, though in a less degree than the whites, and in the colored the affection is far less fatal. Neither age nor sex has any special bearing on susceptibility. That fear, anxiety, worry, or anything which tends to depress the nervous system increases the individual liability is highly probable. The stage of incubation is very variable, ranging from one day to three weeks or even more.

Pathological Anatomy.—The disease involves no constant and peculiar lesions. In rapidly fatal cases, congestion and often hæmorrhage are found, especially in the nervous system, liver, kidneys, and digestive tract. In fatal cases of longer duration more or less parenchymatous degeneration is found. A fatty degeneration of the liver is quite common, and imparts a yellow coloration to the organ, giving rise to the terms *café au lait*, or box-wood liver. Jaundice of the skin and tissues generally is also observable after death, and depends upon causes in no way connected with mechanical obstruction to the flow of bile into the intestine during life. Splenic enlargement is conspicuous by its absence.

Course and Symptoms.—The onset of the disease is generally sudden, but it may be preceded for a few days by malaise and other signs of general constitutional disturbance; the initial chill is seldom severe, reaction following soon and the thermometer rising to 102°–105°; hyperpyrexia is rare. The pulse-rate does not increase proportionately with the fever. The face becomes flushed and the eyes injected and watery; headache and pain in the back are early and usually very prominent symptoms. The bowels are confined; the tongue is apt to be clean if it was so before the disease came on; the stomach is usually somewhat irritable, and there may be vomiting; moderate epigastric tenderness is common; the mind remains clear, as a rule, but delirium is not very uncommon, and in children a convulsion may usher in the attack as in other acute infectious diseases; the condition of the urine is at first not remarkable, but albumen may soon appear. This hot or febrile stage may last from twelve hours to several days. The pulse generally declines in frequency before the disease has reached its maximum. As the fever disappears the other symptoms vanish also, and the second, or "stage of calm," begins. From this point recovery may be rapid and uninterrupted, the whole disease consisting of but a single febrile paroxysm of greater or less intensity and of short duration.

In grave cases, and gravity is often foreshadowed in the first stage by marked capillary congestion of the surface of the body irrespective of the intensity of the other symptoms, and after a stage of calm lasting for some hours, but rarely exceeding twenty-four, more distinctive symptoms appear. The pulse is very compressible, the surface of the body is cool, vomiting occurs or becomes more prominent, and hæmorrhage is now apt to take place. The escape of blood into the stomach, its retention and the changes which it there undergoes, and its susbsequent expulsion, explain the dreaded and characteristic symptom know as "black vomit." Tarry stools sometimes are observed. Hæmorrhage elsewhere is also common, occurring from the gums, the nose, eyes, uterus, kidneys, into the skin, etc. Albuminuria with casts is very common. Jaundice, sometimes of a lemon-yellow hue, comes on, and is rarely lacking in severe cases. From this symptom the name of the disease is derived.

In cases marked by more or less complete suppression of urine, coma and convulsions, probably largely uræmic, come on. Some severe cases are of the "walking" type, the patients going about while the malady is far advanced, or even up to the time of death. As a rule, however, muscular prostration is marked.

If the disease does not prove fatal in this third stage, convalescence comes on more or less gradually, and is followed by complete recovery; relapses, however, occasionally occur.

The duration of the affection is variable, but, on the whole, short, usually being less than a week.

Diagnosis.—In mild cases the symptoms are not distinctive, and the diagnosis at the commencement of an epidemic is not likely to be reached except by an experienced observer, and even by him more or less conjecturally. During an epidemic the severe lumbar pain, the headache, the suffusion of the eyes, and the moderate gastric irritability, are all-sufficient for diagnostic purposes. Severer cases are also marked by capillary congestion of the surface of the body, and the third stage with the black vomit, hæmorrhage, jaundice, slow pulse, scanty urine, and prostration is characteristic. Of course, all the above symptoms are not present in every case. The only disease which can well give rise to confusion is remittent fever with jaundice. This affection has a different temperature-curve, is not confined to the yellow-fever zone, is controlled by quinine, and is not accompanied by black vomit.

Prognosis.—This varies in any given locality with the character of the prevalent epidemic. The death-rate is sometimes very high, sometimes moderate; it is greater in hospital than in private practice.

In the first stage of the disease the chief element in the formation of the prognosis seems to be the presence of marked and general capillary congestion of the skin, a symptom which foretells a severe attack. The absence of this symptom is rather less important than its presence. Cases may turn out to be severe in which it is lacking. The frequent deceptiveness of the stage of calm is to be remembered.

Yellowness, black vomit, and suppression of urine are symptoms denoting the greatest gravity, but do not justify the complete abandonment of hope.

Treatment.—There are no means in our power of aborting the disease. Prevention is to be attained by cleanliness in its large sense, and by careful quarantine against things rather than persons. Individuals from infected localities may safely be admitted into non-infected localities, provided that they and their clothing and effects are thoroughly disinfected. Merchandise, the mails, and the like, must be excluded or disinfected. So also vessels and all other means of communication.

The earlier proper treatment can be instituted, the better. Absolute rest and good ventilation are the first requisites. Emetics and cathartics are not indicated by the disease itself; the condition of the stomach and bowels should be inquired into, and indigestible food or an accumulation of fæces should be removed if present. A hot mustard foot-bath early in the attack is useful. For the lumbar pains, opium or morphine are indicated. Sinapisms, or other similar external counter-irritants, with ice internally, and hydrocyanic acid or chloroform, are serviceable against gastric irritability. High fever is to be combated by cold spongings, the wet pack, and the cold bath. For hæmorrhage, styptic remedies may be used, though it is doubtful whether, when given internally, they are really of much benefit. Of course, no medication is to be resorted to which is likely to heighten a tendency to emesis.

[Much work has been done with antitoxines for yellow fever, but with no settled results, as yet. Sternberg's formula for internal use is said to be valuable for quieting the stomach and promoting diuresis. It is as follows: Sod. bicarb., gr. cl; hydrarg. chlorid. corros., gr. ⅓; aquæ puræ, O ij. M. S. Three tablespoonfuls ice-cold hourly.—V.]

Suppression of urine is to be met by dry cups to the loins, diuretic remedies internally if the condition of the stomach allows, or the hot-air bath. The results of pilocarpine are disappointing according to Bemiss, who states that he has seen good effects from large enemata of water, preferably cold, if there be notable fever in these cases. Prostration is an indication for the use of alcoholic stimulants, among which iced dry champagne ranks high. It will be seen that the treatment is entirely symptomatic. The disease is self-limited, has a short course, and the patient will recover if he can be kept alive until the poison is exhausted. During the attack and until convalescence is thoroughly established the management of the diet is all-important. Small quantities of the most easily assimilable food may be given at short intervals if they are tolerated by the stomach; if not, alimentation must be by the rectum, and the lower bowel in this disease is generally in a fair condition for this method of nutrition.

Courage and hopefulness on the part of the physician may do much good, and the services of a skillful and experienced nurse are of the utmost importance. I am told that in New Orleans, and perhaps elsewhere, it is customary, for those who are not protected and can afford this course, to secure in advance a nurse as soon as an epidemic breaks out. They then take to their beds at the first sign of illness.]

CHAPTER XVII

PLAGUE

(Bubonic Plague. Malignant Adenitis. Black Death)

[**Definition.**—Plague is an acute, febrile, infectious disease, occurring endemically and epidemically, somewhat contagious, and occasioned by the *bacillus pestis bubonicæ.*

Historical Remarks.—Although it is difficult to identify diseases from the descriptions of ancient authors, Hirsch believes that the first historical account of the plague relates to an epidemic affecting Libya, Egypt, and Syria, at the end of the third or the beginning of the second century before Christ. Pandemics occurred in the sixth century of our era; the fourteenth, when, under the name of the "Black Death," it is estimated to have destroyed twenty-five million of the inhabitants of Europe; and the seventeenth century. The great plague of London in 1665 destroyed seventy thousand out of a population of four hundred and sixty thousand.

From the end of the seventeenth century the disease gradually yielded to the march of sanitary improvement. Like leprosy, it is now unknown in countries where it formerly was a terrible scourge. It remains endemic in northern Africa, southwestern Arabia, India, southern China, and a large district in the neighborhood of the Caspian Sea (Payne). At the present writing the disease is extended widely over the habitable globe, but the immunity from its ravages which civilized races now enjoy well illustrates the prophylactic value of modern hygiene.

Plague became active in south China in 1894, afflicted Hong-Kong from 1895 to 1898, and has occurred there within a year. In three years, ending September, 1899, there were 319,040 cases in the Bombay presidency, and of these, 250,677 were fatal. It appeared in Egypt in 1899; also in Oporto, Algiers, and South Africa. In the year 1900 there were cases in the Chinese quarter of San Francisco, Honolulu, Manila, Australia, and Glasgow.

Ætiology.—The plague is due to a minute organism, known as the bacillus pestis bubonicæ. This organism is constantly found in those suffering from the disease and in their bodies after death, and a pure culture of it will cause in animals a disease unquestionably like that seen in men. In 1898, an assistant in a pathological laboratory in Vienna came down with the disease, while occupied with infected animals—there being no possibility of human contagion. The attack was fatal and entailed the fatal illness of the attending physician. There are three cases on record of successful inoculation, one by a physician, Whyte, upon himself in 1802, and fatal, and two upon condemned criminals, who took the disease but survived. These three cases, however, occurred at times when the disease was epidemic.

The mode of growth of the bacillus is characteristic. It stains readily with the ordinary aniline dyes—e. g., Loeffler's blue or weak carbol-fuchsine. It is decolorized by Gram's method. Its ends stain more deeply than the central portion, giving it somewhat the appearance of a diplococcus. It has no spores. Like other micro-organisms, its virulence varies greatly under varying circumstances of culture and transmission through animals. When an epidemic is imminent, the first cases are often mild, then the intensity of the poison increases rapidly, abating again toward the end of the visitation. Lower animals and even insects are liable to infection. The list includes rats, mice, dogs, and also flies, fleas, lice, mosquitoes, ants, and bugs. The external circumstances which most favor the spread of the disease are overcrowding, poverty, and filth. Great extremes of temperature are unfavorable to its development; a certain degree of moisture, on the other hand, fosters its growth. The geological character of a place, or the elevation above the sea, has little influence. The immense practical importance of proper social and hygienic conditions is illustrated by the percentage of deaths among the different races in Hong-Kong during the late epidemic: Chinese, 90.4; Hindoos, 77; Japanese, 60; Europeans, 18.2. These figures indicate very fairly the relative hygienic conditions of the classes enumerated. It is undoubtedly lack of a favorable soil that has freed Europe and the civilized parts of America from the disease.

The mode of infection varies. It is believed that most cases are due to the entrance of the germ through some trauma, or fissure of the skin—e. g., through the bite of insects or the scratches occasioned by the same. In support of this view, the fact has been emphasized that the characteristic buboes are mainly in the groin, and among barefooted people; but against it is the experience of Europeans, who, with their lower extremities protected, also have a preponderance of inguinal adenitis. In cases with pulmonary localization the sputum is dangerous, either if it is projected upon the conjunctivæ, or presumably if it is inhaled when suspended in the air mixed with minute particles of moisture. Localities and houses seem to hold and transmit the infection, persons in the lowest part of the house, nearest the soil, being most apt to suffer. It seems very difficult to disinfect a dwelling by ordinary methods, the poison sometimes surviving a deluge of liquid disinfectants. On the other hand, soldiers wisely directed can engage in the purification of a dangerous locality with impunity.

Certain articles are capable of transmitting the infection. The Sanitary Conference at Venice, in 1898, enumerated in this class: used body linen, clothing, bedding, and other personal effects, rags, bagging, carpets, old embroidery, green hides and skins, raw silk and wool, animal and human hair.

Animals fed upon the infected viscera acquire the disease, but it has not been proved that man becomes infected through the ingesta, although the bacilli are found in the dejections. At any rate, during an epidemic it would be wise to have all drinking water boiled, and to eat no raw fruit or other uncooked food.

Course and Symptoms.—The period of incubation is usually three to five days. It may be as short as thirty-six hours, or, exceptionally, extend to fourteen days. The Conference at Venice settled upon ten days, for quarantine purposes. During incubation the symptoms are uncharacteristic, if any at all appear. The onset of the disease is usually abrupt, with rigors or chilliness, intense headache, and general pains. The temperature may be 104°–105° F. (40°–41° C.) on the first day, but is more apt to reach such a height by the third day. The pulse is somewhat characteristic, being rapid, out of proportion to the general condition, ranging from 100 to 120 or even 140. It soon becomes feeble, dicrotic, hard to count, and, toward death, intermittent. The heart is weak, and its first sound gradually becomes indistinct, and its right side dilated. The condition of the tongue is regarded as important for diagnosis. It has a white, moist coat at first, later becoming dry and brownish. Exceptionally, it may be brown from the start. Quite constantly the edges and tip and often the median furrow are clean.

The throat and tonsils are red and congested. Sometimes the tonsils seem to be the gate of entrance for the poison, and in such cases the cervical glands are apt to be much enlarged. The eyes are congested and sunken, with a dark ring around them.

The general appearance of the patient gives the impression of sepsis. The countenance may have a "fatuous, listless, unconcerned" expression, or may be "pain-drawn and haggard." In children the onset in this, as in other infectious diseases, may be marked by convulsions. Delirium is common; this may be quiet or, exceptionally, maniacal. The gait is early affected, becoming feeble and staggering, and suggesting alcoholic intoxication. The speech is thick and husky, due to lingual paresis, and is quite characteristic.

Adenitis is present in sixty-five to one hundred per cent. of the cases. It is this circumstance which has given rise to the name bubonic plague. The change in the glands usually becomes apparent by the third day, but it may precede the constitutional symptoms or may be delayed to the sixth day. At first an enlarged gland is felt, varying in size from a walnut to a small orange. This is tender and often extremely painful; soon there is a collateral œdema, making a large swelling in the neighborhood. The favorite location of the adenitis is the femoral or inguinal region. It is usually unilateral. Other points of development are the axilla, neck, and less often the back of the knee and the elbow. The parotid gland also may be affected. Sometimes more than one group of glands are affected at the same time, and *post mortem* there may be found enlargement of every gland in the body. The buboes may remain hard until death; in other cases they suppurate, which is regarded as favorable. On the other hand, if they soften or disappear during the height of an attack, death may be expected. By the eighth or tenth day the skin over a bubo may slough; and if a patient survives, convalescence may be prolonged for weeks by the adenitis with its complications. There are apt to be subcutaneous hæmorrhages in the skin over buboes, as well as petechiæ in various other parts of the surface, or more extensive hæmorrhagic stains. Exceptionally, there may be a rash resembling that seen in severe exanthematic typhus. Hæmorrhage is a sign of severity; it may occur from the nose, mouth, stomach, lungs, intestine, kidneys, or womb. A pregnant woman is almost certain to miscarry.

Boils and abscesses may occur in the disease. Certain lesions called carbuncles sometimes appear. They are really areas of moist gangrene, extending horizontally instead of deeply into the tissues, and they may attain a diameter of several inches.

The urine is scanty, and is often retained or actually suppressed. Apart from renal hæmorrhage it shows merely acute congestion, as in other infectious diseases. The liver is enlarged and tender; the spleen is always very much enlarged, up to three or four times its usual size. There may be persistent vomiting at the onset of the disease. Constipation is present, as a rule, but there may be severe diarrhœa. In ordinary cases respiration is somewhat hurried.

Fluid withdrawn from a bubo in its early stages shows the characteristic bacillus. When suppuration has occurred the organism may not be found, having probably been destroyed by the agents of suppuration. The circulating blood contains the organisms, but they are isolated from it with some difficulty. After death the viscera, particularly the spleen, are found to contain great numbers of the germs.

In the pneumonic cases of plague the disease is septicæmic in its characteristics and fatality; hence, cough is a very important symptom. The signs in the chest are remarkably slight. There may be a small lobule of consolidation found, particularly in the lower part of the back or at the apex. The signs vary from one day to another. The expectoration may be scanty or absent, and if present may seem merely bronchitic. It is more apt to be sero-mucous than muco-purulent. Often the sputum is almost a pure culture of the specific organisms. Pneumonic cases are almost invariably fatal before the end of the third day. As a rule, they are not associated with enlargement of the glands in any part of the body.

A third variety of plague is the septicæmic, in which death occurs by the second or third day. There is delirium, merging into coma; all the glands are enlarged but not prominent; hæmorrhage is frequent and apyrexia not uncommon.

Pestis Minor.—At the beginning of an epidemic there may be cases of very little severity, but with bubonic manifestations of a mild type. This form almost always runs a favorable course, and lasts about two weeks. Its importance in the way of prophylaxis and public hygiene may be very great.

Diagnosis.—The general picture of the disease is that of a profound sepsis, *plus* the peculiar adenitis. Sometimes the changes in the glands are attended with no subjective symptoms, and the physician is in danger of regarding the case as, for instance, typhoid or typhus fever. Typhoid fever has a more gradual onset, a relatively slower pulse, rose-spots, and eventually a Widal reaction. Typhus fever runs fourteen days instead of a week, is more regularly associated with an eruption, and has different ætiological relations. The enlarged glands might be mistaken for tuberculosis or syphilis, and the symptomatic parotitis has been regarded as mumps, but due care would make mistakes of this sort infrequent. Diphtheria has been suspected in a case attended by such changes in the cervical glands that the mouth could not readily be inspected. Cases with inguinal adenitis have been regarded as gonorrhœa, in both sexes (Hossack and Colvin).

Malaria has been confounded with plague, but the temperature curve, the efficacy of quinine, and the discovery of plasmodia in the blood should enable one to recognize it. So much œdema is sometimes associated with the buboes as to simulate erysipelas. Cases of severe influenza and of pneumonic plague greatly resemble each other in everything, except prognosis and the micro-organisms of the sputum. Other diseases which have been simulated by plague are puerperal fever, septico-pyæmia, cerebro-spinal meningitis, cerebral embolism, epilepsy, uræmic coma, diarrhœa, debility, marasmus, and alcoholic poisoning (Hossack).

Prognosis.—When at its height, the disease is fatal in perhaps ninety per cent. of the cases. The average fatality of epidemics has been stated as from thirty-five to fifty per cent. Death may occur as early as the second or third day; if life is preserved beyond the sixth there is hope of recovery. Petechiæ, hæmorrhages, and pulmonary signs are of fatal omen.

Treatment.—With the present ease of human intercommunication we can not hope, with the strictest quarantine, to exclude every infectious germ, but we can render the exposed locality unsuitable for its growth. This is well illustrated by the recent experience of Glasgow, where the disease has now ceased after a minimum loss of life (27 cases and 8 deaths). Major Edie, United States health-officer at Manila, says:* "The opinion I have formed of plague from the experience of the past six months is that little difficulty will be found in controlling it in any intelligent community with modern sanitary conditions." The physicians, nurses, and visitors at well-regulated plague hospitals do not catch the disease.

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* Surgeon-General's report, 1900.

Therapeutic measures have been, until recently, mainly symptomatic and apparently futile. Two forms of specific treatment have been introduced. Yersin has prepared an antitoxic serum from the horse, precisely analogous to the antidiphtheritic serum obtained from the same animal. This preparation keeps well, is harmless, painless, and confers immediate immunity to the disease, lasting about fourteen days. It is to a certain extent curative in cases already diseased, the more so the earlier it is given. Haffkine has prepared a vaccine fluid by subjecting a culture of the bacilli, in broth, to a temperature of 70° C. for one hour. This sterilized fluid contains the dead germs, and also the toxines developed incidentally to their growth. It confers immunity to infection, beginning the seventh day after inoculation. The injection is best repeated two or three times, at intervals of eight days. The local reaction from this is considerable, but does not last more than two or three days, and the person treated remains immune for months. Haffkine's fluid is said to aggravate an impending attack, if it is given during the period of incubation, so that a person who might otherwise have survived the disease may lose his life. The most recent writers on the treatment of the plague are not in full accord as to the value of these injections just described. Major Edie (already quoted) says: "A thorough test has not been given to either the Haffkine material or the Yersin serum, but we have not been favorably impressed with either." The reports from India are more encouraging.—V.]

CHAPTER XVIII

EPIDEMIC CEREBRO-SPINAL MENINGITIS

(Spotted Fever. Cerebro-spinal Fever)

Ætiology.—The epidemic form of cerebro-spinal meningitis has been known only since the beginning of the nineteenth century. The first epidemics were observed in southern France and in Geneva. Smaller ones occurred in Germany in 1822 and 1853; but it was not till 1863 that the disease became at all frequent among us. Since that date there have been more or less extensive epidemics almost every year. The southern and central portions of Germany are particularly liable to them. Sporadic cases may occur at any time.

Most of the epidemics appear in the winter and spring. We do not know any particular factors which promote the disease. It often seems to be decidedly endemic. Barracks, work-houses, and the like have been marked by tolerably extensive epidemics. Whether the disease can be carried by patients to places previously free from it is still uncertain. It is not directly contagious. Children and young adults are the most frequent victims; but now and then elderly persons are attacked. Sex can not be shown to have much influence.

That the disease is infectious is clearly shown not only by its epidemic and endemic character, but by its whole course. The peculiar pathogenic organisms and the manner of infection have not yet, however, been positively ascertained. Diplococci are usually found in the purulent exudation in the meninges, which are so much like Fränkel's pneumonia diplococci that many authors have considered the germs of croupous pneumonia and of epidemic meningitis as identical. It is confirmatory of this that many clinical facts suggest a relationship between the two diseases: the appearance of herpes, the comparative frequency of secondary purulent meningitis in pneumonia, the occasional co-existence of epidemics of pneumonia and meningitis, etc. On the other hand, we must not overlook the clinical differences, especially the atypical course of epidemic meningitis, which almost never ends by crisis. The chief point is that, assuming that pneumonia and epidemic meningitis are due to identical germs, we can not explain why the same germ at certain times finds its way to the lungs, and at other times and in other localities goes to the cerebral meninges. In the separate epidemic occurrence of cerebro-spinal meningitis we find a strong argument for the specific individuality of the disease. Weichselbaum and others have recently found in epidemic meningitis another peculiar diplococcus which is called the *diplococcus intracellularis meningitidis.* This diplococcus, arranged in pairs or tetrads, is found chiefly within the pus cells, and externally closely resembles the gonococcus. Further research is necessary to determine the significance and constancy of its presence. [Later researches seem to show that this coccus is the actual cause of the disease.—K.]

With regard to the gate of entrance of infection in primary epidemic meningitis, perhaps the specific poison may enter through the nostrils and the cribriform plate to the otherwise so well protected envelopes of the central nervous system.

[Sanitary conditions seem to play a less important *rôle* in this than in many other infectious diseases. During the epidemic which visited New England in 1873 the writer was interne at the Massachusetts General Hospital, and there saw a number of cases. The disease was also prevalent among horses at the same time, and it is curious to note that a like association of the affection in men and animals was observed in Vermont in 1811, and in New York city in 1871. During the year 1873, 216 deaths were returned as due to this malady in the city of Boston.]

Pathological Anatomy.—The autopsy discloses an acute purulent cerebrospinal leptomeningitis. It is only in rapidly fatal cases that slight and incipient lesions have been met with. As a rule, the extent and intensity of the objective lesions correspond to the severity of the symptoms. In the brain the purulent inflammation attacks the convexity as well as the base. It is usually most marked along the larger blood-vessels and in the fissures of the cortex. Of the spinal cord the posterior surface suffers most. Frequently the lumbar portion is more affected than the parts above. It is, however, exceptional for the disease to be limited to the meninges; it is prone to extend into the underlying parenchyma. The microscope reveals clumps of pus-corpuscles about the blood-vessels, where they penetrate into the tissues, and not infrequently there are numerous centers of genuine encephalitis. These latter may be visible to the naked eye. Exceptionally there may even be cerebral abscesses of considerable size. The vessels are distended with blood, clear into the central ganglia, and ecchymoses are frequent. The cerebral ventricles are usually enlarged, and filled with a cloudy serum, or even with pus. It is plain that these lesions of the cerebro-spinal parenchyma greatly modify the clinical picture, and that they must frequently have more to do with the severity of the symptoms than has the leptomeningitis itself.

Clinical History.—Prodromata are relatively rare, and if present they are not severe, being confined to general malaise, with slight headache, and pain in the limbs. Usually the disease begins rather suddenly; there is intense headache, often felt mainly in the occiput, pain and stiffness in the back of the neck, and great general discomfort. It is not rare for vomiting to occur at first. Very often there are among the early symptoms such important mental disturbances as stupor or delirium. There is usually fever from the first. An initial rigor may occur, but it is not the rule.

The intensity of these first symptoms is not always the same. Subsequently to them the course of the disease may vary greatly. First there are very acute, violent forms, termed "explosive" (*meningitis cerebro-spinalis siderans, méningite foudroyante*), in which the cerebral symptoms are very severe, and the patient survives only a few days or even hours. Again, there are abortive cases. These begin with equally threatening symptoms, but after a few days completely recover with remarkable rapidity. The majority of cases last about two to four weeks. In severe cases death may come as early as the first week. The disease is often protracted to six or eight weeks' duration, or even longer, and may end in death after all. Cases that last a good while sometimes exhibit a remarkably intermittent character. Finally, there are a considerable number of mild cases in which none of the symptoms are very pronounced, and recovery is relatively early.

The symptoms of the disease may be divided into (1) the severe general symptoms, referable to the brain and spinal cord; (2) the more localized, nervous symptoms; and (3) the results of the constitutional infection, including fever and symptoms in other parts of the body.

1. Among the less definite cerebral symptoms headache is important. It is usually terribly severe. It is chiefly occipital, but sometimes is frontal or temporal. Like most of the symptoms of meningitis, the headache undergoes very frequent changes in intensity during the course of the disease. For a time it may remit, only to recur with fresh severity. Marked vertigo and a sense of fullness in the head may accompany it.

The pain in the head is re-enforced by intense pain in the nape of the neck and back, due to the spinal meningitis. There is almost always considerable tenderness along the whole spinal column. The erector spinæ is contracted, making the back straight and rigid, or even producing opisthotonos; and the head is bent backward by the reflex contraction of the neighboring muscles.

In most of the severe cases intelligence is blunted; we find all degrees of disturbance, from slight drowsiness to delirium on the one hand, or deep coma on the other. Some cases begin with marked maniacal excitement. These symptoms likewise may undergo frequent variation in their intensity. General convulsions occur in very severe cases alone, and are of evil omen.

The vomiting is also to be regarded as of cerebral origin. It frequently is an early symptom, but may be deferred.

2. Symptoms referable to the individual cerebral nerves are manifold and variable. The most frequent disturbances are in the nerves that supply the muscles of the eye. They include strabismus; nystagmus, or slow movements independent of volition; unilateral or bilateral ptosis; slow reaction of the pupils, or inequality of them, or myosis or mydriasis. In the area of distribution of the facial there is often a noticeable contraction of the muscles, giving the face a peculiar, painfully distorted look. Trismus, or tetanus of the masseters, is rare, and usually a bad sign.

Disturbance of the nerves of special sense is frequent. Deafness may be due to the stupor, but is often the result of an extension of the inflammation to the acoustic nerve. The purulent inflammation may be propagated as far as the labyrinth, or even into the middle ear. Tinnitus aurium is also frequent. Disturbances of vision are far less frequently observed, but optic neuritis has been repeatedly found by the ophthalmoscope. Severe purulent irido-choroiditis has been also observed. It is probably due to extension of the inflammation along the sheath of the optic nerve. Conjunctivitis and keratitis sometimes occur; but they are probably caused by external injuries rendered possible by the imperfect closure of the lids, or the diminished sensitiveness of the parts. We have several times found the sense of smell diminished.

Disturbances in the area of distribution of the spinal nerves are, on the whole, less frequent. The only one of value in diagnosis is the cutaneous hyperæsthesia. It is apt to be particularly severe in the extremities, and it may be so extreme that the light touch of a finger or a needle causes great pain. Sometimes there is a slight twitching in the muscles of the extremities. This has, however, no special significance. There is often rigidity and stiffness of the muscles. Kernig has called attention to the frequent appearance of flexure contractions in the legs, and sometimes also in the arms, if the patients are caused to sit up or if the thigh is passively bent upon the trunk. As might be expected, there is no invariable rule about the reflexes. The cutaneous reflexes are usually well marked, and the tendon reflexes may be; but in some cases we have found the tendon reflexes markedly diminished or even abolished. Such a condition is probably due to some lesion of the fibers of the posterior nerve-roots.

All of the nervous symptoms above enumerated result from one of two causes—either the roots of the nerves are affected by the purulent exudation, or the inflammation extends inward to the central organs themselves. This extension is the explanation also of other symptoms sometimes observed—viz., hemiplegia, paraplegia, partial convulsions, and aphasia.

3. In addition to all these nervous disturbances, we see also symptoms referable to other parts of the body. Of this class there is one cutaneous affection which is a very valuable aid to diagnosis. Herpes labialis or herpes facialis is apt to appear soon after the beginning of the meningitis. It is seen in more than half the cases, and as frequently in severe as in mild attacks. Other eruptions occur now and then—e. g., roseola, urticaria, or petechiæ. Sometimes they are so symmetrically distributed upon the two halves of the body as to suggest the idea of a nervous origin.

The digestive system seldom displays severe symptoms beyond the vomiting already mentioned. Anorexia and constipation are, indeed, usually present, as in many grave diseases. We have seen mild dysentery a few times. Now and then a slight jaundice has been noticed. The spleen is often somewhat swollen, but very rarely attains great size.

Swelling of the joints has been observed quite often; it is much more frequent in some epidemics than in others. The enlargement may be an early or a later symptom. It does not usually prove serious.

The urinary apparatus is seldom affected. The urine may contain some albumen and a few casts. Polyuria is an interesting symptom, probably of nervous origin. It is more apt to occur in the latter part of the disease. In a number of cases sugar has been found in the urine. Cystitis is a secondary disorder which is not very rare, particularly in severe cases when the catheter has been used.

Pulmonary and bronchial symptoms are likewise secondary. They occur very often in bad cases. It is evident how easily the stupor of the patient may lead to the inhalation of solid matter, with consequent bronchitis and lobular pneumonia.

Lesions of the circulatory system are rare. Acute endocarditis has been observed only a few times. The pulse is usually somewhat accelerated, seldom rendered slow. Very frequently the pulse-rate is remarkably variable, undoubtedly because of variation in the supply of nervous force. Slight irregularities in the pulse are also common. In the blood we find quite a marked leucocytosis.

4. The fever in epidemic meningitis conforms to no single type. It does not correspond at all to the severity of the other symptoms; the worst cases may run their course with little or no fever. In most instances the fever has irregular remissions. It seldom exceeds 104° (40° C.). Sometimes the fever exhibits a decidedly intermittent character. It is in these cases particularly that we find the variation in the intensity of all the symptoms of which mention has been made repeatedly. The variations in the temperature do not, however, always run parallel with the changes in the other symptoms. In mild cases the fever is usually

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In most of the severe cases intelligence is blunted; we find all degrees of disturbance, from slight drowsiness to delirium on the one hand, or deep coma on the other. Some cases begin with marked maniacal excitement. These symptoms likewise may undergo frequent variation in their intensity. General convulsions occur in very severe cases alone, and are of evil omen.

The vomiting is also to be regarded as of cerebral origin. It frequently is an early symptom, but may be deferred.

2. Symptoms referable to the individual cerebral nerves are manifold and variable. The most frequent disturbances are in the nerves that supply the muscles of the eye. They include strabismus; nystagmus, or slow movements independent of volition; unilateral or bilateral ptosis; slow reaction of the pupils, or inequality of them, or myosis or mydriasis. In the area of distribution of the facial there is often a noticeable contraction of the muscles, giving the face a peculiar, painfully distorted look. Trismus, or tetanus of the masseters, is rare, and usually a bad sign.

Disturbance of the nerves of special sense is frequent. Deafness may be due to the stupor, but is often the result of an extension of the inflammation to the acoustic nerve. The purulent inflammation may be propagated as far as the labyrinth, or even into the middle ear. Tinnitus aurium is also frequent. Disturbances of vision are far less frequently observed, but optic neuritis has been repeatedly found by the ophthalmoscope. Severe purulent irido-choroiditis has been also observed. It is probably due to extension of the inflammation along the sheath of the optic nerve. Conjunctivitis and keratitis sometimes occur; but they are probably caused by external injuries rendered possible by the imperfect closure of the lids, or the diminished sensitiveness of the parts. We have several times found the sense of smell diminished.

Disturbances in the area of distribution of the spinal nerves are, on the whole, less frequent. The only one of value in diagnosis is the cutaneous hyperæsthesia. It is apt to be particularly severe in the extremities, and it may be so extreme that bination of croupous pneumonia (*q. v.*) and purulent meningitis has been repeatedly observed. Still, it is not easy to determine whether the cause of this secondary meningitis is actually identical with that of the epidemic form (*vide supra*). In other acute diseases, such as typhoid and articular rheumatism, when they occur at the time of an epidemic, the "tendency to meningitis" is potent enough to make meningeal symptoms more frequent than usual. It has not been clearly demonstrated, however, that this fact is actually due to the epidemic meningitis.

The **prognosis** depends chiefly upon the severity of the cerebral symptoms. Yet we should be guarded in our utterances, even when the case seems mild, or has apparently made the first steps toward convalescence. The disease sometimes changes for the worse at a late period. In general the mortality is about thirty to forty per cent. Probably this estimate does not take into account many very mild cases.

Treatment is purely symptomatic. There is no specific for meningitis. A valuable remedy is cold applications. Ice-bags are placed upon the head, and, if possible, along the spine. There are long and narrow rubber bags for the latter purpose. These applications are borne well by most patients and afford decided relief. The local abstraction of blood has also an undeniably beneficial influence, however difficult this may be to explain. Leeches are put behind the ears, and cupping-glasses on the back of the neck and along the spine. Mercurial ointment is often rubbed in, not only locally but also in the same way as in treating syphilis. Its efficacy is doubtful. The narcotics are of great value. The best is morphine given subcutaneously. It lessens the pain, and often affords the uneasy and delirious patient rest and sleep. Chloral and bromide of potash may also be employed. Iodide of potash is often given internally, to the amount of twenty to thirty grains (grammes 1.5–2) in a day, especially in cases with a slow course. The evacuation of a part of the meningitic exudation by lumbar puncture (*vide supra*) is sometimes followed by a temporary improvement of the symptoms, but no actual and permanent therapeutic result has yet been obtained by lumbar puncture.

The fever hardly ever requires special treatment. Quinine exerts no influence in the intermittent type. Antipyrine is better borne, and it also, like the other nervines, sometimes relieves the nervous symptoms. Cool baths are not to be recommended, but hot baths or hot packs sometimes seem to be of decided benefit. Later, warm baths are often beneficial. Local complications—e. g., affecting the eye or the ear—require special treatment. The swelling of the joints which sometimes occurs we have thought to be somewhat relieved by salicylic acid.

CHAPTER XIX

SEPTIC AND PYÆMIC DISEASES

(*Spontaneous Septicopyæmia*)

The septic and pyæmic processes which follow serious injuries or operations belong to surgery; but analogous diseases occur in persons who are apparently in perfect condition. They take the form of an extremely severe acute infectious disease, usually fatal. There can scarcely be a doubt that in almost all these cases there is some small break in the continuity of the external skin or the mucous membrane which affords an entrance for the infectious material; but, since the infection itself is wholly unnoticed, the general disease seems a primary affection, and its correct interpretation often occasions the physician considerable difficulty. Even if the septic infection be correctly recognized, it is sometimes impossible to determine the point of origin. For such cases Leube has introduced the name of "cryptogenic" septicopyæmia. We must add, however, that in many cases at least, a careful inquiry and examination will reveal the origin of the infection. In many cases, however, a complete explanation of the occurrence of the infection is given only by the autopsy, and the origin of some cases remains obscure even after a careful autopsy.

The causes of pyæmic and septic affections are chiefly the same pus cocci which produce many circumscribed inflammations and suppurations, especially the streptococcus pyogenes, the staphylococcus pyogenes albus, and the staphylococcus pyogenes aureus. We can not make any definite clinical distinction in the septic affections according to the special cocci that produce them; but, in general, it is the rule that the severe, fatal pyæmic and septicæmic affections are most often due to streptococci—for example, many cases of pyæmia, of puerperal sepsis, of severe ulcerative endocarditis, etc. The staphylococci seem on the whole to cause more benign forms of suppurative or septic disease. As we shall see later, it is not improbable that acute articular rheumatism must be regarded as such a staphylococcus infection. Mixed infections with streptococci and staphylococci also occur, and, finally, other micro-organisms (pneumonia diplococci, gonococci) may sometimes cause forms of disease which have many points of similarity to the other septic diseases.

The essential thing in all septic infections is the general disease of the body caused by the entrance of the germs into the circulation and their increase therein. The form and severity of the disease thus caused depends probably in part upon the special variety and in part upon the varying specific "virulence" of the bacteria that have entered the system, but the form and severity also depend upon the greater or less resistance of the body, which varies in individuals. If the micrococci enter the general circulation only, they cause a large number of intoxication symptoms, due to the action of different "toxines" formed by the chemical changes in the micrococci. The micrococci, however, may collect in the smaller blood-vessels ("micrococcus foci," "micrococcus emboli"), and in this way may cause circumscribed or extensive suppuration in the various organs. In prevailing parlance we call this multiple abscess formation pyæmia, and the cases with severe (toxic) general symptoms, hæmorrhage, etc. (*vide infra*), but without suppuration, we call septicæmia or sepsis. Since, however, the two groups of symptoms may be associated with each other in many ways, we often speak of septicopyæmia.

It is very important for the correct understanding of all these conditions to bear in mind that, in different cases, different organs or parts of the body may become the chief seat for the localization of the micrococci. Hence severe local disorders may arise, which, of course, may give diverse aspects to the clinical picture according to the special localization. This is the reason why many of these affections were formerly regarded as separate diseases, although in reality they were only different localizations and forms of the same infectious process. Among these affections were the so-called "acute osteomyelitis" almost always caused by the staphylococcus pyogenes aureus, "malignant endocarditis," certain forms of "malignant erysipelas," etc. In their clinical aspect these differences are, of course, still of great importance, but the ætiological connection of all these cases must always be emphasized, because only by putting a proper stress upon this point can we obtain a correct understanding of all the combinations manifested in the clinical course of the disease.

Before we consider the details of the anatomical changes in septic affections, we would mention those circumstances (exciting causes), which, we know by ex-

perience, most commonly cause or render possible the occurrence of septic infection. In each individual case it must be our task, as we have said, either during the patient's life or at the autopsy, to determine if possible the point of origin of the infection. The following are chiefly to be considered: 1. The puerperal processes take the first place. Both after delivery, and still more frequently after abortion, the raw surface of the uterus may be the door of entrance for the septic poison, but a gross pathological change is not necessarily to be seen in the uterus itself or in its appendages. We do find, often enough, diphtheritic and gangrenous inflammation at the place where the placenta was inserted, or purulent thrombi in the veins of the uterus and of the pelvis, etc.; but in other cases the uterus is merely a gate of entrance for the poison, remaining itself normal. 2. The septic poison may also be absorbed through slight abrasions of the skin, injuries, felons, whitlows, etc.; and these may be almost completely healed by the time the severe symptoms of disease are developed. Bed-sores belong in this category. 3. Ulcers of the mucous membranes may give rise to infection. This is the explanation of those cases of sepsis which, in rare instances, follow all sorts of intestinal ulcers (simple intestinal ulcers, typhoid, dysentery, etc.), diphtheritic processes in the pharynx, even mild tonsillar affections, etc. In the description of scarlet fever and pharyngeal diphtheria we have already considered this important form of secondary sepsis in detail. 4. Lastly, we sometimes find no other source for the pyæmia than a suppurating disease of the bones, joints, or other parts, previously existing. We must here suppose that some connection was formed between the original pus cavity and the blood or lymph channels, and that thus the micrococci entered the general circulation and were enabled rapidly to increase.

The above enumeration by no means exhausts all the possibilities; still, it will be found to explain most cases. The more minutely we search for a place of entrance for the septic virus, the rarer will the "cryptogenic" cases be in which the starting-point of the infection remains unknown. [The editor has seen two clear cases, one confirmed by autopsy, of general septic infection and malignant endocarditis as a sequel of gonorrhœa.]

Pathological Anatomy.—The most striking feature at the autopsy of such cases is that there is never found a lesion of one organ exclusively. Several, or it may be almost all of the organs, exhibit numerous limited foci of disease. The lesions sometimes consist for the most part of multiple abscesses, sometimes of numerous ecchymoses, and sometimes of combinations of the two. The abscesses are found chiefly in the lungs, kidneys, liver, spleen, muscles, heart, brain, and thyroid gland. Quite extensive purulent inflammation is also found. This attacks the joints or causes phlegmon of the muscles or skin by preference, but it also attacks the pleura, the meninges, and the eye, where it causes purulent choroiditis, panophthalmitis, and purulent degeneration of the vitreous. Purulent phlebitis also occurs. The ecchymoses are most frequent upon the surface of the body, the serous membranes (pericardium, pleura), the retina, the conjunctiva, the brain, the pelvis of the kidney, etc. Beside these multiple abscesses and ecchymoses, there is frequently another disorder, which seems to be the very focus of the disease, viz., acute ulcerative endocarditis (cf. the appropriate chapter). This usually attacks the mitral valve, more rarely the valves of the aorta, and quite exceptionally the valves of the right side of the heart. Finally come a number of changes common to all severe constitutional infectious diseases —acute splenic tumor, "cloudy swelling" of the liver and kidneys, a dryness and dark-red color of the muscles, etc.

In regard to the special origin of all these symptoms we may certainly refer the abscesses, the purulent inflammation, and the endocarditis to the immediate presence of the micrococci themselves, while the parenchymatous

degenerations of different organs, the hæmorrhages, and probably the splenic enlargement and the diffuse acute nephritis, are to be regarded as due to toxic action. Microscopic examination of the internal organs gives us a very instructive insight into the morbid process. This often reveals the presence of many tiny foci of disease, in the center of which we often see a little blood-vessel completely filled with micrococci ("micrococcus embolus"). As Weigert first found, the primary action of a little focus of micrococci upon the surrounding tissues consists of a circumscribed cell necrosis, a small focus of "coagulation-necrotic" cells without nuclei. This may be followed later by circumscribed suppuration.

Clinical History.—It is our intention to discuss below those cases chiefly which are of interest to the physician rather than the surgeon—i. e., where the septicopyæmia is an apparently primary, acute, and grave disease. Many of the essential traits of this type of disease are identical with those of the pyæmia which complicates the effects of serious wounds or the inflammation subsequent to childbirth; but it is precisely because no cause at all presents itself that many cases of this form of sepsis seem so obscure, and are so often wrongly diagnosticated. Besides, the patient may be very ill indeed before the physician sees him; and this adds greatly to the difficulties of a correct diagnosis.

The beginning of the disease is usually rather abrupt. An apparently healthy person is attacked with febrile symptoms, headache, and "rheumatic" pains in the muscles, joints, and loins. There may also be gastro-intestinal symptoms of considerable severity, including vomiting and diarrhœa. Usually the patient feels ill enough to take speedily to his bed. The symptoms now increase rapidly, and develop into a severe illness which may resemble either a bad case of typhoid fever or miliary tuberculosis. Or the cerebral symptoms, such as headache, stupor, and delirium, may become so prominent that the attack seems like meningitis. If the trouble in the joints (*vide infra*) predominates and there are signs of endocarditis, the disease may at first be taken for a violent attack of acute articular rheumatism.

Taking up the separate symptoms, we shall first name those which belong to every severe acute infectious disease and have nothing characteristic about them. In this list belong the general prostration, the anorexia, the mental disturbance, the stupor and delirium, the headache, the subjective symptoms of fever, the dryness of the tongue, and finally the acute splenic tumor which can often be made out. There are, however, other and more characteristic symptoms; and it is chiefly upon these that the diagnosis rests, provided we can make one at all. These are:

1. *The Course of the Fever.*—In many cases it must be confessed that this is not at all characteristic. It may even be so like that of typhoid fever, at least for some time, as to lead to a wrong diagnosis. In other cases, however, the temperature-curve does aid us greatly, viz., when it represents an intermitting fever with marked elevations, reaching 106° (41° C.) and higher, and often accompanied by a chill, and with subsequent deep depressions. The curve may thus come to resemble closely that of a quotidian or even tertian intermittent fever. This true "pyæmic" fever, in which the rise in temperature is usually associated with a severe chill, occurs chiefly in the cases with multiple abscesses. Sometimes, again, the course of the fever is made up of similar paroxysmal elevations, separated by periods of ordinary remitting fever.

2. *Cutaneous Symptoms.*—These are very frequent, and a great aid to diagnosis. The hæmorrhages into the skin are of chief importance. They may be either punctiform petechiæ or more extensive ecchymoses. Of other cutaneous appearances, the first in relative frequency is an erythema resembling scarlatina. It is not improbable, as we have already said, that many cases which have been described as severe scarlet fever occurring during childbed were in reality septic

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disease. We also sometimes see eruptions resembling erythema exsudativum multiforme or erythema nodosum, and also in some cases roseola, pustular eruptions, and herpes. Extensive inflammations of the skin like erysipelas are especially characteristic of certain cases. They are seen on the lateral surfaces of the thorax, the back, the thighs, etc., and often end in phlegmonous suppuration. When they occur we can usually make a very positive diagnosis of septic infection.

3. *Circulatory Disturbances.*—An ability to recognize the cardiac lesions would be very desirable; but often this is impossible before death. Endocardial murmurs are often wanting, even in cases where the autopsy discloses abundant exudation and ulcers upon the valves. Still, in some cases of this sort we have found the heart-sounds noticeably deficient in clearness. Sometimes we hear very loud or low, blowing sounds. The cardiac dullness is often quite normal, in other cases somewhat increased. In some cases fibrinous or purulent pericarditis develops. Functional disturbances of the heart's action are almost always present. The heart's action is excited and very rapid (120 to 140 or more), or in rare cases abnormally slow. Irregularity and inequality of the pulse are common. The tension of the pulse is usually low, and the frequent pallor and mild cyanosis of the patient point to a diminished energy in the heart's action. In the blood we find almost invariably a more or less pronounced leucocytosis. The presence of micrococci in the blood would naturally be of the greatest significance. They have, in fact, been repeatedly found in septic affections during life, but more extensive clinical bacteriological investigations are still needed in regard to this point.

4. The grave *cerebral symptoms* are for the most part quite analogous to those of other severe acute infectious diseases. They may be present, and yet no marked objective lesions may be found after death. In other cases they have an anatomical basis—in purulent meningitis, hæmorrhagic pachymeningitis, cerebral hæmorrhage, or abscess. These conditions, just enumerated, may excite localized cerebral symptoms, e. g., hemiplegia.

5. *Affections of the joints* are comparatively frequent, and of great value in diagnosis. We may find serous, or more likely purulent inflammation, or even periarticular abscesses. If they appear early in the attack, they may, as we have said, lead to an erroneous diagnosis of acute articular rheumatism. Suppurative processes affecting the periosteum and the marrow of the bones not infrequently accompany the joint affections. Only in rare cases do the bones remain wholly unaffected, as is evident from the frequent occurrence of pain in the long bones. If there is decided suppuration in the bones we speak of an acute osteomyelitis. This is seen especially in the lower extremities. It is almost always occasioned by the staphylococcus aureus. In earlier times such cases were termed bone-typhoid. Finally, abscesses and extensive phlegmonous suppuration in the muscles are not uncommon.

6. *Renal changes* are very frequent, but abscesses and hæmorrhages may be numerous in the kidneys, or hæmorrhages in the mucous membrane of the pelvis of the kidney, without materially altering the character of the urine. In most cases, however, an acute septic nephritis is conjoined with the infarctions and abscesses, and then the urine exhibits all the characteristics of acute Bright's disease, having a small or large amount of albumen, red and white blood-corpuscles, epithelium, and casts.

7. The *pulmonary symptoms* are in part secondary. Bronchitis and lobular pneumonia develop as in all other severe constitutional diseases. The pulmonary abscesses of themselves give rise usually to no objective symptoms—or, at most, to a marked dyspnœa, out of all proportion to the scanty physical signs. Empyema is a not infrequent result of infection of the pleura, due to the foci of disease

which are situated upon the outer surface of the lungs, or perhaps sometimes to the condition of the blood. If the aspirating-needle shows the actual existence of empyema, this fact may make the diagnosis of the constitutional disease much easier. A fibrinous pleurisy of slight degree is often found at the autopsy, and it may be diagnosticated during life by the detection of a slight pleuritic friction-rub.

8. Of the *abdominal symptoms* we have already mentioned the acute splenic tumor. It is almost impossible to diagnosticate infarctions and abscesses in the spleen. If the spleen is enlarged and noticeably painful, we may suspect their existence. There are sometimes quite severe intestinal symptoms, such as a profuse "septic diarrhœa," in cases where the autopsy does not show any particularly grave lesions. Still intestinal ecchymoses and intestinal diphtheria have sometimes been observed. We should mention that often the skin has a faint jaundiced hue. This is sometimes the result of duodenal catarrh, but more frequently it is to be regarded as a "hepato-hæmatogenous" jaundice due to a decomposition of the blood.

9. *Ocular Disturbances.*—The purulent inflammations of the eye, which are probably of embolic origin and which may develop into diffuse septic panophthalmitis, have been known for some time. Lately, Litten and others have called attention to more minute changes in the fundus of the eye. These are revealed through the ophthalmoscope, and have great diagnostic value. Chief among them is retinal hæmorrhage, which is sometimes accompanied by a white spot in the center, corresponding to a necrosis of the retina in that place; but there may be similar white spots without hæmorrhage.

Course of the Disease and Prognosis.—In severe general septic infection death sometimes occurs in a few days. In other cases the disease continues longer, the symptoms lasting two or three weeks or even more. Improvement sometimes takes place, followed by a relapse. Here, too, the result is often fatal, although the infection may finally be completely overcome. We may also accept as certain that there are mild, curable forms of septic disease. In these the signs of general infection may predominate, such as fever with general intoxication, cardiac weakness, pains in the joints, albuminuria or exanthemata, or, in other cases, the chief symptoms may be due to a special localization of the germs or their toxines (acute endocarditis, septic nephritis, septic inflammation of the serous membranes, septic enteritis, etc.). As we shall see later, acute articular rheumatism (polyarthritis acuta) is probably nothing but a benign septic infection.

Diagnosis.—It is self-evident that a disease which combines symptoms so manifold and so ambiguous must be very difficult to recognize. We will recapitulate the chief diseases to be excluded. A case may greatly resemble typhoid fever when there is persistent prostration, diarrhœa, an eruption like roseola, and an enlarged spleen. In discriminating, we should consider with great care the possible ætiology—e. g., external injuries, etc.; we should remember that most septic affections begin rather more rapidly; and we should look for swelling of the joints, cutaneous ecchymoses, nephritis, phlegmonous suppurations, an intermitting form of fever, and septic disease of the retina. We have repeatedly found that leucocytosis was a very valuable sign, since it is almost always absent in typhoid fever, but a positive result from Widal's serum reaction (*vide* page 22) renders a diagnosis of typhoid very probable. It is all the more possible for the disease to resemble meningitis, because, as we have said, meningeal disturbance may be one of the symptoms of the sepsis and color the whole picture. Here the characteristic symptoms of septic poisoning already mentioned would be of some value in diagnosis, and the physical signs of endocarditis or of a greatly enlarged spleen would be worth still more. There may be much difficulty in the differential diagnosis between acute sepsis and acute miliary tuberculosis. Here we should consider carefully each separate symptom, and, above all, the ætiology, searching for something that would explain the occurrence of sepsis on the one hand, or of acute miliary tuberculosis (*q. v.*) on the other. If we found miliary tubercles in the choroid by means of the ophthalmoscope, or tubercle bacilli in the sputum or in the blood (which is, of course, not always possible), all doubt would vanish. At the beginning of a septic attack the rigors may arouse suspicions of intermittent fever. Usually the early appearance of other symptoms corrects this idea; but, if not, the powerlessness of quinine will. If a severe acute nephritis has developed itself in a septic case, all the symptoms may be erroneously referred to uræmia; but persistent observation will usually lead us to the right conclusion. As to the conditions of great prostration resembling acute sepsis, which occur in acute (primary) ulcerative endocarditis and in severe articular rheumatism, see the appropriate chapters.

In general the diagnosis of acute sepsis can rarely be settled during the first days of the disease, but on further observation and with due attention a tolerably definite diagnosis can often be made. The chief elements for diagnosis are a careful consideration of the ætiology, attention to the whole clinical picture, and to any individual symptoms especially characteristic of septic infection (purulent inflammation, chills and fever, cutaneous and retinal hæmorrhages, nephritis, marked leucocytosis, etc.).

We must refer the reader to the appropriate chapters for the morbid conditions allied to acute sepsis, such as "primary acute endocarditis," "acute articular rheumatism," "purpura hæmorrhagica," etc.

The **treatment** is merely symptomatic. Of course we try again and again to cut short the attacks of fever by large doses of quinine or antipyrine, but never with any but temporary success. With marked swelling of the joints we may try salicylic acid. Of other remedies, cardiac stimulants are most frequently employed, and narcotics if necessary. Great stress is to be laid on keeping up the physical strength by careful nourishment. Circumscribed suppuration (empyema, phlegmon, etc.), when it exists, of course requires the proper surgical treatment.

CHAPTER XX

HYDROPHOBIA

(*Rabies canina. Lyssa*)

Ætiology. Rabies in Dogs.—A peculiar infectious disease sometimes occurs in dogs, and more rarely in some other animals—viz., the wolf, fox, cat, etc. Men who are bitten by the animal may catch the disease, and thus suffer from very severe symptoms originating in the central nervous system.

Two forms of madness are distinguished in dogs—the raving madness and the quiet madness. Bollinger describes the raving form as beginning with prodromata, the melancholy stage, lasting one to three days. The animal is low-spirited, timorous, and without appetite. Then comes the stage of irritation or of mania, in which the animal is possessed with an impulse to bite. It seems determined to run away and rove about, and it utters a peculiar howl. The dog will not touch his ordinary food, but he often swallows straw, hair, earth, bits of wood, etc. In the third or paralytic stage paralysis appears. The dog looks lean and wretched, and always dies on the tenth day at the latest. In what is called the quiet madness there is no maniacal stage. The symptoms of paralysis, affecting chiefly the hind limbs and the lower jaw, occur earlier and are sooner fatal. Marked pathological changes are not found. There are pulmonary and intestinal catarrh and passive congestion of the viscera, and the stomach often contains foreign bodies in place of the usual partially digested food.

[On the Western plains hydrophobia is said not infrequently to follow skunk bites. The bite is inflicted during sleep on persons passing the night in the open air or in tents to which the animal can gain access.]

Rabies is transferred to the human being almost invariably by the bite of some raving animal, and this animal is almost always a dog; much more rarely a wolf or cat. It is of practical importance, and it has been repeatedly confirmed, that the bite of such an animal, although still in the incubation period of hydrophobia, can convey the disease to man. The poison, which is not yet known in its pure form, is evidently contained in the saliva or slaver and in the blood of mad animals, and can, by means of these substances, be successfully inoculated in other animals. Pasteur has discovered another way to produce the disease experimentally. He takes minute portions of the brain, medulla oblongata, or some other internal viscus of a mad dog, and either injects them into the veins of a healthy animal, or trephines, and then inserts them beneath the meninges. The virulence of the rabic poison when thus manipulated undergoes, under special conditions, very peculiar alterations, which will be detailed at the close of this chapter.

About one half of those who are bitten by mad animals exhibit no subsequent symptoms. Still this can scarcely be due to inherent immunity from the disease, and it must result chiefly from imperfect infection. Experience has shown that infection is especially apt to follow when a nerve has been injured by the bite; and infection occurs more readily the larger and the nearer the brain the wound is. Wounds of the head are therefore the most dangerous. The duration of incubation until rabies finally breaks out seems to vary greatly. As a rule it is about three to six months, but observers have reported instances both of shorter and of much longer duration.

Clinical History.—The disease begins with a general feeling of indisposition, anorexia, headache, and uneasiness. This last is partially explained, to be sure, by a dread of what is impending. If the bite was in the face, frequent convulsive sneezing may occur. Even now, in this prodromal stage, a marked aversion to liquids is a usual and early symptom. The attempt to swallow excites slight convulsive disturbances. Painful sensations may arise once more in the bitten place, although this has usually been cicatrized long before, and the neighboring lymph-glands are often found to be swollen.

Only a day or two later the second, hydrophobic stage begins. The especial characteristic of this consists in the peculiar attacks of tonic convulsions. The pharynx suffers most, but convulsions also attack the muscles of respiration and those of the trunk and extremities. A terrible feeling of anxiety and oppression accompanies these attacks, so that one who has once witnessed the sight can never forget it. The convulsions always seem to be reflex, and are produced by the slightest causes, particularly by any attempt to swallow, or sometimes by the mere sight of water. They recur at gradually diminishing intervals, and last from a few minutes to half an hour. The excitement of the patient may reach the pitch of delirium or mania. The pulse is at first full and rapid, but later it is small and irregular. The temperature is usually only slightly elevated, but it may be high. There is great thirst, accompanied by burning pain in the throat. Usually there is marked salivation.

This condition lasts one to three days. Then death occurs, ushered in by violent convulsions. Death may also be preceded by a brief third stage of paralysis, during which there are no convulsive attacks. Cases of recovery in man, if they ever happen, are extremely rare.

the light touch of a finger or a needle causes great pain. Sometimes there is a slight twitching in the muscles of the extremities. This has, however, no special significance. There is often rigidity and stiffness of the muscles. Kernig has called attention to the frequent appearance of flexure contractions in the legs, and sometimes also in the arms, if the patients are caused to sit up or if the thigh is passively bent upon the trunk. As might be expected, there is no invariable rule about the reflexes. The cutaneous reflexes are usually well marked, and the tendon reflexes may be; but in some cases we have found the tendon reflexes markedly diminished or even abolished. Such a condition is probably due to some lesion of the fibers of the posterior nerve-roots.

All of the nervous symptoms above enumerated result from one of two causes —either the roots of the nerves are affected by the purulent exudation, or the inflammation extends inward to the central organs themselves. This extension is the explanation also of other symptoms sometimes observed—viz., hemiplegia, paraplegia, partial convulsions, and aphasia.

3. In addition to all these nervous disturbances, we see also symptoms referable to other parts of the body. Of this class there is one cutaneous affection which is a very valuable aid to diagnosis. Herpes labialis or herpes facialis is apt to appear soon after the beginning of the meningitis. It is seen in more than half the cases, and as frequently in severe as in mild attacks. Other eruptions occur now and then—e. g., roseola, urticaria, or petechiæ. Sometimes they are so symmetrically distributed upon the two halves of the body as to suggest the idea of a nervous origin.

The digestive system seldom displays severe symptoms beyond the vomiting already mentioned. Anorexia and constipation are, indeed, usually present, as in many grave diseases. We have seen mild dysentery a few times. Now and then a slight jaundice has been noticed. The spleen is often somewhat swollen, but very rarely attains great size.

Swelling of the joints has been observed quite often; it is much more frequent in some epidemics than in others. The enlargement may be an early or a later symptom. It does not usually prove serious.

The urinary apparatus is seldom affected. The urine may contain some albumen and a few casts. Polyuria is an interesting symptom, probably of nervous origin. It is more apt to occur in the latter part of the disease. In a number of cases sugar has been found in the urine. Cystitis is a secondary disorder which is not very rare, particularly in severe cases when the catheter has been used.

Pulmonary and bronchial symptoms are likewise secondary. They occur very often in bad cases. It is evident how easily the stupor of the patient may lead to the inhalation of solid matter, with consequent bronchitis and lobular pneumonia.

Lesions of the circulatory system are rare. Acute endocarditis has been observed only a few times. The pulse is usually somewhat accelerated, seldom rendered slow. Very frequently the pulse-rate is remarkably variable, undoubtedly because of variation in the supply of nervous force. Slight irregularities in the pulse are also common. In the blood we find quite a marked leucocytosis.

4. The fever in epidemic meningitis conforms to no single type. It does not correspond at all to the severity of the other symptoms; the worst cases may run their course with little or no fever. In most instances the fever has irregular remissions. It seldom exceeds 104° (40° C.). Sometimes the fever exhibits a decidedly intermittent character. It is in these cases particularly that we find the variation in the intensity of all the symptoms of which mention has been made repeatedly. The variations in the temperature do not, however, always run parallel with the changes in the other symptoms. In mild cases the fever is usually

moderate and brief. The abortive attacks may present high temperatures at first, but these quickly abate. In case of a fatal issue there is sometimes hyperpyrexia before death, reaching 108° to 109° (42°–43° C.). In the severer but not fatal cases the fever declines slowly but irregularly. The fever may be over, long before the other symptoms disappear.

It is impossible to portray all the forms, symptoms, and courses the disease may have. The chief varieties have been already mentioned; but in reality these are only types which run into one another without sharply defined border-lines. It is in itself a characteristic of epidemic meningitis that most of the more tedious cases have a variable, uncertain course. We may even meet with a complete intermission of all the symptoms, lasting for quite a while, so that the return of the trouble may fairly be called a relapse.

Sequelæ are not rare after severe cases. Persistent deafness is the most frequent. It results from the complications, already mentioned, which affect the labyrinth and the middle ear. Little children may become deaf and dumb. Again, vision may be deranged, because of retinitis, atrophy of the optic nerve, or corneal opacities, etc. It is not very rare for meningitis to leave grave nervous disorders behind it. These are frequently the symptoms of a chronic hydrocephalus. We may observe headache, sudden unconsciousness, or even convulsions, mental impairment, and weakness of the extremities. Or there may be localized disturbances due to permanent injury of limited portions of the brain or spinal cord, such as hemiplegia, paraplegia, and aphasia. From many of these conditions there may be a gradual recovery, but others prove incurable.

The **diagnosis** of cerebro-spinal meningitis is not difficult in a well-developed case, particularly if the prevalence of an epidemic puts us in mind of the disease. Diagnosis is more difficult in sporadic cases, and most difficult of all when the patient does not come under observation till he is very ill and when we can not obtain the previous history. Important factors are the abrupt onset, the speedy appearance of grave cerebral symptoms, the characteristic headache and pain in the back, the stiffness of the neck, and the herpes labialis.

If we find evident symptoms of meningitis, we have still to decide whether the case is one of primary epidemic disease, or secondary, due perhaps to extension from some other part. Bearing this last possibility in mind, we should examine the ears carefully; for, as is well known, chronic otitis media may set up a purulent meningitis. Again, it may be very difficult to exclude a tubercular meningitis. Here we should consider other circumstances that might render tuberculosis probable, such as the general condition of the patient, heredity, previous pleurisy, the results of thoracic examination, or scrofulous disease of the bones or joints. The existence of herpes points toward epidemic meningitis, for it is exceptional in the other forms of the disease. It is sometimes difficult to distinguish between meningitis and severe cases of other acute infectious diseases—e. g., typhoid fever and septic diseases. Here we must weigh all the circumstances carefully.

Lumbar puncture has greatly aided the diagnosis of meningitis. This was recently introduced by Quincke, and it has often been performed since. As Quincke first showed, we can usually reach the subarachnoid space of the cauda equina without difficulty between the third and fourth lumbar vertebræ with an aspirating needle about eight centimetres long. The patient should lie on one side. In this way we can obtain more or less of any meningitic exudation that may be present. In epidemic meningitis we often obtain a sero-purulent or purulent exudation which can be examined bacteriologically. [The discovery of the diplococcus intracellularis in the fluid withdrawn by lumbar puncture is of extreme importance in the diagnosis of doubtful or sporadic cases.—K.]

This is a good opportunity to mention the secondary meningitis which is said to occur with relative frequency just at the time of an epidemic. The combination of croupous pneumonia (*q. v.*) and purulent meningitis has been repeatedly observed. Still, it is not easy to determine whether the cause of this secondary meningitis is actually identical with that of the epidemic form (*vide supra*). In other acute diseases, such as typhoid and articular rheumatism, when they occur at the time of an epidemic, the "tendency to meningitis" is potent enough to make meningeal symptoms more frequent than usual. It has not been clearly demonstrated, however, that this fact is actually due to the epidemic meningitis.

The **prognosis** depends chiefly upon the severity of the cerebral symptoms. Yet we should be guarded in our utterances, even when the case seems mild, or has apparently made the first steps toward convalescence. The disease sometimes changes for the worse at a late period. In general the mortality is about thirty to forty per cent. Probably this estimate does not take into account many very mild cases.

Treatment is purely symptomatic. There is no specific for meningitis. A valuable remedy is cold applications. Ice-bags are placed upon the head, and, if possible, along the spine. There are long and narrow rubber bags for the latter purpose. These applications are borne well by most patients and afford decided relief. The local abstraction of blood has also an undeniably beneficial influence, however difficult this may be to explain. Leeches are put behind the ears, and cupping-glasses on the back of the neck and along the spine. Mercurial ointment is often rubbed in, not only locally but also in the same way as in treating syphilis. Its efficacy is doubtful. The narcotics are of great value. The best is morphine given subcutaneously. It lessens the pain, and often affords the uneasy and delirious patient rest and sleep. Chloral and bromide of potash may also be employed. Iodide of potash is often given internally, to the amount of twenty to thirty grains (grammes 1.5–2) in a day, especially in cases with a slow course. The evacuation of a part of the meningitic exudation by lumbar puncture (*vide supra*) is sometimes followed by a temporary improvement of the symptoms, but no actual and permanent therapeutic result has yet been obtained by lumbar puncture.

The fever hardly ever requires special treatment. Quinine exerts no influence in the intermittent type. Antipyrine is better borne, and it also, like the other nervines, sometimes relieves the nervous symptoms. Cool baths are not to be recommended, but hot baths or hot packs sometimes seem to be of decided benefit. Later, warm baths are often beneficial. Local complications—e. g., affecting the eye or the ear—require special treatment. The swelling of the joints which sometimes occurs we have thought to be somewhat relieved by salicylic acid.

CHAPTER XIX

SEPTIC AND PYÆMIC DISEASES

(*Spontaneous Septicopyæmia*)

The septic and pyæmic processes which follow serious injuries or operations belong to surgery; but analogous diseases occur in persons who are apparently in perfect condition. They take the form of an extremely severe acute infectious disease, usually fatal. There can scarcely be a doubt that in almost all these cases there is some small break in the continuity of the external skin or the mucous membrane which affords an entrance for the infectious material; but, since the infection itself is wholly unnoticed, the general disease seems a primary affection, and its correct interpretation often occasions the physician considerable difficulty. Even if the septic infection be correctly recognized, it is sometimes impossible to determine the point of origin. For such cases Leube has introduced the name of "cryptogenic" septicopyæmia. We must add, however, that in many cases at least, a careful inquiry and examination will reveal the origin of the infection. In many cases, however, a complete explanation of the occurrence of the infection is given only by the autopsy, and the origin of some cases remains obscure even after a careful autopsy.

The causes of pyæmic and septic affections are chiefly the same pus cocci which produce many circumscribed inflammations and suppurations, especially the streptococcus pyogenes, the staphylococcus pyogenes albus, and the staphylococcus pyogenes aureus. We can not make any definite clinical distinction in the septic affections according to the special cocci that produce them; but, in general, it is the rule that the severe, fatal pyæmic and septicæmic affections are most often due to streptococci—for example, many cases of pyæmia, of puerperal sepsis, of severe ulcerative endocarditis, etc. The staphylococci seem on the whole to cause more benign forms of suppurative or septic disease. As we shall see later, it is not improbable that acute articular rheumatism must be regarded as such a staphylococcus infection. Mixed infections with streptococci and staphylococci also occur, and, finally, other micro-organisms (pneumonia diplococci, gonococci) may sometimes cause forms of disease which have many points of similarity to the other septic diseases.

The essential thing in all septic infections is the general disease of the body caused by the entrance of the germs into the circulation and their increase therein. The form and severity of the disease thus caused depends probably in part upon the special variety and in part upon the varying specific "virulence" of the bacteria that have entered the system, but the form and severity also depend upon the greater or less resistance of the body, which varies in individuals. If the micrococci enter the general circulation only, they cause a large number of intoxication symptoms, due to the action of different "toxines" formed by the chemical changes in the micrococci. The micrococci, however, may collect in the smaller blood-vessels ("micrococcus foci," "micrococcus emboli"), and in this way may cause circumscribed or extensive suppuration in the various organs. In prevailing parlance we call this multiple abscess formation pyæmia, and the cases with severe (toxic) general symptoms, hæmorrhage, etc. (*vide infra*), but without suppuration, we call septicæmia or sepsis. Since, however, the two groups of symptoms may be associated with each other in many ways, we often speak of septicopyæmia.

It is very important for the correct understanding of all these conditions to bear in mind that, in different cases, different organs or parts of the body may become the chief seat for the localization of the micrococci. Hence severe local disorders may arise, which, of course, may give diverse aspects to the clinical picture according to the special localization. This is the reason why many of these affections were formerly regarded as separate diseases, although in reality they were only different localizations and forms of the same infectious process. Among these affections were the so-called "acute osteomyelitis" almost always caused by the staphylococcus pyogenes aureus, "malignant endocarditis," certain forms of "malignant erysipelas," etc. In their clinical aspect these differences are, of course, still of great importance, but the ætiological connection of all these cases must always be emphasized, because only by putting a proper stress upon this point can we obtain a correct understanding of all the combinations manifested in the clinical course of the disease.

Before we consider the details of the anatomical changes in septic affections, we would mention those circumstances (exciting causes), which, we know by ex-

perience, most commonly cause or render possible the occurrence of septic infection. In each individual case it must be our task, as we have said, either during the patient's life or at the autopsy, to determine if possible the point of origin of the infection. The following are chiefly to be considered: 1. The puerperal processes take the first place. Both after delivery, and still more frequently after abortion, the raw surface of the uterus may be the door of entrance for the septic poison, but a gross pathological change is not necessarily to be seen in the uterus itself or in its appendages. We do find, often enough, diphtheritic and gangrenous inflammation at the place where the placenta was inserted, or purulent thrombi in the veins of the uterus and of the pelvis, etc.; but in other cases the uterus is merely a gate of entrance for the poison, remaining itself normal. 2. The septic poison may also be absorbed through slight abrasions of the skin, injuries, felons, whitlows, etc.; and these may be almost completely healed by the time the severe symptoms of disease are developed. Bed-sores belong in this category. 3. Ulcers of the mucous membranes may give rise to infection. This is the explanation of those cases of sepsis which, in rare instances, follow all sorts of intestinal ulcers (simple intestinal ulcers, typhoid, dysentery, etc.), diphtheritic processes in the pharynx, even mild tonsillar affections, etc. In the description of scarlet fever and pharyngeal diphtheria we have already considered this important form of secondary sepsis in detail. 4. Lastly, we sometimes find no other source for the pyæmia than a suppurating disease of the bones, joints, or other parts, previously existing. We must here suppose that some connection was formed between the original pus cavity and the blood or lymph channels, and that thus the micrococci entered the general circulation and were enabled rapidly to increase.

The above enumeration by no means exhausts all the possibilities; still, it will be found to explain most cases. The more minutely we search for a place of entrance for the septic virus, the rarer will the "cryptogenic" cases be in which the starting-point of the infection remains unknown. [The editor has seen two clear cases, one confirmed by autopsy, of general septic infection and malignant endocarditis as a sequel of gonorrhœa.]

Pathological Anatomy.—The most striking feature at the autopsy of such cases is that there is never found a lesion of one organ exclusively. Several, or it may be almost all of the organs, exhibit numerous limited foci of disease. The lesions sometimes consist for the most part of multiple abscesses, sometimes of numerous ecchymoses, and sometimes of combinations of the two. The abscesses are found chiefly in the lungs, kidneys, liver, spleen, muscles, heart, brain, and thyroid gland. Quite extensive purulent inflammation is also found. This attacks the joints or causes phlegmon of the muscles or skin by preference, but it also attacks the pleura, the meninges, and the eye, where it causes purulent choroiditis, panophthalmitis, and purulent degeneration of the vitreous. Purulent phlebitis also occurs. The ecchymoses are most frequent upon the surface of the body, the serous membranes (pericardium, pleura), the retina, the conjunctiva, the brain, the pelvis of the kidney, etc. Beside these multiple abscesses and ecchymoses, there is frequently another disorder, which seems to be the very focus of the disease, viz., acute ulcerative endocarditis (cf. the appropriate chapter). This usually attacks the mitral valve, more rarely the valves of the aorta, and quite exceptionally the valves of the right side of the heart. Finally come a number of changes common to all severe constitutional infectious diseases —acute splenic tumor, "cloudy swelling" of the liver and kidneys, a dryness and dark-red color of the muscles, etc.

In regard to the special origin of all these symptoms we may certainly refer the abscesses, the purulent inflammation, and the endocarditis to the immediate presence of the micrococci themselves, while the parenchymatous

degenerations of different organs, the hæmorrhages, and probably the splenic enlargement and the diffuse acute nephritis, are to be regarded as due to toxic action. Microscopic examination of the internal organs gives us a very instructive insight into the morbid process. This often reveals the presence of many tiny foci of disease, in the center of which we often see a little blood-vessel completely filled with micrococci ("micrococcus embolus"). As Weigert first found, the primary action of a little focus of micrococci upon the surrounding tissues consists of a circumscribed cell necrosis, a small focus of "coagulation-necrotic" cells without nuclei. This may be followed later by circumscribed suppuration.

Clinical History.—It is our intention to discuss below those cases chiefly which are of interest to the physician rather than the surgeon—i. e., where the septicopyæmia is an apparently primary, acute, and grave disease. Many of the essential traits of this type of disease are identical with those of the pyæmia which complicates the effects of serious wounds or the inflammation subsequent to childbirth; but it is precisely because no cause at all presents itself that many cases of this form of sepsis seem so obscure, and are so often wrongly diagnosticated. Besides, the patient may be very ill indeed before the physician sees him; and this adds greatly to the difficulties of a correct diagnosis.

The beginning of the disease is usually rather abrupt. An apparently healthy person is attacked with febrile symptoms, headache, and "rheumatic" pains in the muscles, joints, and loins. There may also be gastro-intestinal symptoms of considerable severity, including vomiting and diarrhœa. Usually the patient feels ill enough to take speedily to his bed. The symptoms now increase rapidly, and develop into a severe illness which may resemble either a bad case of typhoid fever or miliary tuberculosis. Or the cerebral symptoms, such as headache, stupor, and delirium, may become so prominent that the attack seems like meningitis. If the trouble in the joints (*vide infra*) predominates and there are signs of endocarditis, the disease may at first be taken for a violent attack of acute articular rheumatism.

Taking up the separate symptoms, we shall first name those which belong to every severe acute infectious disease and have nothing characteristic about them. In this list belong the general prostration, the anorexia, the mental disturbance, the stupor and delirium, the headache, the subjective symptoms of fever, the dryness of the tongue, and finally the acute splenic tumor which can often be made out. There are, however, other and more characteristic symptoms; and it is chiefly upon these that the diagnosis rests, provided we can make one at all. These are:

1. *The Course of the Fever.*—In many cases it must be confessed that this is not at all characteristic. It may even be so like that of typhoid fever, at least for some time, as to lead to a wrong diagnosis. In other cases, however, the temperature-curve does aid us greatly, viz., when it represents an intermitting fever with marked elevations, reaching 106° (41° C.) and higher, and often accompanied by a chill, and with subsequent deep depressions. The curve may thus come to resemble closely that of a quotidian or even tertian intermittent fever. This true "pyæmic" fever, in which the rise in temperature is usually associated with a severe chill, occurs chiefly in the cases with multiple abscesses. Sometimes, again, the course of the fever is made up of similar paroxysmal elevations, separated by periods of ordinary remitting fever.

2. *Cutaneous Symptoms.*—These are very frequent, and a great aid to diagnosis. The hæmorrhages into the skin are of chief importance. They may be either punctiform petechiæ or more extensive ecchymoses. Of other cutaneous appearances, the first in relative frequency is an erythema resembling scarlatina. It is not improbable, as we have already said, that many cases which have been described as severe scarlet fever occurring during childbed were in reality septic

disease. We also sometimes see eruptions resembling erythema exsudativum multiforme or erythema nodosum, and also in some cases roseola, pustular eruptions, and herpes. Extensive inflammations of the skin like erysipelas are especially characteristic of certain cases. They are seen on the lateral surfaces of the thorax, the back, the thighs, etc., and often end in phlegmonous suppuration. When they occur we can usually make a very positive diagnosis of septic infection.

3. *Circulatory Disturbances.*—An ability to recognize the cardiac lesions would be very desirable; but often this is impossible before death. Endocardial murmurs are often wanting, even in cases where the autopsy discloses abundant exudation and ulcers upon the valves. Still, in some cases of this sort we have found the heart-sounds noticeably deficient in clearness. Sometimes we hear very loud or low, blowing sounds. The cardiac dullness is often quite normal, in other cases somewhat increased. In some cases fibrinous or purulent pericarditis develops. Functional disturbances of the heart's action are almost always present. The heart's action is excited and very rapid (120 to 140 or more), or in rare cases abnormally slow. Irregularity and inequality of the pulse are common. The tension of the pulse is usually low, and the frequent pallor and mild cyanosis of the patient point to a diminished energy in the heart's action. In the blood we find almost invariably a more or less pronounced leucocytosis. The presence of micrococci in the blood would naturally be of the greatest significance. They have, in fact, been repeatedly found in septic affections during life, but more extensive clinical bacteriological investigations are still needed in regard to this point.

4. The grave *cerebral symptoms* are for the most part quite analogous to those of other severe acute infectious diseases. They may be present, and yet no marked objective lesions may be found after death. In other cases they have an anatomical basis—in purulent meningitis, hæmorrhagic pachymeningitis, cerebral hæmorrhage, or abscess. These conditions, just enumerated, may excite localized cerebral symptoms, e. g., hemiplegia.

5. *Affections of the joints* are comparatively frequent, and of great value in diagnosis. We may find serous, or more likely purulent inflammation, or even periarticular abscesses. If they appear early in the attack, they may, as we have said, lead to an erroneous diagnosis of acute articular rheumatism. Suppurative processes affecting the periosteum and the marrow of the bones not infrequently accompany the joint affections. Only in rare cases do the bones remain wholly unaffected, as is evident from the frequent occurrence of pain in the long bones. If there is decided suppuration in the bones we speak of an acute osteomyelitis. This is seen especially in the lower extremities. It is almost always occasioned by the staphylococcus aureus. In earlier times such cases were termed bone-typhoid. Finally, abscesses and extensive phlegmonous suppuration in the muscles are not uncommon.

6. *Renal changes* are very frequent, but abscesses and hæmorrhages may be numerous in the kidneys, or hæmorrhages in the mucous membrane of the pelvis of the kidney, without materially altering the character of the urine. In most cases, however, an acute septic nephritis is conjoined with the infarctions and abscesses, and then the urine exhibits all the characteristics of acute Bright's disease, having a small or large amount of albumen, red and white blood-corpuscles, epithelium, and casts.

7. The *pulmonary symptoms* are in part secondary. Bronchitis and lobular pneumonia develop as in all other severe constitutional diseases. The pulmonary abscesses of themselves give rise usually to no objective symptoms—or, at most, to a marked dyspnœa, out of all proportion to the scanty physical signs. Empyema is a not infrequent result of infection of the pleura, due to the foci of disease

which are situated upon the outer surface of the lungs, or perhaps sometimes to the condition of the blood. If the aspirating-needle shows the actual existence of empyema, this fact may make the diagnosis of the constitutional disease much easier. A fibrinous pleurisy of slight degree is often found at the autopsy, and it may be diagnosticated during life by the detection of a slight pleuritic friction-rub.

8. Of the *abdominal symptoms* we have already mentioned the acute splenic tumor. It is almost impossible to diagnosticate infarctions and abscesses in the spleen. If the spleen is enlarged and noticeably painful, we may suspect their existence. There are sometimes quite severe intestinal symptoms, such as a profuse "septic diarrhœa," in cases where the autopsy does not show any particularly grave lesions. Still intestinal ecchymoses and intestinal diphtheria have sometimes been observed. We should mention that often the skin has a faint jaundiced hue. This is sometimes the result of duodenal catarrh, but more frequently it is to be regarded as a "hepato-hæmatogenous" jaundice due to a decomposition of the blood.

9. *Ocular Disturbances.*—The purulent inflammations of the eye, which are probably of embolic origin and which may develop into diffuse septic panophthalmitis, have been known for some time. Lately, Litten and others have called attention to more minute changes in the fundus of the eye. These are revealed through the ophthalmoscope, and have great diagnostic value. Chief among them is retinal hæmorrhage, which is sometimes accompanied by a white spot in the center, corresponding to a necrosis of the retina in that place; but there may be similar white spots without hæmorrhage.

Course of the Disease and Prognosis.—In severe general septic infection death sometimes occurs in a few days. In other cases the disease continues longer, the symptoms lasting two or three weeks or even more. Improvement sometimes takes place, followed by a relapse. Here, too, the result is often fatal, although the infection may finally be completely overcome. We may also accept as certain that there are mild, curable forms of septic disease. In these the signs of general infection may predominate, such as fever with general intoxication, cardiac weakness, pains in the joints, albuminuria or exanthemata, or, in other cases, the chief symptoms may be due to a special localization of the germs or their toxines (acute endocarditis, septic nephritis, septic inflammation of the serous membranes, septic enteritis, etc.). As we shall see later, acute articular rheumatism (polyarthritis acuta) is probably nothing but a benign septic infection.

Diagnosis.—It is self-evident that a disease which combines symptoms so manifold and so ambiguous must be very difficult to recognize. We will recapitulate the chief diseases to be excluded. A case may greatly resemble typhoid fever when there is persistent prostration, diarrhœa, an eruption like roseola, and an enlarged spleen. In discriminating, we should consider with great care the possible ætiology—e. g., external injuries, etc.; we should remember that most septic affections begin rather more rapidly; and we should look for swelling of the joints, cutaneous ecchymoses, nephritis, phlegmonous suppurations, an intermitting form of fever, and septic disease of the retina. We have repeatedly found that leucocytosis was a very valuable sign, since it is almost always absent in typhoid fever, but a positive result from Widal's serum reaction (*vide* page 29) renders a diagnosis of typhoid very probable. It is all the more possible for the disease to resemble meningitis, because, as we have said, meningeal disturbance may be one of the symptoms of the sepsis and color the whole picture. Here the characteristic symptoms of septic poisoning already mentioned would be of some value in diagnosis, and the physical signs of endocarditis or of a greatly enlarged spleen would be worth still more. There may be much difficulty in the differential diagnosis between acute sepsis and acute miliary than one thousand embryos. These latter begin their travels soon after birth, and reach the voluntary muscles. As to the routes they choose we are still somewhat in doubt. Some authorities state that the trichinæ penetrate through the walls of the intestine and the abdominal cavity into the connective tissue. Others affirm that they enter the lymphatic vessels, or exceptionally the blood-vessels. They penetrate into the primitive fibers of the muscles, and cause them to disintegrate. Finally, they coil themselves up, attain the size of muscular trichinæ in about fourteen days, and become encapsulated. Each capsule usually contains but one, although it may inclose as many as four. The capsule is formed partly by an excretion from the trichina, and partly from the reflex hyperplasia of the surrounding connective tissue. The process of development is now complete. The muscular trichinæ seem, unlike the intestinal form, to have a very long lease of life, and usually endure till the death of their host. They are often found accidentally at autopsies. They are most abundant in the diaphragm, the intercostal muscles, the muscles of the larynx and throat, and the biceps.

Ætiology of Trichinosis.—The only cause yet known for trichinatous disease in man is the ingestion of trichinatous raw or underdone pork—e. g., smoked ham. Swine are pre-eminently subject to trichinæ. They probably become infected in various ways, e. g., from the fæces of human beings and swine suffering from trichinosis, or through the ingestion of the trichinatous flesh of other swine. The waste of slaughter-houses is often fed out to swine, and the disease thus disseminated. Many affirm that swine are also infected by eating rats infested with trichinæ, but the contrary condition, whereby rats become trichinatous from eating the flesh of diseased hogs, seems more in accordance with the facts.

Clinical History.—The symptoms in man correspond in general to the developmental and vital processes of the trichinæ, as above depicted. In individual cases, however, the separate stages are quite often obscured, probably because all the parasites do not develop simultaneously, or because there are relapses. The first symptoms are gastro-intestinal. At the commencement there is a feeling of pressure in the epigastrium, with nausea and vomiting. Later, diarrhœa is prominent, becoming in some cases so violent as to remind one of cholera. It is not impossible, although rare, to find intestinal trichinæ in the stools. Sometimes there is constipation instead of diarrhœa. In some cases the initial gastro-intestinal symptoms are but slight. Frequently, even in the beginning of the disease, there is complaint of pain and stiffness in the muscles, too early for it to be due to the migrations of the trichinæ.

The genuine severe muscular symptoms, due to the myositis produced by the trichinæ in the muscles, do not begin till the second week, or even later. In many cases, where the invading parasites seem to be relatively few in number, the muscular symptoms are slight, or wholly absent. In the more severe cases, however, they may be extremely violent and distressing. The muscles become swollen, firm and hard, very tender on pressure, and very painful. The patient avoids all movements and contraction of the muscles as much as possible, lying motionless in bed, with flexed arms and with legs either extended or likewise flexed. The patellar reflex almost always disappears, and on testing the electrical reactions there is found a considerable diminution of muscular excitability to both the galvanic and faradic currents, sometimes associated with delayed contractions, and abnormally long duration of the same after the stimulus ceases (Eisenlohr). The masseters and the pharyngeal and laryngeal muscles are attacked, so that there is difficulty in mastication and deglutition, and hoarseness. The involvement of the ocular muscles causes pain in the eyes. The condition of the diaphragm, intercostals, and abdominal muscles causes serious difficulty in respiration. There is distressing dyspnœa, and expectoration is so hampered that secretions accumulate in the air-passages. In some fatal cases of trichinosis death is due principally to this impairment of respiration. The condition may be aggravated by diffuse bronchitis or lobular pneumonia.

Third in the list of important symptoms comes œdema. It appears toward the end of the first week in the eyelids. Somewhat later it involves the upper and lower extremities. What produces it is not quite clear. It has been regarded as in part inflammatory and in part the result of occlusion and thrombosis of the smaller lymphatics. Cutaneous eruptions also develop—e. g., vesicles, wheals, petechiæ, and pustules. Frequently there is profuse perspiration, consequent upon which abundant crops of miliaria or sudamina may appear.

In well-marked cases there may be quite high fever and other severe constitutional symptoms in addition to the local disturbances already discussed. The temperature may for a time reach 104° to 106° (40°–41° C.); but the fever is seldom continuous for any length of time, being usually interrupted by frequent and considerable intermissions. There are also a rapid pulse, headache, stupor, and other symptoms suggesting typhus or typhoid fever. In fact, the first case in which trichinosis was recognized at the autopsy (by Zenker of Dresden) had been regarded before death as typhoid. The urine may be albuminous; and, in rare instances, nephritis is seen.

[The blood shows a leucocytosis, with a great relative and absolute increase in polymorphonuclear eosinophilic cells.—V.]

The duration of the disease varies widely. There are mild cases often unrecognized, which get well after slight symptoms have lasted two or three weeks. More pronounced cases occupy six to eight weeks, or even a much longer time. Of the more severe cases about one third prove fatal, usually from the fourth to the sixth week. Sometimes death is caused by the severity of the constitutional disturbance, but usually from disabled respiration. Even if the case ends favorably, recovery is often very tedious.

Pathology.—The autopsy reveals little that is characteristic excepting the changes in the muscles. There are sometimes the signs of hæmorrhagic catarrhal inflammation of the small intestine. The spleen is not enlarged. Very often the liver is decidedly fatty. What should cause this in trichinosis has not yet been determined. The lungs often present islets of lobular pneumonia, or sometimes even of gangrene. The trichinæ are found in the muscles, beginning with the fifth week. They can be recognized by the naked eye as little whitish lines. We have already named the muscles chiefly infested. Under the microscope we see the fibers in which the trichinæ lie transformed into a fine granular mass. The nuclei of the muscular fibrillæ are greatly increased in number in the neighborhood of the coiled-up parasite. Finally, the sarcolemma collapses, and becomes greatly thickened upon its external surface by a hyperplasia of connective tissue. The muscles also present many other degenerative changes, such as a flaky disintegration, waxy degeneration, and the formation of vacuoles. There is furthermore a marked increase of nuclei in the interstitial tissue of the muscles. Within the intestines are sometimes to be found, even after several weeks' illness, numerous living intestinal trichinæ—a fact of importance from a therapeutic point of view.

Diagnosis.—The diagnosis of trichinosis is generally not difficult, since the peculiar symptoms of the disease, especially the extensive, painful inflammation of the muscles and the œdema, occur in this way in only one other rare disease, primary acute polymyositis (*q. v.*). Trichinosis is distinguished from this partly by the peculiar ætiology (affecting several people, the use of raw pork, etc.), and partly by the initial gastro-intestinal symptoms. Trichinosis may also be confounded with multiple neuritis and perhaps with acute articular rheumatism, but careful observation of the patient will usually make the diagnosis clear. An absolutely certain diagnosis may be arrived at by finding intestinal trichinæ in the dejections, but, as we have said, this is not an easy matter.

Treatment.—The trichinæ may still be alive in pork that has been smoked or salted or half-cooked—e. g., many sausages are unsafe. The only possible prophylaxis, therefore, as far as the individual is concerned, is to avoid all such food. A real protection for the public against the disease is also afforded by governmental microscopic inspection of meat, as already established in many places.

When an individual has become infected with trichinæ, if it is possible that intestinal trichinæ still are present, the treatment must always begin with the exhibition of purgatives, such as compound infusion of senna, calomel, or castor-oil. Since trichinæ may be found in the intestines as long as eight weeks after the beginning of the first symptoms, we should not neglect local action on the intestinal contents even in the later stages of the disease. Of the remedies which are calculated to destroy the intestinal trichinæ, glycerine, which was first recommended by Fiedler, seems to be the most efficient. It must be given in rather large doses, say a tablespoonful every hour. Other drugs are much less reliable, but we will name among them benzine in the total daily dose of one to two drachms (grm. 4 to 8) in capsules, and picric acid in pills—the daily dose being five to eight grains (grm. 0.3–0.5).

Treatment is unfortunately almost wholly powerless against the myositic symptoms of trichinosis and their sequelæ. The muscular pains can be alleviated by narcotics, particularly morphine subcutaneously, poultices, and chloroform-oil as an embrocation.* Protracted warm baths are excellent. Antipyrine and salicylic acid are also said to do good in many cases.

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* Generally one part of chloroform to ten of olive-oil. It is not officinal in Germany, but is weaker than the linimentum chloroformi (U. S. P.).—Trans.

DISEASES OF THE RESPIRATORY ORGANS

SECTION I

DISEASES OF THE NOSE[*]

CHAPTER I

CORYZA

(*Snuffles. Rhinitis. Cold in the Head*)

Ætiology.—The well-known symptoms of coryza depend upon a catarrhal inflammation of the nasal mucous membrane. Although this catarrh may often be due to infectious influences, still we can not deny that it is one of those diseases which may be caused by taking cold. Daily experience teaches us how often coryza follows an evident exposure to cold, such as wetting the feet, but it is of course possible that exposure to cold may merely favor infection. We may mention its contagiousness as an argument in favor of its infectious character, and this may be illustrated by the fact that it may be conveyed by handkerchiefs, kissing, etc., but an experimental transmission of common coryza has not yet been successful.

Coryza may also arise from the action of chemical irritants or mechanical irritants, such as dust, on the nasal mucous membrane. The iodine coryza, which occurs from the internal use of iodine, is especially noteworthy. In this form iodine can easily be detected in the nasal secretion. The idiosyncrasy of many people to ipecacuanha is also well known, the very smell of it setting up a coryza. A severe coryza is the chief symptom, too, in hay fever,[†] which is probably due to the action of the pollen of certain grasses on the respiratory mucous membrane. Finally, we must bear in mind that coryza may often be only a symptom of some other disease such as measles, syphilis, or glanders, and that severe purulent inflammation of the nasal mucous membrane may be excited by the presence of the secretion from a gonorrhœal or blennorrhœal conjunctivitis.

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[*] Special treatises on the pathology and therapeutics of nasal diseases are to be found in the following works: Michel, "Krankheiten der Nasenhöhle." Fraenkel, "Diseases of the Nose," in "Ziemssen's Cyclopedia." Störk, "Klinik der Krankheiten des Kehlkopfes, der Nase, und des Rachens." Schech, "Krankheiten der Mundhöhle, des Rachens, und der Nase." Moldenhauer, "Krankheiten der Nasenhöhlen." C. Rosenthal, "Die Erkrankungen der Nase und des Kehlkopfes." W. Kiesselbach in Penzoldt and Stintzing's "Handbuch der Therapie," vol. iii, etc. [Bosworth, "Diseases of the Nose and Throat." Burnett, "Diseases of the Ear, Nose, and Throat." Ingals, "Diseases of the Chest, Throat, and Nasal Cavities."]

[†] The disease called hay fever (*catarrhus æstivus*) is of frequent occurrence in England and North America, although rare with us in Germany. It usually affects men in middle life, less often women. Some individuals are peculiarly liable to the disease. For them an attack may be produced merely by walking across a meadow or near a grain-field at that season when the grasses are in bloom, i. e., about May to July. As already intimated, it is supposed that the grains of pollen excite the disease, being diffused in the air and thus drawn into the nostrils. At any rate, they have repeatedly been found in the nasal secretion and also in the tears of affected persons. The *symptoms* consist in a very severe coryza, with burning of the nose and violent sneezing. The erectile tissue of the nose is probably acutely swollen. Usually these symptoms are accompanied by a well-marked conjunctivitis with œdema of the eyelids. In severer cases there is furthermore a catarrh of the larynx and bronchi. There is frequently a tendency to violent attacks of asthma ("hay-asthma"), especially at night (see the chapter on bronchial asthma). The *treatment* consists first in avoiding the cause by change of

The **symptoms** of coryza are in most of the milder cases of a local nature only. The secretion is troublesome; at first it is scanty and mucous, but later it becomes more abundant and watery; and sometimes it is purulent. The nasal passages are not infrequently closed from the swelling of the mucous membrane. The patient necessarily has to breathe through the mouth, which explains the well-known nasal speech. This closure of the nares may give rise to dangerous attacks of dyspnœa in children, especially in infants, who have to breathe through the nose when sucking at the breast. The sense of smell is always diminished. The local sensations of pain and burning are due chiefly to a mild inflammation of the skin of the nostrils and upper lip set up by the irritation of the secretion. The irritated condition of the inflamed mucous membrane occasions a feeling of tickling and itching in the nose, and frequently by a reflex action violent sneezing. The symptoms are more severe if the cavities adjacent to the nose are attacked by catarrh, and if in them accumulations of secretion occur. Marked pain in the forehead occurs in catarrh of the frontal sinuses. The sinuses of the ethmoid and sphenoid bones, and the antrum of Highmore, may also be implicated. Much more frequently a severe coryza sets up inflammation in adjacent mucous membranes. Thus we find, following coryza, conjunctivitis, affection of the ear, sore throat, or laryngitis. In persistent coryza eczema is not infrequently excited on the skin of the upper lip, and mention has already been made of the fact that coryza may sometimes act as the exciting cause of erysipelas.

In severe coryza we may sometimes have quite a marked general disturbance, and often slight elevations of temperature. The " feverish cold " of children, for instance, is well known.

Treatment.—Special treatment is usually unnecessary, for most cases recover of themselves in a few days. It seems doubtful whether the internal use of quinine in fresh coryza is of service, as is claimed. With abundant secretion, especially in fresh cases, Hager's " coryza remedy " (as an inhalation) is worthy of trial; this consists of ten parts each of alcohol and carbolic acid, and five parts of ammonia-water. Painting the nasal mucous membrane with a solution of cocaine (two to five per cent.) is also greatly praised [but it may lead to the formation of the cocaine habit.—K.] When the secretion forms abundant dry scabs, an attempt should be made to wash them out by injections of warm fluids, such as warm milk. The upper lip and the nostrils should be smeared with vaseline or simple ointment to protect the skin from the action of the secretion. Only in the rare cases of a severe purulent catarrh can energetic local treatment of the nasal mucous membrane be necessary. Here we may use douches or sprays of astringents like tannin or alum, or let the patient snuff them up, or we may apply caustics like nitrate of silver. In children who can not blow the nose, it is advisable to cleanse the nose frequently with a small sponge and a one-per-cent. solution of boracic acid.

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residence, as by going to the sea-shore. For the nasal catarrh douches are most to be recommended, such as a solution of 1 part of quinine to 500-1,000 parts of water, or a solution of carbolic acid. The administration of iodide of potassium might be tried, particularly where there is asthma.

CHAPTER II

CHRONIC RHINITIS

(*Rhinitis chronica hypertrophica et atrophica. Ozæna*)

1. **Chronic Hypertrophic Rhinitis.**—It is in many cases impossible to determine the causes of hypertrophic rhinitis. Sometimes the condition seems to develop as a sequel to frequently repeated nasal catarrh, although in this case the relation is often reversed—it being the chronic rhinitis which occasions a predisposition to the frequent acute exacerbations of the catarrh. Certain diatheses (anæmia, scrofula) appear to influence the development of the disease. This is also true of occupations which expose the individual to dust or smoke, and sometimes true of malformation of the nose (for instance, deviation of the septum) and perhaps also of hereditary predisposition.

The anatomical changes consist of a slow but progressive swelling and hypertrophy of the mucous membrane. This seems spongy, and of a red or reddish-gray color. The greatest change is almost always found over the inferior turbinated bone, and next to that over the middle turbinated. In advanced cases the mucous membrane presents rough, uneven swellings, and even polypi. These changes are often visible upon inspection of the nostrils anteriorly, but they may escape discovery until a rhinoscopic examination of the posterior choanæ is made.

The disturbance occasioned by chronic hypertrophic rhinitis may be very considerable. Respiration through the nose is obstructed, the voice becomes nasal, the senses of smell and taste are impaired. The nasal secretion is for the most part increased, but it may be diminished. Often there is a tendency to nose-bleed. Many patients complain also of headache.

The frequent involvement of neighboring organs is important. This applies particularly to the ear. Deafness is caused both by the obstruction of the openings of the Eustachian tubes, and not infrequently also by extension of the catarrh to the lining membrane of the tubes and the middle ear. Very frequently the disease is associated with chronic naso-pharyngitis or pharyngitis. The visible portion of the nose is not infrequently affected, as shown by redness and swelling of its tip.

A fact of especial interest is that such a diseased state of the nasal mucous membrane may give rise to reflex neuroses (Voltolini, Hack, and others). Although, in our opinion, many of the specialists on the nose go too far in this direction, there is no room for doubt that attacks of migraine, vertigo, certain varieties of headache, and, above all, many forms of bronchial asthma, may bear a close relation to diseases of the nose. We shall revert to this point later on. (See especially the chapter on bronchial asthma.)

The *treatment* of chronic hypertrophic rhinitis, in order to be successful, demands complete destruction and removal of the hypertrophic portions by means of the galvano-cautery. For particulars, we must refer to the directions of specialists. In milder cases, however, benefit may follow the insufflation of a powder composed of one part of nitrate of silver to ten or twenty of common starch, or applications of lunar caustic may be made.

2. **Simple Chronic Atrophic and Fetid Atrophic Rhinitis. Ozæna Simplex.**—The disease consists in a slow, progressive atrophy not only of the mucous membrane with its vessels and glands, but finally also of the bones, and this atrophy is not preceded by hypertrophy. Thus the nasal cavities become abnormally large. The turbinated bones grow smaller and smaller, so that finally they are represented merely by narrow ridges. Furthermore, the scanty purulent secretion has a tendency to dry up and form adherent greenish-yellow scabs and crusts, which undergo a peculiar putrefactive decomposition and give rise to an unbear-

able stench. We do not yet know what special form of bacterium causes this putrefactive decomposition of the secretion. When this very characteristic and extremely repulsive stench occurs in the nose, we usually give the affection the brief name of ozæna (ὄζειν, to stink), but in the other and practically less important cases we speak of a simple atrophic rhinitis. The latter may sometimes become ozæna.

Ozæna generally develops in childhood. It usually begins insidiously, but in other cases apparently is a sequel of some acute disease, such as measles, etc. Anæmia and scrofula deservedly rank as important predisposing causes. Störk thinks that ozæna is usually connected with syphilis in the parents. It is also worthy of note that patients with ozæna often have from birth a nose with a flat, broad bridge, which is perhaps a family trait, favoring the development of the disease.

The subjective symptoms are often not marked. This is partly explained by the fact that the patient has usually completely lost his sense of smell, but for that very reason the discomfort of his friends may be the greater. The feeling of dryness in the nose may prove annoying, and there are often complaints of headache and of pressure in the eyes. Inasmuch as the naso-pharynx and the posterior pharyngeal wall are almost always implicated in the process, the patient often suffers from hacking and a tendency to cough and vomit. Such portions of the secretion as are swallowed sometimes give rise to a considerable chronic disturbance of the stomach. Upon physical examination we are first struck by the unusual breadth of the nostrils. With the rhinoscope the extent of the atrophy is still better seen. The mucous membrane is pale or slightly red, and covered with dry scabs. Sometimes superficial ulcers are formed. Usually, as we have said, the superior portion of the pharyngeal mucous membrane shares in the disease. The posterior wall of the pharynx is seen to be atrophied, smooth as if it were varnished, and often covered with crusts. The process may involve the soft palate, and even the larynx, and not infrequently the disease is associated with inflammation of the middle ear.

It should be added that the true ozæna must not be confounded with other processes which likewise give rise to a foul smell from the nose. Tuberculous disease of the nasal mucous membrane and nasal bones is not rare, particularly in "scrofulous" children (Demme); nor should we forget the syphilitic affections of the nose, tertiary and hereditary syphilis.

Treatment.—An alleviation of ozæna can be accomplished only by the aid of local applications as prescribed by specialists. Even then the treatment is a prolonged one, and demands much patience on the part of both patient and physician. A complete cure of atrophic rhinitis is impossible. Besides local applications, we must also bear in mind the necessity of constitutional treatment.

The object of local treatment is to remove the secretion in order to get rid of the bad odor. Nasal douches, with disinfectant solutions such as permanganate of potassium (1–3,000) or carbolic or corrosive sublimate, are here most useful. The solution is carefully injected into the nose, or the fluid is allowed to run gently into one nostril from an irrigator while the patient keeps his head bent forward; it then runs through the naso-pharynx and out through the other nostril. The patient soon learns to retain the fluid in the pharynx and eject it from the mouth. All nasal douches must at first be used with care and under the eye of the physician. The fluid should be injected at the lowest pressure possible, so that none of it may enter the adjacent cavities or the Eustachian tube. Furthermore, all solutions used as a douche must be lukewarm—90° to 95° (25°–28° R.). Besides the regular use of douches, painting and the insufflation of powders, such as boracic acid, aceto-tartrate of aluminum, etc., are sometimes employed. The insertion of tampons of dry absorbent cotton is to be recommended; under their use the secretion dries less easily and the odor is diminished. These tampons should be changed daily. It is advantageous to medicate the tampons with a one-per-cent. solution of creoline or with Peruvian balsam or some similar drug. Tincture of iodine is also recommended. Of late many attempts have been made to treat chronic nasal catarrh by the galvano-cautery. With regard to the details of this as well as of other methods, we must refer to special treatises on the subject.

CHAPTER III

NOSE-BLEED

(*Epistaxis*)

ALTHOUGH in many cases nose-bleed is only a symptom, still we are justified in a short description of it, partly because frequently repeated nose-bleeds often first call our attention to some other existing disease, and partly because the treatment is of practical importance.

Many persons are subject to habitual nose-bleed, which comes on either from slight causes, from violently blowing the nose, from physical exertion, from overheating, or even without any special cause. This habitual nose-bleed is sometimes, but by no means always, the sign of a general hæmorrhagic diathesis, which is hereditary in many families. (See the chapter on hæmophilia.) In other cases the nose-bleed is the result of some chronic disease. It occurs especially in leukæmia, in disease of the heart, in contracted kidney, and as a symptom of the so-called hæmorrhagic diseases, such as scurvy, purpura hæmorrhagica, etc. Finally, diseases of the nose itself may give rise to hæmorrhage. The periodic occurrence of nose-bleed in young girls as a form of so-called "vicarious menstruation" has often been described, but we must always be very guarded in admitting it as a fact. Nose-bleed sometimes occurs at the beginning of many infectious diseases, especially typhoid and scarlet fevers, etc. We may add that the point of hæmorrhage is very frequently at the anterior lower end of the cartilaginous septum (Kiesselbach).

In many cases nose-bleed is a very transitory symptom, wholly without danger, and in one sense it may even be advantageous. When there is headache, or a feeling of fullness in the head, there is often relief after an epistaxis. Nose-bleed is dangerous, however, when it takes place in those who are already weak and anæmic, or when it is so persistent and abundant as to cause a marked general anæmia. The latter is recognized by the pallor of the face, by the appearance of general weakness, by vertigo, tinnitus, and a weakened pulse. In such cases the physician's interference is always necessary. In every case of nose-bleed it is important to examine the posterior wall of the pharynx in order to see whether the blood is not flowing backward from the posterior nares. The hæmorrhage is often thought to stop when no more blood comes from the nostrils, and yet the blood keeps flowing posteriorly.

In every severe nose-bleed rest is the chief thing to be enjoined, and the patient must be told to avoid unnecessarily blowing, wiping, or drying the nose. By quietly and persistently closing the nostrils with a handkerchief a thrombus? is clot. often formed without any further medication, and the bleeding stops. The application of cold water (iced water), in which a little vinegar may be put, is a good thing. If the bleeding does not stop, we may next try a tampon of common absorbent cotton or styptic cotton in the nostril from which the blood comes. If this does not succeed, the posterior nares must be plugged by means of a "Bellocq's canula." In case of emergency we may use an elastic catheter, which is passed through the inferior meatus into the pharynx and out by the mouth. The tampon is fastened to the catheter and brought up into the posterior nares by drawing the catheter back through the nose. Internal remedies to check the flow of blood are very uncertain in their action.

SECTION II

Diseases of the Larynx

CHAPTER I

ACUTE LARYNGEAL CATARRH

(*Acute Laryngitis*)

Ætiology.—Taking cold plays a prominent part in the popular ætiology of acute laryngeal catarrh. Its influence can not be wholly denied, although the more intimate relation between taking cold and the origin of a catarrh is still unknown. The disposition to laryngitis differs very much in different people, so that some take a catarrh much more easily and more frequently than others. Besides cold, direct irritants which attack the laryngeal mucous membrane often set up a laryngitis; among these are in particular the inhalation of smoke and of noxious gases and vapors. Many laryngeal catarrhs, too, arise from excessive speaking, shouting, or singing, particularly if other injurious influences act on the larynx at the same time. Finally, laryngitis may appear as a complication or as a secondary affection, in other diseases, especially in measles, less frequently in typhoid, scarlet fever, and erysipelas. Catarrh of the larynx is very often combined with catarrh of the nose, the pharynx, and the larger bronchi.

Symptomatology.—Although the symptoms of laryngitis usually make the diagnosis easy and certain, yet an accurate understanding of the extent and intensity of the catarrh can be obtained only by a laryngoscopic examination,* which therefore should be employed in every severe case. The laryngeal mirror shows a decided reddening and swelling of the mucous membrane, varying with the intensity of the catarrh, and most marked on the true and false vocal cords and between the arytænoid cartilages. We often see small collections of mucus here and there on the membrane. In individual cases different parts of the larynx are especially affected. In intense inflammations superficial erosions are often met with, especially on the vocal cords. In other cases the mucous membrane shows a grayish-white coloring in some places, due to a thickening of the epithelium. Small hæmorrhages in the mucous membrane are also occasionally seen. Very often we see on phonation an incomplete closure of the glottis, so that a little oval space is left between the vocal cords. This slight "catarrhal paresis of the vocal cords" is probably of muscular origin, and depends chiefly upon an affection of the thyro-arytænoid muscles.

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* More extensive observations on laryngoscopy and on many details of the pathology of laryngeal diseases, which have been carefully investigated by specialists and which can not be mentioned here, are to be found in the following works: Türck, "Klinik der Krankheiten des Kehlkopfes," 1866. Semeleder, "Laryngoskopie," 1863. Tobold, "Laryngoskopie," 1874. Störk, "Klinik der Krankheiten des Kehlkopfes, der Nase, u. des Rachens," 1880. Mackenzie, "Diseases of the Throat and Nose," 1880. B. Fraenkel and v. Ziemssen, "Diseases of the Larynx," in Ziemssen's "Cyclopædia." Gottstein, "Krankheiten des Kehlkopfes," third edition, 1890. Schrötter, "Vorlesungen über die Krankheiten des Kehlkopfes." [See also the treatises of Bosworth, Burnett, and Ingals referred to on page 131.]

Of the other symptoms of laryngeal catarrh, hoarseness is particularly to be mentioned, for in many cases the diagnosis of laryngitis may be made from this alone. It is either due directly to the anatomical changes of the cords, or to the paresis just mentioned. The degree of hoarseness is of course very different in different cases, and varies from a simple "roughening" or "deadening" of the voice to a complete loss of voice (aphonia).

The cough in laryngitis may be very severe, and is often recognizable by its harsh, hoarse ring as a "laryngeal cough." It is usually dry at first, and later on it is associated with a scanty muco-purulent expectoration, which is sometimes tinged with blood.

Pain in the larynx is generally only moderate. The subjective symptoms consist chiefly of a disagreeable feeling of itching, burning, and dryness in the throat. After prolonged speaking, however, the pain in the larynx may sometimes be quite severe. External pressure on the larynx is often somewhat painful. Difficulty in swallowing, when it occurs, is due usually to an accompanying pharyngitis, but it may also be dependent upon an affection of the epiglottis and the arytænoid cartilages.

The general health is affected in very different degrees. Many patients feel quite well except for the hoarseness, while others are affected with considerable debility, mild headache, and even at times slight febrile disturbances. Of late years we have frequently seen cases of primary acute laryngitis, with great hoarseness and marked catarrhal inflammation of the upper part of the larynx, especially of the vocal cords. These cases begin with high fever (over 104° [40° C.]) and quite severe general symptoms, and make a complete recovery in a week or two. They apparently have an infectious origin, and are perhaps connected with influenza.

Dyspnœa is not present in the common laryngitis of adults, even if there is decided swelling of the false vocal cords or of the ary-epiglottic folds. There is, however, a severe form of acute laryngitis, the so-called *laryngitis hypoglottica acuta gravis* (*chorditis vocalis inferior*), affecting not only children, but adults, in which well-marked symptoms of suffocation may be present. In this form there is an acute, very well-marked swelling of the mucous membrane in the inferior, sub-chordal, laryngeal space, which leads to a stenosis. The rare cases of phlegmonous inflammation of the larynx may also cause considerable stenosis and dyspnœa.

In children, however, on account of the greater narrowness of the child's larynx, symptoms of stenosis are not rare even in the milder forms of laryngitis, and therefore they have led to the establishment of a special disease, the so-called false croup.

The **false croup** (*laryngitis stridula*) of children usually follows a slight coryza. A harsh, hollow, ringing cough comes on, almost always suddenly and usually at night, by which the child is awakened out of sleep. The paroxysms of coughing are broken by long-drawn, noisy inspirations. The child is anxious and restless, the respiration is labored. The accessory muscles of respiration are brought into action, but the deep inspiratory retraction of the lower intercostal spaces and the epigastrium shows how imperfectly the air enters the lungs. The pulse is small and rapid. The attack lasts several hours, when the cough gradually becomes looser and the breathing easier. Finally the child falls asleep and usually wakes the next morning quite lively and playful, a slight cough being the only relic of the terrifying events of the night before. The next night the same severe attack may be repeated, and perhaps for two or three nights more. After that there remains, as a rule, nothing but a slight catarrh, which completely disappears in a week or two. The anatomical cause of false croup is acute laryngitis with marked swelling of the mucous membrane on and below the vocal cords. In the narrow space of the child's larynx this speedily causes marked stenosis, and the individual attacks are probably excited by the accumulation and drying of the secretion during the night. There is never any true croupous-diphtheritic change to be seen either in the pharynx or the larynx. It is remarkable that many children, and sometimes several children of the same family, have a specially marked predisposition to false croup. The statement, therefore, that a child has had the croup several times almost always means that it has had this form of false croup just described.

Acute laryngitis lasts only a few days in mild cases, and a week or more in severe cases. With improper care and unreasonable conduct on the patient's part an acute catarrh may run into the chronic form. We hardly ever see a fatal result in adults, even in the severe form, and in children false croup very rarely has an unfavorable termination unless the child is extremely weak or rachitic.

The **treatment** of acute laryngitis requires that especial attention be paid to the removal of all injurious influences. In every severe laryngitis the patient should stay in his room, and children are better off in bed. The patient should talk as little as possible. In all severe cases smoking, too, is to be forbidden. It is a good plan to furnish plenty of warm drink. Hot milk, mixed with Seltzer or Ems water, is readily taken by most patients. If there is a steam atomizer at our disposal, we may let the patient inhale simple steam, or a one- or two-per-cent. solution of common salt. Inhalations of astringents are usually unnecessary. The patient may also breathe steam without any special apparatus. When there is marked irritation from coughing we may give a little morphine. With more marked local symptoms, especially if there is much pain on swallowing, from swelling of the epiglottis and the mucous membrane over the arytænoid cartilages, the patient may suck pieces of ice slowly. In severe cases of acute laryngitis, with evident symptoms of stenosis, ice must be energetically used as an internal and an external application. Sometimes, too, a few leeches applied in the region of the larynx afford distinct relief. Among external applications a mustard plaster over the front of the neck is to be recommended when there are marked local symptoms. Cold, wet compresses about the neck are also of advantage in all cases.

In the false croup of children we should use, as a rule, the same treatment as has just been described. The child should take plenty of warm drink, inhale warm steam or salt solution, and a mustard paste or hot poultices should be applied to the neck. An ice-bag on the neck is sometimes useful. We should be rather cautious with regard to the favorite treatment with emetics, such as ipecac and sulphate of copper, although it can not be denied that they sometimes work very well in severe dyspnœa.

These means are entirely sufficient for the treatment of acute laryngitis. It is only exceptionally that we find ourselves led to employ in acute laryngeal catarrh an energetic local treatment of the laryngeal mucous membrane, like painting with a 1–15 solution of nitrate of silver.

We must bear in mind that a rational hardening process is of distinct prophylactic value in persons, especially in children, with a recognized tendency to laryngitis, sore throat, etc. The best method is to bathe the neck and chest with cold water regularly morning and night.

[A mild emetic can do no possible harm in false croup, and very often cuts the attack short. The application of a sponge, moistened with water as hot as the child will bear, to the region of the larynx deserves mention.]

CHAPTER II

CHRONIC LARYNGITIS

(Chronic Laryngeal Catarrh)

Ætiology.—Chronic laryngitis develops from an acute catarrh, or comes on gradually from the action of injurious influences on the larynx (see the preceding chapter). Chronic laryngitis, therefore, is in many cases due to the occupation, and is seen especially in singers, public speakers, criers, inn-keepers, workmen exposed to dust, etc. It is very frequent in drunkards, and in such cases it is almost always associated with a chronic pharyngitis. It is not probably true that, as frequently stated, too long a uvula may set up laryngitis by irritation of the entrance to the larynx.

Symptomatology.—A laryngoscopic examination is very desirable in acute laryngeal catarrh, but it is the physician's absolute duty to make one in chronic laryngitis, for only too frequently a persistent hoarseness is referred to a "simple" catarrh when the laryngoscope gives quite another cause for it, such as paralysis of the vocal cords or new growths. Furthermore, we must always remember that a chronic laryngitis may be a complication of tuberculosis, syphilis, or chronic nephritis. We should never omit a careful examination of the rest of the body as well as the larynx.

The laryngoscopic appearance in chronic catarrh may be so like that in an acute catarrh that we can not distinguish between them without the history obtained from the patient. The redness of the mucous membrane, however, is usually less intense, and the vocal cords have more of a dirty grayish-red appearance. In most cases there is also considerable swelling of the mucous membrane as well as redness. Much more rarely we find atrophic conditions, resembling those of ozæna and sometimes associated with it. Quite frequently in persistent catarrhs a thickening of particular parts of the mucous membrane is developed, especially of the folds between the arytænoid cartilages. This swelling is of practical importance, because it furnishes a mechanical hindrance to the closure of the arytænoid cartilages, and in that way contributes to the development of the hoarseness. We also find marked thickening of the false vocal cords (especially in public speakers and preachers) and of the true vocal cords. Virchow described, under the name of *pachydermia laryngis*, a condition of the larynx seen especially in drinkers and characterized by a thickening of the epithelium over the whole laryngeal mucous membrane. Türck has described a peculiar form of chronic laryngitis, in which rough prominences are formed in the middle of the true vocal cords, under the name of *chorditis tuberosa*. We not infrequently find in chronic catarrh superficial erosions, especially on the vocal cords. We never see actual ulceration in simple laryngitis. We also very often see a disturbance of motion of one or both vocal cords, especially incomplete closure, due sometimes to muscular paresis and sometimes to mechanical conditions.

The other symptoms of chronic laryngitis are hoarseness, cough, and abnormal sensations in the larynx. The hoarseness is of every degree, from mere roughness, frequent "cracking" of the voice, to almost complete aphonia. The cough is ringing, hoarse, deep, and rough. The expectoration is scanty, usually simply mucous, but sometimes a little bloody. The subjective sensations in the larynx are a feeling of burning and itching, and of dryness and tickling. They usually increase after any protracted use of the voice.

We must also mention as a peculiar and very rare but practically important form of chronic laryngitis the **chorditis vocalis inferior hypertrophica** (Gerhardt), or **laryngitis hypoglottica chronica hypertrophica** (Ziemssen). In this form there is a very gradual hypertrophy, and finally a contraction of the mucous and especially the submucous connective tissue in the inferior laryngeal space. More rarely the same changes are seen in the upper part of the larynx. The special symptom of the disease, besides a chronic hoarseness, is the appearance of a gradually increasing stenosis of the larynx. The respiration is always labored, the inspiration noisy and protracted. In many cases there are at times such attacks of suffocation that life can be prolonged only by tracheotomy. The diagnosis can be made only by the aid of the laryngoscope. We see beneath the glottis a little fissure surrounded by the thick and swollen mucous membrane of the laryngeal walls.

The nature of this affection is not yet fully explained. It often seems to be a simple chronic hypertrophic inflammation, but in other case Schrötter considers it the same bacillary disease of the mucous membranes which is known as *rhinoscleroma* when it occurs in the nose.

The **treatment of chronic laryngeal catarrh** is always a tedious and laborious task, the success of which depends in great measure upon the good will and energy of the patient. In the first place, then, we should endeavor to remove as far as possible those injurious influences which have excited and kept up the catarrh. It is often easier to give good advice here than to follow it. Nevertheless, it is the duty of the physician to impress upon the patient the urgent necessity of taking care of the larynx, and to forbid as far as possible all protracted speaking, singing, staying in smoke or dust, smoking, and the use of alcohol.

Local treatment takes the second place. Among the most useful means to employ are sprays of astringent solutions, such as a one-per-cent. solution of either tannin or alum. If there is much secretion of mucus, turpentine inhalations are advisable. When there is great irritability of the larynx, the patient may also inhale vaporized narcotics, a mixture of fifty parts of cherry-laurel water with a thousand parts of water, or a four-per-cent. solution of bromide of potassium. The inhalations should be used two or three times a day, and last about five minutes each time. Direct applications to the larynx are much more effective than inhalations, but these can be employed only by the aid of a laryngeal mirror. Of these we use, first of all, nitrate of silver, at first in a weak solution (one to fifty); later in a more concentrated form (one to ten, or even one to five). These applications are made every two or three days. Besides nitrate of silver, the larynx may also be painted with pure tincture of iodine, or with iodine and glycerine (iodine, 4 parts, iodide of potassium, 6 parts, and glycerine, 500 parts), or with concentrated solutions of alum or tannin.

Water-cures are also often prescribed in chronic catarrh of the larynx. These are of advantage from the greater care which the patient takes, and from the good air, the catarrh improves. Empirically, we prescribe, especially for "full-blooded" patients, the cold sulphur springs, such as Nenndorf, Eilsen, or Weilbach, or the sulphate of sodium waters, such as Carlsbad and Marienbad, while we send those of delicate constitutions to Ems, Salzbrunn, Salzungen, Reichenhall, etc.

The treatment of laryngitis hypertrophica, when it leads to stenosis, must be mechanical. Schrötter, in particular, has devised several methods of dilating the stenosis gradually by the introduction of bougies and harder dilators. The details of this treatment are to be found in the later special works referred to above.

CHAPTER III

LARYNGEAL PERICHONDRITIS

Ætiology and Pathological Anatomy.—The inflammation of the perichondrium of the laryngeal cartilages is in very rare cases apparently a primary disease. It is much more frequently secondary to other laryngeal affections, especially tuberculosis and syphilis of the larynx. Furthermore, it develops secondarily in severe acute diseases, most frequently in typhoid fever, more rarely in small-pox, diphtheria, etc. Superficial ulcerative processes in the mucous membrane often precede the perichondritis in these cases, and the participation of the perichondrium in the inflammation arises from their gradual deepening. Anatomically, we have to do as a rule with a purulent inflammation, which usually leads to the formation of circumscribed abscesses. Most laryngeal abscesses have their origin in the perichondrium.* The perichondrium is in part destroyed by the abscess and in part elevated from the cartilage. The cartilage then becomes necrotic, breaks in pieces, and is expelled in small particles or in masses.

Perichondritis occurs most frequently in the cricoid and arytænoid cartilages, much more rarely on the internal or external surface of the thyroid cartilage. Hence we distinguish an internal and an external perichondritis. A perichondritis of the epiglottis has also been repeatedly observed.

Symptomatology.—In the rare cases of primary perichondritis, marked laryngeal symptoms are speedily developed in a person previously healthy. These symptoms are pain and tenderness on pressure over the larynx, hoarseness, and cough; and to them are usually soon added the signs of a dangerous stenosis of the larynx. In secondary cases, which occur almost always in patients who are already seriously ill, the symptoms of stenosis are often the first to point to a severe disease of the larynx. On laryngoscopic examination, besides the general redness and swelling in particular places, we can sometimes recognize a circumscribed prominence of the mucous membrane caused by the abscess. If the abscess be already broken we can see the abscess cavity and sometimes the cartilage lying free within it. In most cases we find a considerable collateral œdema of the surrounding mucous membrane, which œdema frequently has a greater share in the production of stenosis than has the primary affection itself. The dreaded œdema of the glottis (œdema of the ary-epiglottic ligament) in typhoid, tuberculosis of the larynx, etc., is usually due to perichondritis of the cricoid or arytænoid cartilages. Finally, we can see with the laryngoscope, especially in perichondritis arytænoidea, a considerable disturbance of motion of the affected arytænoid cartilage, and also of the vocal cords.

Laryngeal perichondritis terminates fatally in a great number of cases from the development of stenosis. In thyroid perichondritis the pus may sometimes gravitate, causing a severe purulent inflammation of the mediastinum. In other cases the most threatening symptoms may be averted for a time, but the primary disease, such as tuberculosis, finally comes to an unfavorable termination. In the rare cases in which recovery occurs after primary perichondritis or after the termination of the primary disease, such as typhoid, this recovery is often incomplete, since a chronic stenosis of the larynx remains from the ensuing cicatricial contractions.

The **diagnosis** is usually obscure during the first period of severe symptoms of stenosis, since it is difficult to make a laryngoscopic examination, and it is also not always easy to determine the condition. We are usually justified, however, in making the diagnosis, if in those diseases which we have mentioned, in which we know by experience that a perichondritis quite frequently occurs, the danger of suffocation arises in addition to the other laryngeal symptoms. It is of practical importance to recognize stenosis of the larynx with certainty, for it demands speedy therapeutic interference.

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* True submucous abscesses, the so-called phlegmonous laryngitis, occur only in very rare cases.

Treatment.—In the beginning of the affection we may try to reduce the inflammation by the internal and external application of ice or by leeches. The pain may be alleviated by injections of morphine or painting with cocaine. If stenosis of the larynx occurs, surgical interference is usually necessary, for only in very rare cases do we see the abscess open of itself with a subsidence of the dangerous symptoms. In the majority of cases the patient can be saved from suffocation only by the timely performance of tracheotomy or laryngotomy. The laryngeal abscess has been repeatedly opened internally by laryngologists with favorable results. If a chronic stenosis of the larynx remains after a favorable termination of the disease, either the patient must wear a tracheal canula all his life, or the attempt may be made to dilate the stenosis gradually by the methods referred to in the preceding chapter.

CHAPTER IV

ŒDEMA OF THE GLOTTIS

THE practical importance of the subject demands a brief special description of œdema of the glottis, by which we mean œdema of the entrance of the larynx, especially of the ary-epiglottic ligaments. We have already learned to recognize laryngeal perichondritis as one of its most frequent causes. In less deeply seated inflammations in the larynx and its neighborhood, however, œdema of the glottis may sometimes develop as a dangerous complication, especially in cases of laryngitis occurring in the course of severe acute diseases, such as typhoid, small-pox, or erysipelas, or in inflammations of the larynx arising from severe mechanical or chemical irritation, as from hot steam or corrosive substances, or from wounds of the larynx, or, finally, from foreign bodies in the larynx. The collateral œdema in angina Ludovici, in intense inflammations of the parotid gland, or the tonsils, etc., may in rare cases extend to the ary-epiglottic ligaments. Finally, œdema of the glottis occurs in rare cases as a complication of general œdema of the body, as a result of Bright's disease, disease of the heart, emphysema of the lungs, etc. Œdema of the glottis, without extensive œdema elsewhere, has been repeatedly observed to come on quite suddenly, especially in acute and chronic nephritis.

The chief symptom of œdema of the glottis is dyspnœa, which comes on as a result of the stenosis of the entrance of the larynx, and is sometimes extreme. At first this is chiefly on inspiration, but it soon comes on with expiration also. Respiration, especially inspiration, is accompanied by a loud laryngeal stridor. As a result of the incomplete entrance of the air, the efforts at inspiration cause depression of the root of the neck, the epigastrium, and the sides of the thorax. We see with the laryngoscope, if the examination be successful, an œdematous swelling of the ary-epiglottic ligaments, and often a swelling of the epiglottis and the false vocal cords. Sometimes we succeed in feeling the swollen parts with the finger.

If the dyspnœa reaches a degree which threatens life, an operation is the only thing which can afford relief. Laryngologists attempt to reduce the swelling by long incisions in the œdematous parts. If this does not succeed, tracheotomy must be performed. If the immediate danger to life is thus averted, further treatment should be directed to the disease which has given rise to the œdema.

CHAPTER V

TUBERCULOSIS OF THE LARYNX

(*Laryngeal Phthisis. Consumption of the Larynx*)

Ætiology.—Since tuberculosis of the larynx is in most cases combined with tuberculosis of other organs, especially of the lungs, we must refer to the description of tuberculosis of the lungs for the general ætiology and pathology of the disease. A particular description of the special appearances in laryngeal tuberculosis is, however, justifiable, because tuberculosis may at times begin in the larynx and may remain isolated there, at least for a time; and, furthermore, in many cases of laryngeal tuberculosis, which are evidently combined with pulmonary tuberculosis, the laryngeal symptoms are predominant in the clinical picture of the disease. Many physicians have, wrongly as we think, disputed the fact that tuberculosis can begin in the larynx. Clinical experience not infrequently teaches us that men, who have been apparently in good health, are attacked with hoarseness, the disease being at first thought to be a common laryngitis, but at last, by its course, proving to be tuberculosis. In spite of the most careful examination, there are not to be found at first the slightest physical signs of disease in the lungs, and not till later do the manifest signs of a pulmonary tuberculosis succeed the symptoms of a laryngeal affection. In such cases it seems to us an affectation to claim that there is a primary pulmonary tuberculosis which could not be made out at first. Everything is much more in favor of the opinion that the tubercular poison, the tubercle bacilli, may sometimes, if not frequently, first fix upon the larynx, excite the first symptoms of tuberculosis there, and then attack the lungs.

In the great majority of cases of laryngeal tuberculosis the symptoms are developed, of course, secondarily in the course of chronic pulmonary phthisis. We shall see that in most cases the disease of the larynx is to be considered as the result of an infection of the mucous membrane of the larynx by the tuberculous sputum which passes over it. More rarely the infectious material seems to reach the larynx by way of the lymph- or blood-vessels. In favor of this theory we may mention that men of great experience (Schrötter and others) have stated that, where the laryngeal trouble was particularly marked on one side, that side was very often affected which corresponded to the affected lung. In about one fourth of all cases of pulmonary tuberculosis this complication occurs, if we include all the mild laryngeal cases. Marked and extensive tuberculosis of the larynx is much rarer, however.

Pathological Anatomy.—Tuberculosis of the laryngeal mucous membrane begins, like tuberculosis of other mucous membranes, with the formation of small subepithelial nodules, which soon become cheesy, break down, and form small ulcers. In the further course of the disease there is very often a more extensive tubercular infiltration, which gives rise on the one hand to irregular thickening of the mucous membrane, and on the other to deep ulceration. The tubercular infiltration is most frequently situated in the interarytænoid space, on the false or true cords (at first on one side), and on the epiglottis. The tuberculous ulceration usually develops later in one of these places. The ulceration may extend from the epiglottis to the neighboring parts of the tongue. In severe cases we often find a marked collateral œdema in the surrounding parts accompanying the inflammation, and sometimes the tubercular perichondritis which has already been described.

The rest of the laryngeal mucous membrane (especially the vocal cords) not involved in the specific tubercular process is usually the seat of a simple catarrh.

Clinical Symptoms.—In the beginning of tuberculosis of the larynx the laryngoscope usually shows nothing but the appearances of a simple catarrh, since the primary miliary nodules of tuberculosis are usually hard to make out. If the catarrhal symptoms be only on one side, we should always suspect tuberculosis. In the later stages, however, most of the special details of the destructive tubercular process, described above (ulcers, infiltration, etc.), can be very satisfactorily made out with the laryngoscope. The infiltration and ulcers are either chiefly on one or on both vocal cords or on the posterior wall of the larynx, or mainly in the neighborhood of one arytænoid cartilage, or the whole epiglottis is irregularly ulcerated on its edge. Single tubercular nodules are often plainly to be seen. In severe cases almost the whole upper portion of the larynx appears like an ulcerated surface covered with mucus. In laryngeal tuberculosis, however, the soft palate usually looks very pale and anæmic. The redness and swelling of the affected parts are more distinct in the living subject than in the cadaver; but the autopsy often shows a far greater extension of the tubercular disease than we can determine by the laryngoscope.

The other clinical symptoms of tuberculosis of the larynx vary very much with the extent and intensity of the process. Sometimes they consist merely in moderate roughness and hoarseness of the voice, but in other cases they increase to the most painful condition which is ever seen in any variety of tuberculosis. This is especially apt to be the case if the ulceration involves the epiglottis and the arytænoid cartilages. Swallowing is then extremely painful, so that the nutrition is very often impaired, and painful attacks of coughing frequently occur. A practiced ear can often distinguish at once the hoarse sound of a "laryngeal cough" from the ordinary pulmonary cough. If severe ulcerations attack the vocal cords, and their free mobility is affected to a marked degree, the hoarseness increases, and finally reaches a complete aphonia. Death finally occurs from general inanition, or, exceptionally, from œdema of the glottis.

The **diagnosis of tuberculosis of the larynx** is not difficult if pulmonary phthisis is already known to be present. When attention has been called to it from the onset of hoarseness or from some disturbance in swallowing, we recognize the character and seat of the changes by the aid of the laryngoscope. The diagnosis, however, may present much difficulty in cases where we are not sure that an affection of the lungs co-exists. As has been said, the symptoms at first are not unlike those of a simple catarrh, and the suspicion of the existence of tuberculosis is first aroused from the stubbornness of the disease, the condition of the patient, the knowledge of some inherited taint, the onset of fever, and the remarkable anæmia and emaciation. With the changes in the larynx which have been described, without evidence of pulmonary tuberculosis, the distinction between tuberculosis and syphilis may be very difficult. In syphilis of the larynx, however, we find that co-existing changes in the pharynx are much commoner than in tuberculosis, and the cicatricial formation which is usually visible furnishes, besides, a very characteristic evidence of syphilis. The diagnosis of tuberculosis of the larynx, however, is made perfectly certain in all doubtful cases by the discovery of tubercle bacilli in the patient's expectoration or in the secretion from the ulcer, which often can be easily obtained by the aid of a fine laryngeal brush. In regard to the laryngoscopic appearances, we may also say that a thickening and infiltration of the epiglottis with a partial ulceration of the same is an appearance which is almost exclusively confined to tuberculosis. The same is true with regard to a marked projecting infiltration of the inter-arytænoid region. In doubtful cases it may aid the diagnosis to inject tuberculine (see the chapter on pulmonary tuberculosis).

Treatment.—For the constitutional treatment of tuberculosis of the larynx the reader is referred to the description of pulmonary tuberculosis. We shall here discuss merely the local treatment. This is in the milder forms the same as for simple laryngeal catarrh. There is no doubt that even genuine tuberculous ulceration of the larynx may be healed. Nevertheless permanent cures of this sort are exceptional. Of course very much depends upon the co-existing state of the lungs. For local treatment insufflations of iodoform and iodol were for a time strongly recommended, but they are now for the most part abandoned. Cauterizing the tuberculous ulcers with nitrate of silver does more harm than good. We sometimes see better results from blowing powdered boracic acid on the ulcers or from a twenty-per-cent. solution of menthol in oil. At present lactic acid is most recommended—cauterizing the ulcers with a solution of increasing strength (twenty to eighty per cent.), at first daily, and later, after the ulcers have been cleaned off, less frequently.

If we were to give a general estimate of the local treatment of laryngeal tuberculosis we should have to admit that its success has not been great. We do not deny that skill and perseverance may be of much service, but only too often do we become discouraged by the failure of the most careful local treatment, and in many cases cauterization of the larynx is only a useless distress to the patient.

With incipient disease there may be considerable improvement and even recovery without any local treatment if the larynx be spared any exertion and given complete rest and if, at the same time, careful general treatment be instituted. The most we need to use is the atomizer or nebulizer with simple salt solution, alkaline waters, etc. We have also found inhalations of a weak carbolic solution, and especially of vapor of Peruvian balsam, of service. In advanced cases, where pronounced pulmonary tuberculosis co-exists, we must content ourselves with a purely palliative treatment. The constant use of cracked ice, and especially a lavish employment of narcotics, are of most service in relieving the pain and the difficulty in swallowing. Subcutaneous injections of morphine a quarter of an hour before a meal often afford great relief. We have seen very good results from cocaine. If we paint the ulcerated mucous membrane at the entrance of the larynx with a ten- or twenty-per-cent. solution of cocaine, in a few minutes such an anæsthesia of the affected parts ensues that swallowing may take place without any pain. The following formula may be used:

℞ Cocainæ muriatis	gr. xv–xxx,	gramme	1.0–2.0;	
Alcohol	ʒss	"	2.0;	
Aquæ destillatæ	ʒij	"	8.0.	M.

Unfortunately the action of cocaine is very transitory, so that the painting must be frequently repeated. With a severe laryngeal cough and irritation relief may also be obtained by inhalations of a two- to three-per-cent. solution of bromide of potassium or a three- to ten-per-cent. solution of bitter-almond water.

If a pronounced stenosis of the larynx develop, tracheotomy is indicated. Many recent observations seem to show that tracheotomy may also be of service in marked laryngeal tuberculosis. When the laryngeal respiration is shut off the tubercular lesions seem to improve faster.

In cases of pronounced but comparatively limited laryngeal tuberculosis, without much implication of the lungs, the attempt has been made, after a preliminary laryngotomy, to remove the diseased parts as completely as possible by surgical means. We still lack extended practical experience in this matter, but further trials of the operation seem very desirable.

CHAPTER VI

PARALYSES OF THE LARYNGEAL MUSCLES

1. **Paralyses in the Distribution of the Superior Laryngeal Nerve.**—The superior laryngeal nerve, arising from the vagus, is the sensory nerve for the mucous membrane of the upper portion of the larynx down to the glottis, and also for the mucous membrane of the epiglottis and its neighborhood. Besides this, it also supplies motor fibers to the crico-thyroid muscle, probably from the accessory nerve. Clinical experience renders it probable that the superior laryngeal nerve also supplies the depressors of the epiglottis, the thyro-epiglottideus, and the arytæno-epiglottidei muscles, and perhaps also the arytænoideus muscle. The last three muscles mentioned, however, perhaps derive some motor fibers from the recurrent nerve also (the inferior laryngeal nerve).

Paralysis of the crico-thyroid muscles and of the depressors of the epiglottis is seen most frequently after recovery from diphtheria. It is usually a part of a more extensive paralysis, and, in addition, is sometimes associated with anæsthesia of those portions of the mucous membrane which, as we have seen, derive their sensory fibers from the superior laryngeal nerve (von Ziemssen). Choking is therefore easy and there is danger of inhalation pneumonia.

Paralysis of the thyro-epiglottideus and the arytæno-epiglottidei muscles is recognized by the immobility and the erect position of the epiglottis, which is directed toward the back of the tongue.

Paralysis of the crico-thyroid muscles makes the voice rough, and especially renders the production of high tones impossible, since for this purpose we need the action of this muscle as a tensor of the vocal cords by approximating the cricoid and thyroid cartilages. The detection of this paralysis by the laryngoscope is extremely difficult. Its chief signs are a concavity of the edges of the vocal cords, a lack of visible vibration in them, and perhaps, in unilateral paralysis, a higher position of the vocal cord on the sound side.

For paralysis of the arytænoideus muscle, *vide infra*.

2. **Paralyses in the Distribution of the Inferior Laryngeal or Recurrent Nerve.**—The recurrent nerve supplies with sensory fibers the mucous membrane of the inferior cavity of the larynx below the glottis, and it is the motor nerve for all the laryngeal muscles except the crico-thyroid, and except possibly the depressors of the epiglottis (*vide supra*). The muscles innervated by it are arranged according to their function in the three following groups:

a. The openers of the glottis—the posterior crico-arytænoid muscles.

b. The closers of the glottis—the lateral crico-arytænoids and the arytænoideus (transverse and oblique).

c. The tensors of the vocal cords—the thyro-arytænoids, which act usually as closers of the glottis, but which very often produce the fine differences in tension in the vocal cords which are necessary in singing and in modulations of speech. They accordingly have the same task as the coarser-working crico-thyroid muscles, which are innervated by the superior laryngeal nerve.

The motor fibers for all these muscles have their special origin in the accessory nerve, from which they pass into the trunk of the vagus, and from this into the laryngeal nerves.

Most of the paralyses of the recurrent nerve are of peripheral origin. Except in the pure muscular pareses (*vide supra*), which arise not infrequently in the course of other laryngeal affections, peripheral paralyses of the vocal cords occur with the greatest relative frequency from an abnormal pressure on the trunk of the recurrent nerve, especially in aneurism of the arch of the aorta, which may cause a left-sided paralysis. Tumors of the bronchial glands, cancer of the œsophagus, thyroid or mediastinal tumors, and, in rare cases, even large pericardial effusions, may also cause a paralysis of the recurrent on one side. Paralyses on the right side are seen quite frequently in contractions at the apex of the right lung and in the rare cases of aneurism of the subclavian artery. The paralyses of the laryngeal muscles, which are sometimes met with after recovery from diphtheria (*q. v.*), also belong to the peripheral paralyses of the recurrent nerve, and their cause is to be found in a degeneration of the branches of the affected nerves. In other cases the paralysis of the recurrent nerve is due to an affection of its fibers in the vagus or even in the accessorius. Excluding certain injuries from operations, these affections are usually due to new growths which cause a paralysis of conduction. Paralyses of the recurrent nerve also arise from affections of the nucleus of the accessory nerve in diseases of the medulla, in the different forms of acute bulbar paralysis, in chronic bulbar paralysis, in multiple sclerosis, etc. The frequent hysterical paralyses affecting the closers of the glottis and the tensors of the vocal cords are to be regarded as central disturbances of innervation (see the chapter on hysteria).

1. **Complete Paralysis of the Recurrent Nerve.**—Paralysis of all the laryngeal muscles supplied by the recurrent nerve occurs quite frequently in the pressure paralysis of the trunk of the recurrent, or of its fibers in the vagus. With the laryngoscope (see Fig. 16) we find the vocal cord on the paralyzed side in a middle position, often falsely called a "cadaveric position," and completely motionless on respiration, and also on phonation. The arytænoid cartilage on the paralyzed side is often turned inward. On phonating as strongly as possible, the vocal cord on the sound side passes beyond the median line, the arytænoid cartilage also crosses the line, and consequently the glottis is put in an oblique position. The other symptoms are sometimes so slight that without a laryngoscopic examination we do not even think of a paralysis. The voice, however, is usually not pure; it often breaks into a falsetto, and the patient is easily tired by speaking. With complete bilateral paralysis of the recurrent nerve, which is very rare, we find both vocal cords motionless in a middle position. Complete aphonia exists, and it is impossible to cough, since in coughing we have to make at first a complete closure of the glottis. There is no dyspnœa, however, if the patient keeps quiet.

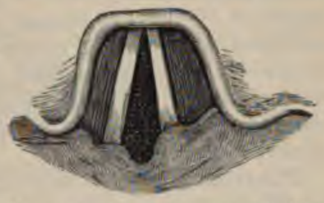


Fig. 16.—(From Ziemssen.) Position on inspiration in paralysis of the left vocal cord, or paralysis of conduction in the recurrent nerve.

It is an interesting fact, first reported by Rosenbach, that in incomplete recurrent paralysis almost invariably the abductors of the vocal cords (the openers of the glottis) are alone paralyzed, whence the cords remain in adduction. Only with the further advance of the disease does complete recurrent paralysis occur; then the adductors also become paralyzed, and the vocal cords become completely motionless, and assume the middle, so-called cadaveric position.

2. **Paralysis of the Dilators of the Glottis, the Posterior Crico-arytænoid Muscles.**—Bilateral paralysis of these muscles is quite a rare phenomenon, but clinically it is of the utmost importance, since it results in a condition of most marked inspiratory dyspnœa. Neuritic changes, central diseases (tabes, multi-

ple sclerosis, etc.), cancer of the œsophagus, etc., may lead to paralysis of the posterior muscles. In many cases the cause is obscure. The sequelæ of the paralysis usually develop slowly and gradually. The affection may last for years. The greatest impairment of breathing probably occurs when the vocal cords are fixed in adduction by the contracture of the antagonistic closers of the glottis. The dyspnœa may increase, especially from external causes, to severe attacks of suffocation, and tracheotomy is frequently necessary. The respiration is affected in such a way that inspiration only is difficult, protracted, and noisy, while expiration is free and unhindered. This depends on a valve-like action of the vocal cords. They are drawn together by the dilatation of the thorax on inspiration, while the current of air in expiration easily pushes them aside. Phonation is usually entirely undisturbed. With the laryngoscope (see Fig. 17) we find the glottis changed to a small slit, which grows narrower instead of wider on inspiration. The prognosis is usually unfavorable. Only in the hysterical can these apparently severe conditions appear and disappear again in a short time.

In unilateral paralysis of the posterior crico-arytænoid muscle there is, as a rule, no marked dyspnœa. The voice is somewhat hoarse, and with the laryngoscope we can see that the paralyzed vocal cord does not deviate outward on inspiration.

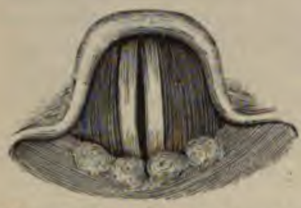


FIG. 17.—(From ZIEMSSEN.) Complete bilateral paralysis of the posticus at the moment of inspiration.

3. **Paralysis of the Thyro-arytænoid Muscles.**—The paralysis or paresis of these muscles, which run into the vocal cords, and which are their chief tensors, is one of the most frequent of the paralyses of the laryngeal muscles. It occurs especially in acute and chronic catarrh of the laryngeal mucous membrane, and is often the chief cause of the accompanying hoarseness. It also frequently develops as the result of an habitual strain of the voice in singers and public speakers, and it is one of the commonest causes of hysterical aphonia.

Paralysis of the thyro-arytænoid muscles may be bilateral or unilateral. It is frequently associated with a paresis of the other closers of the glottis, the arytænoidei and the crico-thyroid muscles. With the laryngoscope (see Fig. 18), in the ordinary bilateral paresis of the thyro-arytænoid muscles, we see that on phonation the glottis does not close completely, but that an oval space is left between the vocal cords.

In unilateral paralysis the affected cord shows a concavity of its edge. The voice is always more or less hoarse and low and the speech is strained.

In many cases, after a cure of the original catarrh, a complete recovery from the paralysis may follow by taking good care of the voice. Hysterical paralyses are diagnosticated by their sudden disappearance and reappearance, usually after some psychical disturbance. They are quite common in children of the age of ten to fourteen years, especially in girls. (See chapter on hysteria.)

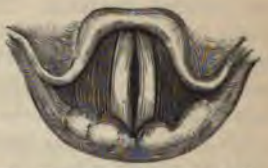


FIG. 18.—(From ZIEMSSEN.) Paralysis of both internal thyro-arytænoid muscles in the course of an acute laryngitis.

4. **Paralysis of the arytænoideus muscle** is rarely an isolated phenomenon. It is sometimes seen in laryngeal catarrh or in hysterical aphonia. The voice is quite hoarse, and with the laryngoscope (see Fig. 19) we find on phonation that the whole anterior part of the vocal cords closes well, but that the cartilaginous glottis remains open as a triangular gap on account of the imperfect motion of the arytænoid cartilages toward each other. When the thyro-arytænoids are para-

lyzed with the arytænoideus, the glottis shows on phonation a narrow hour-glass opening (see Fig. 20). Both the anterior and the posterior portions of the glottis fail to close, while the vocal processes take their usual median position on phonation from the normal turning of the arytænoid cartilages inward by the action of the lateral crico-arytænoid muscles.

5. **Paralysis of the lateral crico-arytænoid muscles,** as an uncomplicated condition, has never been observed with certainty. Some cases of a complete and simultaneous paralysis of all the closers of the glottis have been described, however, in which the vocal cords are immovable laterally and the glottis remains abnormally wide open.

We may expect success from the **treatment** of paralysis of the vocal cords only when the primary disease is capable of cure. If catarrhal or other diseases of the larynx co-exist, we must first treat these by the methods already mentioned.

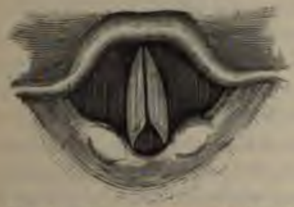


FIG. 19.—(From ZIEMSSEN.) Paralysis of the arytænoideus in acute laryngitis.

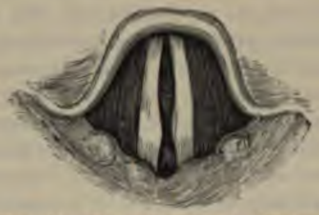


FIG. 20.—(From ZIEMSSEN.) Bilateral paralysis of the thyro-arytænoids combined with paresis of the arytænoideus.

Paralysis from the compression of tumors, etc., may be relieved in rare cases by extirpation, or by partial resolution of the tumors when of strumous origin. In catarrhal, diphtheritic, and the so-called "rheumatic" pareses—that is, those which occur without any assignable cause—and also in all hysterical aphonias, electricity often works very well. Von Ziemssen has made electrodes for the endolaryngeal stimulation of single muscles, but external galvanization is usually sufficient in practice. In hysterical aphonia the chief task is to accustom the patient again to the necessary and proper voluntary innervation. We usually accomplish our purpose most rapidly by commanding the patient to cough and then say "Ah," while we faradize or galvanize the neck. It is often of advantage to reverse the current. If the patient succeeds in saying "Ah" once, the voice usually returns very quickly. Internally we may try subcutaneous injections of strychnine in doses of gr. $\frac{1}{20}$ to gr. $\frac{1}{6}$ daily (grm. 0.003 to 0.01).

CHAPTER VII

SPASM OF THE GLOTTIS

(*Laryngismus Stridulus. Millar's Asthma. Thymic Asthma*)

Ætiology.—Spasm of the glottis is a disease which occurs almost exclusively in children under three years of age, and consists of attacks of spasmodic closure of the glottis, and consequently of most severe dyspnœa. Boys are more frequently attacked by this disease than girls, but the cause of this is wholly unknown. The old name of thymic asthma arose from the idea that the attacks were due to an increase in the size of the thymus gland, but this opinion is wholly unfounded. The relation between spasm of the glottis and rachitis is remarkable, but it is unexplained. Nearly two thirds of all the children who suffer from spasm of the glottis are rachitic, but the opinion which was once held that spasm of the glottis has a special relation to the rachitic craniotabes is not clearly proved. The fact that it is often combined with eclampsia, in that the attacks of spasm of the glottis are aggravated by eclamptic attacks, and that the two alternate with each other, is an argument in favor of a central origin for the disease. In the cases which come on, as they often do, at the time of dentition, we think it possible to assume a reflex origin for the spasm, just as we may in those cases which seem to follow a laryngitis due to taking cold.

Symptomatology.—The single attacks usually come on suddenly by day or by night, either without any cause or from some external influence, such as crying, swallowing fluid, or some psychical disturbance. They usually begin with a deep inspiration, followed by complete cessation of respiration. The child becomes pale, cyanotic, looks anxiously about, rolls his eyes, and makes strained and labored efforts at respiration. In severe cases there is a temporary loss of consciousness, and tonic and clonic spasms in the muscles of the extremities and the trunk, as has been mentioned. The attack lasts from some seconds up to two minutes. In very severe cases the attack may be immediately fatal. As a rule, however, the spasm passes off, deep, noisy inspirations follow, and in a short time the child is completely well. The severity of the attacks varies, moreover, in different cases, and it varies very markedly, too, in the same child. Sometimes we have only one attack or a small number of them, while in other children they may come on ten or twenty times a day, and even oftener, and may last with varying intensity for months. If the child reaches his third year the disease almost always disappears, but quite a large number of the children who suffer from spasm of the glottis die before that age, either in the attack itself or from other affections.

Genuine spasm of the glottis hardly ever occurs in adults, but similar attacks, which, of course, have quite a different significance, are sometimes observed in hysteria.

The **treatment** must be especially directed to the child's general condition. The child is usually pale and emaciated, and if we succeed in improving its nutrition with iron and cod-liver oil, the attacks become less frequent, and milder, and finally may wholly disappear. If rachitis exists, we should first of all try phosphorus (1 part in 10,000 of cod-liver oil). The child should also be kept in moderately warm air and guarded from any exposure to cold. Internal remedies to prevent the recurrence of the attacks are very uncertain in their action. We may employ one or two grains (gramme 0.05–0.10) of chloral hydrate in solution every hour or two; bromide of potassium, ten to thirty grains daily (grammes 0.5–2.0); extract of belladonna, etc.

In the attack itself the child must be raised up. The face should be sprinkled with water, or, if the attack be of long duration, a cool shower-bath should be given. Friction should be applied to the skin, aided by mustard, or a mustard plaster applied to the chest and calves. If the attacks are very frequent and intense, we must use narcotics, either inhalations of chloroform or subcutaneous injections of morphine, with care, in doses for a child of $\frac{1}{60}$ to $\frac{1}{12}$ of a grain (gramme 0.001 to 0.005).

CHAPTER VIII

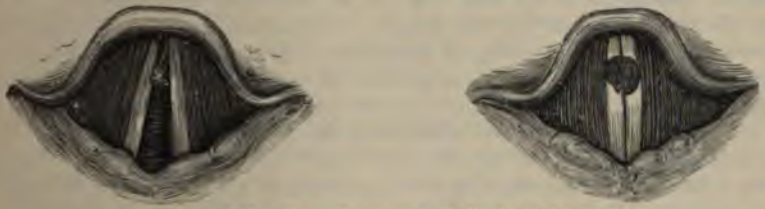
NEW GROWTHS IN THE LARYNX

SINCE new growths in the larynx are of interest mainly to specialists and surgeons, we will here only glance briefly at them. We must remember especially, however, that they can be recognized only by the aid of the laryngoscope. It unfortunately often happens that a patient is treated for a long time without success for a "chronic laryngeal catarrh," when the laryngoscope finally shows that a new growth is the cause of the hoarseness. It is of especial importance, however, to make a diagnosis as early as possible, particularly in carcinoma, since the earlier the operation is done the better is the chance for success (*vide infra*).

A. BENIGNANT GROWTHS IN THE LARYNX

1. Papilloma is one of the commonest new growths in the larynx. It forms glandular, cauliflower-like excrescences, which are usually situated on the anterior part of the vocal cords, rarely on the false cords. The base of the swelling is broad or pediculated. The growths are often multiple. We do not know the special cause of their origin. It is worthy of note that papilloma, like other benignant growths in the larynx, is seen much more frequently in men than in women.

2. Fibroma in the larynx is comparatively common. The pediculated tumors known as "laryngeal polypi" are usually fibromata. They are generally situated on the vocal cords and form whitish or reddish-brown swellings, from the size of a pea to that of a cherry, vascular and dense or soft (see Figs. 21 and 22). There is no evidence that persons who use their voices very much are especially liable to the formation of fibromata.



FIGS. 21 and 22.—(From ZIEMSSEN.) Pediculated fibromata.

3. Cysts and "mucous polypi" rarely occur. They are probably due to the retention of the secretion in a mucous gland from the stoppage of its orifice. We find them in the ventricles of Morgagni, on the epiglottis, etc. In a very few cases we also see in the larynx lipomata, myxomata, separated portions of thyroid gland tissue which have begun to grow, enchondromata, etc.

The symptoms which are excited by benignant tumors in the larynx depend partly upon the situation and partly upon the size of the new growth. Small polypi may exist wholly without symptoms, and they are found only by chance on laryngoscopic investigation. Usually, however, the presence of disturbances of phonation (hoarseness, a marked change in pitch, secondary tones), persistent cough, or respiratory disturbances when the tumor is a large one, are the symptoms which give occasion for an examination. Abnormal sensations in the larynx are by no means the rule, and pain is wholly absent.

B. MALIGNANT GROWTHS. CARCINOMA OF THE LARYNX

Sarcomata, arising from the true or false vocal cords, are very rare. The only malignant new growth of the larynx of great practical importance is carcinoma.

Carcinomata develop usually in old people, either primarily in the larynx or secondarily from affection of the neighboring organs. In the first case the vocal cords or the ventricles of Morgagni are the points most frequently attacked. An extension of the disease to the larynx is seen especially in cancer of the tongue or pharynx, rarely in cancer of the œsophagus.

The symptoms of cancer of the larynx develop slowly. Hoarseness, dysphagia, pains in the larynx often shooting up into one ear or the temporal region, the appearance of respiratory symptoms, and finally, but only in the later stages as a rule, the signs of general weakness and emaciation which are seen in almost all forms of carcinoma, form the picture of the disease. Enlargement of the cervical lymph-glands is sometimes an early symptom, but it may be wholly lacking. The diagnosis is possible only by the aid of the laryngoscope. Besides this, a digital examination may at times be of diagnostic value by the detection of the characteristic hardness about the entrance or in the neighborhood of the larynx. A general description of the laryngoscopic appearances can not be given on account of the diverse character of the cases. We see the uneven, injected new growth, covered with mucus and often ulcerated, and besides this at times the secondary appearances of catarrh, a developing perichondritis, etc. In many instances the diagnosis is quite easy, but in other cases protracted observation is required. It may be difficult, however, at times, to distinguish it from tuberculosis or from syphilis. We may be aided in such cases by a search for tubercle bacilli, by tuberculine injections, or by the results of anti-syphilitic treatment. All the other organs of the patient therefore must always be carefully examined. In doubful cases it is proper to remove a bit of the endolaryngeal growth and examine it histologically.

Surgical **treatment** is the only one for all laryngeal new growths. We must refer to the special works for the details. Laryngologists have devised numerous instruments for the removal of benignant polypi, by which, under the guidance of the laryngoscope, the new growth is cut, snared, crushed, or torn off. The performance of the operation is made much easier by inducing local anæsthesia of the laryngeal mucous membrane by painting with cocaine. Nevertheless we firmly believe that in all severe cases the "endolaryngeal" operations should be superseded by laryngotomy.—Carcinoma of the larynx can be cured only through removal of the tumor by laryngotomy (splitting of the larynx) or by total extirpation of the larynx. Laryngotomy is comparatively free from danger, while total extirpation has met with permanent success as yet in but few instances. If surgical interference is no longer practicable, we can endeavor merely to mitigate the suffering of the patient by means of morphine, cocaine, and other narcotics.

SECTION III

DISEASES OF THE TRACHEA AND THE BRONCHI

CHAPTER I

ACUTE CATARRH OF THE TRACHEA AND THE BRONCHI

(Tracheitis and Acute Catarrhal Bronchitis)

Ætiology.—Acute catarrh of the larger air-passages, of the trachea, and larger bronchi, is a frequent disease, and it may often arise from taking cold. It is conceivable that the inhalation of cold, damp air sometimes directly affects the mucous membrane of the upper air-passages. Bronchial catarrh is very often associated with a coincident catarrh of the larynx, and more rarely of the pharynx. In the ordinary mild forms the catarrh is usually confined to the trachea and the first large branches of the bronchi, while the finer bronchi remain healthy.

More intense inflammation of the bronchial mucous membrane is the result of active mechanical or chemical irritation. A severe bronchitis develops after the inhalation of noxious gases, nitrous and sulphurous oxides, chlorine, bromine, etc., as is often observed in operatives. The inhalation of smoke and dust, especially vegetable dust, works in the same injurious fashion, and the followers of many trades and employments, such as millers, wool-workers, colliers, etc., are especially subject to disease from this cause. In this form of bronchitis the catarrh often extends to the finer bronchi.

The bronchitis which develops in the course of other acute and chronic diseases is still commoner than the primary forms already mentioned. It is often due to infectious causes, such as certain infectious diseases, especially measles, whooping-cough, and influenza. In these diseases bronchitis is one of the most constant local affections, and is probably dependent immediately upon the primary infection. Bronchitis, however, develops secondarily in most of the other acute infectious diseases, and is due largely to the inhalation of noxious substances from the upper part of the air-passages. This is the explanation of the bronchitis in diphtheritic processes in the pharynx and larynx, in so far as it does not depend upon a direct extension of the disease, and also of the bronchitis in small-pox, etc. Bronchitis may also be met with in all other forms of severe disease, because retention of secretion, putrefactive processes, inflammation, thrush, etc., occur in the cavity of the mouth and pharynx, and from them chemical or organic irritants may easily be inhaled into the bronchi. The imperfect expectoration in all severe diseases is a still more harmful factor than this inhalation. The secretion remains in the bronchi, processes of decomposition arise in the stagnating mucus, bacteria collect and lead to a bronchitis, and finally to the lobular pneumonia which is so often found (*vide infra*). The lessened power of resisting injurious influences, produced in the mucous membrane by severe bodily disease, also facilitates the development of catarrhal inflammation. In all diseases in which deglutition is affected (as in paralysis of the epiglottis or of the pharyngeal muscles), and in all diseases attended by frequent vomiting or choking (cancer of the œsophagus, etc.), secondary bronchitis with its sequelæ may ensue from the inhalation of small particles of easily decomposable food.

We do not know how far we may claim that infectious agents act as a cause of primary bronchitis, yet it is not improbable that many cases have such an ætiology. It is especially likely that many cases of bronchitis due to "catching cold" really have something infectious about them, and that the preceding exposure to cold has merely lowered the natural powers of resistance, and thus permitted, or at any rate promoted, infection. This infection is probably not due to any special specific bacteria but to the ordinary germs of inflammation (staphylococci, etc.).

Finally, we must mention that acute bronchitis is sometimes merely an exacerbation of a previous chronic bronchitis. We will return later to this important and not uncommon process.

The predisposition to acute bronchitis varies in different persons. We do not know definitely on what ground such an increased predisposition to bronchial disease rests, nor why we meet with it sometimes in the weak and anæmic, and at other times in so-called "full-blooded" persons. Bronchitis is more frequent in children and old people than in those in middle life. Most of the cases occur in the spring and autumn.

Symptoms.—Pain in the chest may be present in some cases of simple catarrhal bronchitis, but usually only in a moderate degree. In severe tracheitis patients often have a painful feeling of soreness in the neck and behind the upper part of the sternum, and this is increased on coughing. The mucous membrane of the bronchi, apparently, has no nerve-fibers which are sensitive to pain, and the pains in the chest which are often present in bronchitis are, as a rule, muscular pains in the intercostal muscles, or the insertion of the diaphragm, due to the severe paroxysms of coughing.

Cough is one of the most constant symptoms of bronchitis, and by it usually the attention of the patient or of the physician is first called to the existing thoracic affection. The cough may of course be due to a laryngitis, if that is also present. There is no doubt, however, that a cough may be excited in a reflex manner from the mucous membrane of the trachea and of the larger as well as of the finer bronchi. Experiments have shown that the point of bifurcation of the trachea is especially irritable, and many severe paroxysms of coughing may be due to an irritation of this very spot from the accumulation of secretion. The intensity of the cough, moreover, is very different in individual cases, which is due in part to the degree and extent of the bronchitis and in part to the individual irritability of the person affected.

The expectoration consists of the secretion and exudation from the inflamed mucous membrane. Its abundance and consistency vary considerably in the different cases. We distinguish a catarrh with an abundant secretion from the so-called " dry catarrh." In the latter only a little viscid sputum is expectorated, but in the former the expectoration is more abundant and muco-purulent, or sero-mucous, a thin fluid separating into layers on standing (*vide infra*). Very often in the beginning of the disease the expectoration is scanty and viscid—the *sputum crudum* of the old physicians; and later it becomes more abundant, more fluid, and more purulent—the *sputum coctum*. In catarrh of the finer bronchi the expectoration may contain little tough mucous or muco-purulent casts of the bronchi. It may be stated in general that the characteristic of simple bronchitic sputum is the mucous admixture in contrast to the purulent or sero-purulent sputum from phthisical cavities, etc. We may readily recognize the mucous contents of the sputum by the tenacity with which it clings to the bottom of the sputum-cup when the cup is tipped. A simple catarrhal expectoration shows nothing peculiar under the microscope. The pus-corpuscles are often swollen, and show more or less marked fatty degeneration. A slight admixture of blood may occasionally be present in severe bronchitis, but it usually has no special significance, being at times merely the result of severe fits of coughing. A more marked and persistent admixture of blood is seen in the catarrhal sputum of some cases of intense bronchitis in drunkards, so that we may even speak of a " hæmorrhagic bronchitis."

Dyspnœa is usually entirely absent in simple mild bronchitis, but marked shortness of breath may be noticed in extensive catarrh of the finer bronchi.

Physical Examination.—We may obtain direct evidence of the condition of the tracheal mucous membrane, with due practice, by the laryngoscope. We see a reddening of the membrane, and sometimes an abnormal abundance of secretion on it, if there is a tracheitis. Other methods of physical examination are at our service for judging of the changes in the bronchi.

Inspection of the thorax shows nothing abnormal in the milder forms of bronchitis. The respiration is somewhat accelerated and the expiration prolonged in severe bronchitis, especially if the finer bronchi are affected. Percussion in uncomplicated bronchitis shows nothing abnormal in the pulmonary resonance. In extensive catarrh of the finer bronchi an acute dilatation of the lung is apt to occur, with a descent of the lower margin of the lung. Auscultation, too, shows nothing unusual in many cases of mild catarrh limited to the trachea and large bronchi, but in the cases in which the smaller bronchi are the seat of the catarrh and there is a marked accumulation of secretion in them, we hear, besides the vesicular respiration, the so-called rhonchi which almost wholly hide it. In dry bronchitis we speak of humming or buzzing sounds (sonorous rhonchi) or shrill, whistling sounds (sibilant rhonchi) according to their pitch. These sounds are probably due to stenosis, and are caused by the passage of the air through narrow portions of the bronchi. The narrowing occurs in part from the swelling of the mucous membrane, in part from the accumulation of secretion. The masses of secretion themselves, if they are set in vibration at the same time, may possibly take part in the production of the humming noises. If the amount of secretion collected in the bronchi is more abundant and of a more fluid consistency, it gives rise to "moist râles" on the passage of the air. These are distinguished as "coarse," "medium," or "fine moist râles," according as they occur in the larger or smaller bronchi.

Other symptoms of disease are often present besides those already mentioned as being due directly to the bronchitis. The general health is usually disturbed in a severe bronchial catarrh. The patient does not feel well, and has less appetite than usual. A moderate amount of fever is often present, especially toward evening. An increase of temperature above 102° or 103° (39° C.) is rarely seen except in children. The patient sometimes complains of headache, which is increased by severe coughing. There can be a question of complications only when the inflammation extends to other parts of the respiratory mucous membrane. Mild bronchitis is often associated with coryza and laryngitis, while severe bronchitis may lead to catarrhal pneumonia (*vide infra*).

The separate forms of bronchitis are distinguished chiefly by the degree of extension of the catarrh.

1. **The Milder Forms of Acute Bronchitis.**—In most cases of simple primary bronchitis, as well as in many milder attacks of secondary bronchitis, the catarrh is limited to the mucous membrane of the larger bronchi. Exposure to cold and other injurious influences are frequent causes of the primary form. The symptoms are moderate. The cough, however, may be quite troublesome. Often fever is absent or but slight. Upon auscultation, particularly over the lower lobes, but sometimes over the entire lungs, are heard numerous rather coarse wheezing or rattling sounds, usually with some symmetry of distribution; but in many cases, as we have said, there may be nothing abnormal heard, so that the diagnosis will have to rest merely upon the subjective discomfort in the chest, the cough, and the expectoration. With proper care, simple primary bronchitis runs its course in a few days, or at the most in a few weeks, and ends in complete recovery. If the patient exposes himself recklessly, or the ætiological factors continue to be active, the disease may, however, grow worse, prove to be very tedious, and finally develop into chronic bronchitis.

2. **The Severer Febrile Forms of Acute Bronchitis.**—Sometimes acute bronchitis assumes a severer form, whether because the influences which give rise to it are unusually violent, or because it is due to some special and perhaps infectious cause. In such cases the symptoms are more marked, the bronchial râles more abundant, the general condition of the patient worse. Not infrequently there is fever for several days, or even for one or two weeks, the type being irregularly remittent, but the temperature is usually 102° or 103° (39° to 39.5° C.), although it may go higher. The expectoration is usually muco-purulent, but it often contains much pus; in other cases it may be sero-muco-purulent. The amount is then greater, and it forms distinct layers on standing in a beaker glass. The author has not infrequently noticed that there is more liability in the severer forms of acute bronchitis to have the disease mainly limited to one lobe, or at least to one lung, but we may have a severe acute diffuse bronchitis on both sides.

The ætiology of acute febrile bronchitis has been but little studied. Bacteriological examination of the sputum usually gives no definite results, since we always find many micro-organisms. Many acute cases may be caused by the ordinary pus cocci. At the time of a pneumonia epidemic we have repeatedly seen cases of acute bronchitis with high fever and herpes, but without any signs of pneumonic infiltration. These were probably due to pneumococcus infection. In other cases there is perhaps an influenza infection.

We would add in particular that many acute febrile cases of bronchitis prove on closer investigation (more careful questioning) to be acute exacerbations of a mild chronic bronchitis—"acute recurrent bronchitis." The chronic disease of the bronchial mucous membrane apparently affords a favorable opportunity for new infection. In other instances it is merely an acute exacerbation of the same chronic process. Such an acute bronchitis may of course occur repeatedly in the same patient.

3. **Catarrh of the Finer Bronchi—Capillary Bronchitis.**—A simple primary bronchial catarrh rarely extends to the finest bronchi in adults. The secondary bronchitis, however, which develops in other severe diseases (*vide supra*) often extends into the ultimate divisions of the bronchi, and finally leads to the formation of nodules of lobular pneumonia—"catarrhal pneumonia" (*vide infra*). We recognize the implication of the finer bronchi by hearing the high, shrill, whistling rhonchi (sibilant rhonchi), or the abundant fine, moist râles. Respiratory symptoms may be quite marked in extensive catarrh of the finer bronchi. Respiration is evidently accelerated, the inspiration is of the costal type and requires the aid of the accessory respiratory muscles of the neck (the sternomastoids and scaleni), and expiration is usually prolonged. There is often quite a severe cough. The expectoration is muco-purulent and usually not very abundant.

Capillary bronchitis in children is of great practical importance. Bronchitis in young children always has, as experience tells us, a tendency to attack the smaller bronchi. Extensive bronchitis is seen especially in weak children who are rachitic or predisposed to tuberculosis. Children have a great tendency to be attacked with bronchitis at the time of the first dentition.

The parents' attention is usually called to the disease by the appearance of a cough, which is excited especially by the child's crying. Small children never expectorate, for they swallow the secretion which is coughed up into the pharynx. The rapidity of respiration is very striking, it being increased to sixty or eighty, or even more, in a minute. The respiration is also labored, but it is usually superficial, and in severe cases irregular. There is generally a distinct respiratory play of the alæ nasi. We often see a retraction of the lower lateral portions of the thorax on inspiration as a result of the imperfect entrance of air into the smaller bronchi. The expiration is frequently noisy and groaning in children. We hear extensive fine, moist râles over the lungs. In severe cases the child becomes restless, anxious, perhaps markedly pale and cyanotic, and finally apathetic and stupid. In such cases, however, we have no longer to deal with simple bronchitis, but catarrhal pneumonia has already developed. The disease almost always runs its course with fever, the temperature rising to 104° (40° C.) and over. The pulse is increased to 120 or 140 or more per minute. The duration of the disease is seldom less than two or three weeks, and it may last much longer. Death may ensue, especially in ill-nourished children, partly as a result of general weakness, and also directly from the imperfect respiration. In such cases we find at the autopsy not only diffuse bronchitis, but also almost always lobular pneumonia. In many cases a gradual recovery finally takes place in spite of the most severe symptoms.

The secondary bronchitis in children complicating measles, whooping-cough, diphtheria, etc., has the same tendency to involve the finer bronchi and to lead to lobular pneumonia.

In conclusion, we must mention that acute bronchitis in old persons is also apt to attack the finer bronchi, and it may be dangerous partly from the general exhaustion, partly from the occurrence of respiratory symptoms and cardiac weakness, as in lobular pneumonia.

Diagnosis.—The diagnosis of bronchitis presents no special difficulty. It is obtained directly by the discovery of rhonchi on auscultation. If these fail, we conclude that there is a mild catarrh of the larger bronchi from the presence of cough and expectoration, if no cause for the cough is to be found in an affection of the larynx. The question is more difficult, but it must always be considered, whether a given bronchitis is a common primary catarrh or secondary to some other affection. This question naturally can be decided only by a very careful examination of the body. We must always remember, furthermore, that severe pulmonary affections may be at first quite latent and show objectively merely the signs of simple bronchitis, while later pneumonia, a tubercular affection, or something similar, develops. A bronchitis which is unilateral, or in which the signs are to be found in circumscribed localities, must therefore be regarded as suspicious. It has long been known that bronchitis in the apices of the lungs, the "apex-catarrh," is often the first objective change to be met with in pulmonary phthisis. We can only conjecture, and not pronounce with certainty on objective evidence, whether nodules of lobular pneumonia are present or not in diffuse bronchitis affecting the finer bronchi.

From what has been said, it is clear that we should be cautious in our prognosis regarding cases of severe bronchitis, especially in children and old people. The prognosis in the milder forms of bronchitis is of course always very favorable.

Treatment.—The prophylaxis of primary bronchial catarrh consists in the removal of all the injurious influences mentioned which, as experience shows, may give rise to bronchitis. Careful hardening of the skin to the effects of a change in temperature is of service in persons, particularly children, who have a special tendency to bronchitis, as we have already said in regard to the prophylaxis of laryngitis. It is very important to remember, in this connection, that we can also be successful in our prophylactic measures against secondary bronchitis. Keeping the mouth and pharynx clean, urging deep inspirations, and aiding expectoration by the timely use of tepid baths and shower-baths, may often prevent bronchitis or keep it within bounds, when it would surely develop if the patient were neglected.

Simple hygienic measures suffice in the treatment of mild cases of acute bronchitis. The patient should be kept warm, should remain in his room, or, if there be any fever, in bed. Diaphoretic remedies have long been praised as especially potent in the treatment of acute bronchial catarrh. The patient, therefore, should drink hot tea, pectoral tea * (Brustthee), or elder tea, etc., or hot milk mixed with Seltzer, a remedy whose efficacy is frequently praised by the patient. The tougher the sputum and the more difficult expectoration, the more abundantly should we give warm drinks (Ems water, pectoral tea, etc.). Local treatment of the mucous membrane by inhalations is usually illusory, for only the smallest part of the inhaled fluid reaches the bronchi. We may, however, prescribe inhalations of warm steam, or a one- or two-per-cent. solution of common salt, especially with a dry cough and a secretion which is hard to loosen.

Otherwise we must employ symptomatic treatment. A mustard plaster or a cold, wet compress about the chest does good service with severe subjective thoracic symptoms. In severe cases a few dry cups may be very useful in adults, but local abstractions of blood are never necessary in simple bronchitis. If there is troublesome irritation on coughing, so as to disturb the rest at night, we may prescribe small doses of morphine, five to ten grains of Dover's powder (gramme 0.3–0.5), fifteen to twenty drops of cherry-laurel water, codeine, etc. When expectoration is difficult, we may use the so-called expectorants—ipecac, chloride of ammonium, apomorphine, senega, etc.

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* A favorite German household remedy, consisting of an infusion of eight parts of althæa, three parts of licorice, one part of orris-root, four parts of colt's-foot, and two parts each of mullein and aniseseed.—Trans.

We have already repeatedly mentioned the use of tepid baths and shower-baths, indicated in severe diffuse bronchitis developing secondarily in the course of other acute diseases.

Tepid baths with shower-baths, two or three times a day, are also to be used as a most powerful remedy in severe cases of capillary bronchitis in children. The baths assist expectoration and guard against the possibility of the development of lobular pneumonia. Wet packs applied to the thorax or over the whole body are serviceable. The child should be wrapped to the neck in a sheet which has been previously dipped in water at a temperature varying with the degree of the fever from 68° to 77° (16° to 20° R.) and well wrung out. It is best to leave the arms free. A dry woolen blanket may be wrapped around the moist sheet. This procedure must be repeated three or four times a day. As to other remedies, we use the same as in adults. With weak children our care must be to keep up the strength by giving the most nourishing food possible and small amounts of wine. An emetic is sometimes indicated in cases with an abundant accumulation of mucus in the bronchi, and is of good service. As experience has shown, we should use opiates for small children only with the greatest caution. Senega and benzoin may be employed as expectorants.

In the bronchitis of old people our chief aim should be to keep up and improve the patient's strength. We prescribe liquor ammonii anisatus, infusion of senega, etc., to aid expectoration, which is usually difficult, since the cough is feeble. We must keep close watch on the condition of the heart (digitalis). Tepid baths may be of advantage, but they must be used with care.

CHAPTER II

CHRONIC BRONCHITIS

(*Chronic Bronchial Catarrh*)

Ætiology.—Chronic bronchial catarrh may develop gradually from external causes, or in rare cases it may follow acute bronchitis. The same noxious influences which excite acute bronchitis may, by the frequent repetition of their action, result in chronic bronchitis. The constant inhalation of dust is one of the commonest causes of primary chronic bronchitis (see the chapter on diseases from the inhalation of dust). In many cases it is therefore a definite occupation disease, and is seen in millers, bakers, wool-workers, stone-cutters, colliers, etc.

In a large number of cases severe chronic bronchial catarrh is not an independent disease, but occurs as a complication or a result of other diseased conditions. The combination of chronic bronchitis with emphysema of the lungs (*vide infra*) is the most common. A large number of cases also are the result of some form of heart disease, such as valvular disease or myocarditis, or of disease of the vessels, leading to stasis in the pulmonary circulation, and finally to a chronic catarrh of the bronchi. Chronic bronchial catarrh in renal diseases also depends in part upon circulatory disturbances, and in part on the toxic action of urinary constituents which have not been normally excreted. Finally, we find a more or less extensive chronic catarrh of the bronchi in other chronic affections of the lungs and pleura, as in tuberculosis or pleurisy.

Chronic bronchitis is seen especially in adults and old people, and more frequently in men, on account of their exposure in the various trades, than in women, but children may also have pronounced cases of chronic diffuse bronchitis. Such cases may often be referred to a previous acute disease of the respiratory organs, especially whooping-cough, less often measles, etc. Such cases in childhood often continue into later life, and on careful inquiry we may trace many of the severe cases of chronic bronchitis in adults back to childhood.

Pathological Anatomy.—Chronic bronchitis is characterized anatomically by a persistent and chiefly venous hyperæmia and swelling of the bronchial mucous membrane with increased secretion of mucus and morbid exudation of serum and pus-corpuscles. There is often a hyperplasia of the mucous membrane with puffiness of the surface. In old cases, however, we finally meet with an atrophy of all the layers of the mucous membrane. One of the most frequent results of chronic bronchitis is a cylindrical dilatation of the middle and lesser bronchi—bronchiectasis—especially in the lower lobes. This arises gradually from the loss of elasticity of the diseased bronchial walls, and their diminished resistant powers, as well as from the pressure of the stagnating secretions.

Symptoms and Course of the Disease.—The symptoms which are due to chronic bronchitis are dyspnœa, cough, and expectoration. To these should be added the results of a physical examination, in making a diagnosis.

The cough is of very different severity in different cases. Usually it is worse early in the morning, in the evening, and at night, than in the daytime. The amount of expectoration is also subject to great variations. In many cases there is a dry cough (*catarrhe sec, vide infra*), in which only small amounts of tough, viscid sputum are expectorated. In other cases the expectoration is more abundant and muco-purulent, sometimes rather sero-purulent, and occasionally thin and separating into layers on standing. In catarrh of the finer bronchi the muco-purulent sputum at times indicates its origin by partly formed casts. We may also note the formation of so-called spirals (*vide infra*, bronchial asthma). Microscopically, the expectoration has no special characteristic appearances, but contains only the usual elements of sputum—pus-corpuscles mixed with pavement epithelium, ciliated epithelium, often many bacteria, sometimes needles of fat acids, and rarely a few pointed octahedral crystals, the so-called asthma crystals (*vide infra*). Small amounts of blood may be seen in severe chronic bronchitis, especially in the catarrh of passive congestion, bronchitis with very severe attacks of coughing, etc., but they do not have any bad significance.

Dyspnœa of moderate or even severe degree may be due solely to an extensive catarrh of the finer bronchi with narrowing of their lumen. In many cases in which there is bronchitis the dyspnœa is due chiefly to some co-existing abnormal condition of the heart or lungs.

Physical Examination.—The percussion in bronchitis shows no special change. At most the resonance may be somewhat tympanitic from the relaxation of the lung-tissue, especially in the lower and posterior portions of the lungs, or, with an abundant retention of secretion in the bronchi, it may be a little diminished. The inspiratory descent of the lower edge of the lung is lessened when the entrance of air is much impeded by the contracted or plugged bronchi. Auscultation may give either rhonchi, whistling, hissing, humming, etc., or moist râles, according to the extent of the catarrh and the amount and consistency of the secretion. The sounds are usually to be heard over the whole lung, or especially over the lower lobes, because here the catarrh is usually most marked, and retention of secretion is most apt to occur. The respiratory murmur in some places may be quite obscured by the râles. Otherwise it is vesicular, sometimes exaggerated, sometimes rough and indefinite. Expiration is usually prolonged, because the exit of air from the alveoli is much hindered by the narrowed bronchioles. The respiratory murmur may be much diminished, or even entirely suppressed in places where the bronchi are stopped by secretion, which happens most frequently in the lower lobes.

Except in mild cases, we usually distinguish several different forms of chronic bronchial catarrh, which may run into one another.

1. The dry chronic catarrh (*catarrhe sec* of Laennec) is the form in which the mucous membrane has only a slight secretion. The cough is usually very troublesome and labored, but the patient raises merely a little tough sputum, or none at all. On auscultation we hear sibilant rhonchi, but no moist râles. This form of catarrh is usually associated with pulmonary emphysema, and asthmatic attacks are also frequent. The disease is stubborn, and usually lasts for years.

2. The so-called bronchial blennorrhœa is that form of chronic bronchitis in which we find a very copious sero-purulent exudation on the surface of the mucous membrane. The cough is therefore associated with a very abundant and quite thin expectoration, the amount of which in the twenty-four hours may exceed a pint (half a litre). The expectoration runs together in the sputa-cup and usually separates on standing, the more purulent portion sinking to the bottom, and the sero-mucous portion, which is usually frothy on the surface, remaining at the top. Numerous moist râles are heard in the lungs, especially in the lower portions. These diminish if large amounts of sputum are coughed up. Anatomically, the bronchi are almost always found dilated in this form of chronic bronchitis.

3. The so-called serous bronchorrhœa ("*catarrhe pituiteux*" of Laennec) is quite rare but very interesting. It is characterized by the expectoration of a very large amount of frothy, almost purely serous, or sero-mucous, thin sputum containing only a slight admixture of pus. The cough usually comes on in very violent paroxysms which last from half an hour to an hour or more. The respiratory symptoms are quite severe, especially during these attacks, and have given rise to the old and useful term "*asthma humidum.*" The expectoration collected in twenty-four hours may amount to one or two quarts (litres). Examination of the lungs usually reveals very abundant and extensive moist râles. The resonance on percussion is normal or a little diminished, from the accumulation of secretion.

The special cause of this peculiar disease is quite obscure. There are mild afebrile and also very severe forms of this type of chronic bronchitis. We have seen several chronic cases, which were attended with persistent fever, and which led to great emaciation and weakness. In one case which came to autopsy there was found a marked tuberculosis of the retro-bronchial lymph-glands, while the lungs themselves showed hardly any striking changes. One vagus nerve was wholly imbedded in the tubercular mass of the glands, and it is not impossible that the peculiar attacks of serous expectoration were due to irritation of this nerve. At any rate, we should consider the possibility of such conditions in the future. It is worthy of note that attacks of "humid asthma," with expectoration of large amounts of serous sputum, have also been observed in chronic nephritis, especially in contracted kidney (*q. v.*).

Course of the Disease.—The course of most chronic bronchial catarrhs is very protracted. The disease usually has frequent remissions and fresh exacerbations. The patient is tolerably well in the pleasanter time of the year if he takes good care of himself, but in autumn and winter, or after exposure to various noxious influences, the catarrh grows worse and the patient's symptoms increase. If the disease has lasted for years, we usually find symptoms in the lungs, such as emphysema or chronic tuberculosis, or in the heart, such as secondary dilatation and hypertrophy of the right ventricle, which symptoms gradually become more severe. The details of these conditions are to be found in the appropriate sections.

Diagnosis.—The diagnosis of chronic bronchitis is not difficult in itself, and may easily be made by considering the patient's symptoms and by judging of the result of the physical examination. We must always consider, however, whether the bronchitis is actually a primary disease or a result or a complication of some other chronic disease. Therefore, in every case of chronic bronchitis, the heart and the kidneys (urine) must be carefully examined, as well as the lungs.

Prognosis.—Chronic bronchitis is in most cases a very stubborn affection, which frequently, as we have said, shows improvement, but from which complete recovery is rare. The prognosis also depends greatly upon the patient's circumstances, and upon the possibility of his taking care of himself and avoiding all harmful exposure. In secondary bronchitis the question whether the bronchitis is capable of material improvement or not of course depends mainly upon the prognosis of the primary disease.

The danger in primary chronic bronchitis comes from the final development of its sequelæ, especially from the gradual appearance of pulmonary emphysema, dilatation of the heart, secondary tuberculosis, etc.

Treatment.—The only hope of success in severe cases in any method of treating chronic bronchitis lies in removing the patient completely, at least for a time, from the action of injurious influences. The favorable result of the baths and health resorts that are employed depends largely upon this, that patients enjoy in them complete bodily rest, and are far better protected from dust and the changes in the weather than at home. We must make the patient comprehend the necessity of this condition as the basis of any treatment. If he can not go to a suitable climate during the cold season, he must keep his room in all unpleasant weather, but at other times he may be permitted to stay in the open air. Furthermore, the patient must be warned to avoid as completely as possible those harmful influences which his calling and manner of life entail, and among which especially to be mentioned is the bad air in our inns and restaurants. Food should be easily digestible, and, in persons inclined to corpulence, sparingly taken. Alcohol is to be permitted only in a moderate degree. We combat the tendency to constipation, which is often present, by dietetic remedies, by taking fruit, especially grapes, prunes, etc., honey, Graham bread, or by mild laxatives, such as the bitter waters, Friedrichshall, Ofner, etc., since any persistent constipation aggravates the patient's symptoms.

If the circumstances of the patient permit and his condition requires it, we should send him south in the autumn in order to avoid the evils of a northern winter, but we must always consider whether the patient has the strength to bear the burden and unavoidable discomfort of such a journey without permanent harm. The rule is to send patients with a bronchial catarrh when there is much secretion to health-resorts with a dry climate—for example, to the western Riviera, San Remo, Bordighera, Mentone, etc. The somewhat dry yet cooler climate of Meran, Gries, or Arco is suitable for patients with a stronger constitution. Patients with dry bronchitis usually find themselves at their best in a warm but not too dry climate. If we wish to be sure of avoiding the winter's cold, we must choose Sicily, Egypt, or Madeira for a residence. Of the more northern winter resorts we may mention places on the eastern Riviera, especially Nervi.

We must recommend, in bronchitis, a suitable summer residence, particularly for those who dwell in large and dusty cities. Any private country house in a well-wooded and protected place is suitable. If we wish to send patients to a bath, Marienbad, Kissingen, or Homburg are proper places for corpulent people who also suffer from digestive disturbances, while we may send weaker patients to

Ems, Soden, Badenweiler, Ischl, Reichenhall, etc. A summer residence on the sea, best on the Baltic, is very serviceable for many patients.

The inhalation treatment is much employed in chronic bronchitis, but we should not cherish too high hopes about its use. The best inhalations in dry catarrhs are simple steam, a one- or two-per-cent. solution of common salt or bicarbonate of sodium, Ems water, etc. In cases with marked secretion, inhalations of oil of turpentine are most to be praised. The simplest way is to pour a teaspoonful of oil of turpentine into hot water and inhale the vapor as it arises. The so-called turpentine-pipe, however, is more convenient and more efficacious (Fig. 23). This consists of a flask, which is filled to the height of several inches with water and then with a layer of oil of turpentine or of *oleum pini pumilionis* (P. G.), some two centimetres thick. Two glass tubes, open at both ends, are passed through the cork. One, straight, tube extends down into the layer of water : the lower end of the other is free in the upper part of the flask. The outer portion of this last tube is bent at an angle and is connected with the mouthpiece through which the patient inhales. The formation of turpentine vapor is aided by putting the flask in hot water. We have treated many patients in this way, who, for a number of hours a day, "smoked" their turpentine-pipes.

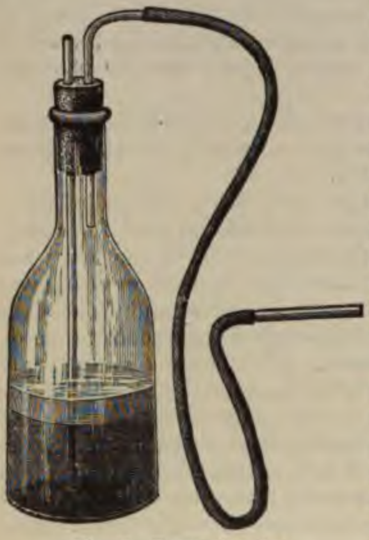

FIG. 23.—Turpentine-pipe.

In treating chronic bronchitis the pneumatic method was considerably employed for a time; that is, the patient was made to breathe artificially compressed air, or to expire into air of less than the atmospheric pressure, by means of a movable pneumatic apparatus, as proposed by Waldenburg and others. Of late, however, this method of treatment has received less favor, inasmuch as actual results have fallen decidedly short of the benefit promised. In Ems, Reichenhall, and other places special pneumatic cabinets have been arranged, filled with compressed air, in which patients remain for varying lengths of time.

One of the most serviceable remedies in dry chronic bronchitis is a large amount of warm drink (pectoral tea, Ems water, Seltzer, etc.). Iodide of potassium in small doses (gr. iij–v, grm. 0.2–0.3, thrice daily) often acts favorably in making a tough secretion fluid. Of expectorants, ipecac and apomorphine are most recommended in this form of bronchitis. For distressing cough we may give Dover's powder, morphine, or codeine. In bronchial blennorrhœa we know empirically that the internal use of balsams causes a distinct diminution of the secretion. Oil of turpentine is the most active, and may be given internally in gelatine capsules, or mixed with milk, in doses of ten or fifteen drops three or four times a day. After each dose of turpentine we should give a glass of milk. Lépine, G. Sée, and other French physicians recommend terpine as still more effectual. This is a derivative of turpentine, and is best employed in pills containing a grain and a half (gramme 0.1), of which two or even more are to be taken three times a day. It may also be given in solution as follows: Terpine, 2½ drachms (grm. 10); alcohol, q. s. ad sol. faciendam; aq. dest., 6 ounces, 6 drachms (200 grm). Misce. Sig: Two or three tablespoonfuls daily. Myrtol, balsam of copaiba, and balsam of Peru are also used internally. The chief expectorants are infusion of senega root, liquor ammonii anisatus, etc. We should be very sparing of narcotics at first, but in severe cases we can not wholly dispense with them.

[The iodide of potassium in doses of five to ten grains thrice daily is sometimes distinctly curative. An out-door life, free diet, moderate alcoholic stimulus, tonics, and woolen clothing do much to promote recovery.]

Local applications to the chest in the form of embrocations, mustard plasters, dry cups, or cool or warm wet compresses are to be used, especially with severe dyspnœa, or with pain and a feeling of oppression in the chest. Warm baths are very well borne by many patients with chronic bronchitis. Sometimes, too, vapor baths, if taken with caution, may be of service, especially in strong and corpulent patients. In dry chronic bronchitis we often give the patient a sweat in bed.

In all secondary chronic catarrhs our chief attention, beyond the symptomatic treatment of the bronchitis, must be directed to the treatment of the underlying disease. If we succeed in once more regulating the heart's action by digitalis when there is uncompensated heart disease, or in establishing diuresis when there is renal disease, or in improving the general physical condition by proper dietetic measures in the corpulent, gouty, alcoholic, etc., we may also in that way cause improvement in the existing bronchial catarrh.

CHAPTER III

FŒTID BRONCHITIS

(*Putrid Bronchitis*)

Ætiology.—By putrid or fœtid bronchitis we mean that form of bronchitis in which the secretion of the mucous membrane undergoes a putrid decomposition, and in which, consequently, the expectoration takes on a peculiar and extremely foul odor. The special agents which cause fœtid bronchitis are still unknown.

The opportunity for the agents of putrefaction to enter the bronchi with the inspired air is of course often given, but a fœtid bronchitis naturally is excited only when they can remain there and increase. Fœtid bronchitis therefore develops quite rarely in a previously healthy lung (primary fœtid bronchitis). The retention and the further development of the bacteria of putrefaction are chiefly favored, as we know, by diseased conditions which already exist in the bronchi. A great number of cases of fœtid bronchial catarrh therefore develop secondarily upon other pulmonary affections of longer standing. Thus the expectoration may quite suddenly change and take on a fœtid character in the course of a chronic or rarely of an acute bronchitis or of phthisis. Bronchiectasis (*vide infra*) greatly favors the development of this putrid change, for in it the retention and stagnation of large amounts of secretion promote and occasion the putrid decomposition. If a putrid decomposition of the secretion begins in one part of the bronchial system, the further extension of the process follows from direct infection.

In rare cases putrid bronchitis also develops as a result of an embolic pulmonary gangrene.

Symptoms and Course; Anatomical Changes.—If a fœtid bronchitis arises in the course of some other chronic pulmonary disease, its appearance may be marked by a sudden impairment of the general condition, by high fever, often associated with numerous chills, and by an increase of the thoracic symptoms, such as pain and cough. The change in the expectoration, the peculiarity of which was first accurately described by Traube, is characteristic. The expectoration shows the same character in cases of apparently primary fœtid bronchitis.

The sputum has a very repulsive, sweetish, putrid smell. The expectoration is usually quite abundant; the consistency is rather thin. On standing, the sputum shows a very marked division into three layers. This division is due to the abundant serous exudation in the bronchial mucous membrane and to the thin fluid character of the expectoration, which permits an unequal division and a settling of the solid constituents. The upper layer consists of a very frothy, muco-purulent stratum, consisting in part of individual masses, from which a number of coarser or finer fibers float down into the middle layer. This middle layer consists of a dirty-green muco-serous fluid. At the bottom of the vessel is found the third layer, which is often the thickest, and is composed entirely of pus. It consists of pus-corpuscles which have sunk to the bottom, and is of a rather thin, greasy consistency. With the naked eye we generally recognize a number of little whitish-gray plugs and particles in it. These so-called "Dittrich's plugs," which are easily crushed under a cover-glass, are quite characteristic. Microscopically, they consist of decomposed pus-corpuscles, detritus, and bacteria, and usually contain very many needles of fat acids arranged in bundles (see Fig. 24). We often find also in the sputum large masses of fungi, especially great bunches of twisted leptothrix fibers, which, by an unpracticed eye, may readily be mistaken for elastic fibers. The latter are, of course, never found in the expectoration of a simple fœtid bronchitis, but only in the deep-seated, destructive processes in the lung, like gangrene. On chemical examination of the sputum, the ordinary products of putrefaction may be found—volatile fat acids, especially butyric and valerianic acids, also sulphuretted hydrogen, leucine, tyrosine, etc.

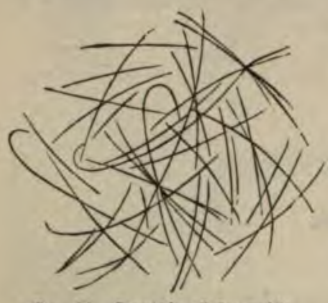
FIG. 24.—Crystals of fat acids.

It is very characteristic of many cases of fœtid bronchitis that the sputum does not have a putrid character at all times. Sometimes for days the patient coughs up a simple catarrhal secretion, and then all at once comes an outburst of intolerable stinking expectoration. This is probably because a circumscribed putrid focus is frequently shut off, the expectoration coming solely from the other bronchi, where there is merely a simple catarrh, until finally the accumulation of putrid secretion is all at once coughed up again in large amount by mouthfuls.

When fœtid sputum is coughed up, the patient's breath also becomes very foul-smelling, so that he often becomes a burden to his associates.

The signs which fœtid bronchitis gives on physical examination are those of an ordinary bronchitis; but it is characteristic of many cases, in distinction from ordinary chronic bronchitis, that the physical signs (râles, dry rhonchi) are limited chiefly to a definite portion of the lung, usually one lower lobe. In a great number of cases we also find signs of consolidation and contraction of the lung, of pleurisy, etc., which do not belong to fœtid bronchitis as such, but are due to complications or sequelæ.

The most frequent of these sequelæ is the development of a "reactive" lobular inflammation, a genuine pneumonia, which follows a catarrh of the finer bronchi. These pneumonias frequently run into gangrene, so that we very often find a number of larger or smaller nodules of gangrene besides the extensive fœtid bronchitis in the lungs. In many of these cases the fœtid bronchitis is certainly the primary process, and the development of the nodules of gangrene is secondary; yet we shall see later that the reverse may also be true. Fœtid bronchitis and gangrene of the lungs run into each other so often, both clinically and anatomically, that there is no sharp line to be drawn between them. If the nodules are superficial, and reach the pleura, the infection attacks this, and we have a purulent or even an ichorous pleurisy.

The smaller and medium-sized bronchi are almost always found in a condition of cylindrical dilatation in old fœtid bronchitis. Their mucous membrane is intensely inflamed, and often ulcerated superficially. On its surface we see in the cadaver the greasy purulent masses and the plugs which we find in the expectoration during life.

Whatever may be the case with the general course of fœtid bronchitis, its beginning is often quite sudden and acute, both in the primary and in the secondary forms, as we have said. The patient is attacked with fever, which may often be quite high, and with a stitch in the side, cough, and expectoration. Later, the characteristic peculiarities described above appear. The further course of the disease is almost always chronic, lasting for years, but subject to many variations. Very often manifest improvement, and even apparent recovery, takes place, when suddenly there is a new attack of fever and thoracic symptoms. The general condition and nutrition of the patient may be quite good for a long time, except during the periods of marked exacerbation of the disease. Patients with chronic fœtid bronchitis often appear somewhat bloated, but also pale and slightly cyanotic. Peculiar clubbed thickenings of the terminal phalanges of the fingers, or more rarely of the toes, gradually develop, as in many cases of bronchiectasis. Slight œdema of the lower extremities is also sometimes present.

Symptoms referable to other organs may be wholly absent. We see most frequently disturbances of the stomach, loss of appetite, and nausea, which probably comes from swallowing the fœtid sputum. Patients also complain of occasional rheumatic pains in the muscles and joints, which are probably due to an absorption of septic matter. In conclusion it must be mentioned that, in fortunately rare cases of fœtid pulmonary disease, pyogenic germs reach the brain by metastasis, and give rise to purulent meningitis or cerebral abscess.

The danger of the disease, apart from the exceptional occurrence just mentioned, lies in the possible extension of the process to the lungs and the development of pulmonary gangrene and its sequelæ. We hardly ever find a simple fœtid bronchitis in the cadaver, but we almost always see other processes besides, which have been mentioned above—reactive pneumonia, pulmonary gangrene, pleurisy, etc. All these processes develop very readily, and make rapid progress in old, decrepit persons, who live under bad hygienic conditions, in whom putrid processes in the lungs are frequent.

The **diagnosis** of fœtid bronchitis is not difficult in itself, for the diagnosis of a putrid process in the lung may be made from the stinking sputum alone. It may be difficult to decide whether we have to do merely with a fœtid bronchitis, or with a pulmonary gangrene also. Decisive indications of pulmonary gangrene are derived from physical examination—dullness, bronchial respiration, and coarse, moist râles, signs of a cavity—and also the discovery of elastic fibers and fragments of parenchyma in the expectoration.

The **prognosis** must be made with care in every case of fœtid bronchitis. If the external circumstances of the patient are favorable, he may remain in tolerable health for years. We must always be prepared for the appearance of new exacerbations of the disease and of affections of the lung itself, and must also bear in mind the rarer sequelæ (empyema, abscess of the brain).

Treatment.—The chief aim of treatment must be to bring the putrid processes in the bronchi to a stand-still by the death of the agents of putrefaction. The difficulty of fulfilling this task lies in the impossibility of getting the disinfecting material to act on the bronchial mucous membrane in the necessary amount and concentration. Nevertheless, we can, without doubt, at least relieve a fœtid bronchitis and keep it in check by the judicious use of inhalations. Inhalations of a two-per-cent. solution of carbolic acid are most useful, given for five or ten minutes several times a day. These, however, are sometimes not well borne if long continued, and they may excite mild symptoms of carbolic poisoning—such as headache, malaise, and dark carbolic urine. We have often used with good results the "carbolic mask" recommended by Curschmann, a kind of respirator fastened in front of the nose and mouth, containing cotton in a special receptacle impregnated with carbolic acid, equal parts of carbolic acid and alcohol, or other remedies such as turpentine or creasote. Many patients can wear these masks, with occasional interruptions, for many hours a day. Besides carbolic acid we may use inhalations of turpentine (turpentine-pipe or vapor), oleum pini pumilionis, creasote, etc. The internal use of these and similar remedies also seems frequently to be of service in putrid bronchitis. We recommend especially the internal use of turpentine (in gelatine capsules), myrtol (also in gelatine capsules), also terpine hydrate (six to ten two-grain [gramme 0.1] pills a day), creasote, etc.

In other respects all the general hygienic and symptomatic methods of treatment recommended for common chronic bronchitis (expectorants, narcotics, climatic health resorts, etc.) are also useful in fœtid bronchitis. The sputum should be disinfected by putting strong carbolic acid, etc., into the sputum-cup to lessen the bad odor. It is a very good plan to keep the carbolic spray at work in the patient's room as often and as long as possible; or the air may be impregnated with oleum pini pumilionis.

CHAPTER IV

CROUPOUS BRONCHITIS

(Fibrinous or Pseudo-membranous Bronchitis)

Croupous bronchitis is a peculiar form of disease of the bronchial mucous membrane, of very rare occurrence, in which there is a formation of extensive fibrinous patches in the bronchi. Only that form of croupous bronchitis which occurs primarily in the bronchi is to be considered here, and not the secondary form, which on the one side is associated with diphtheria in the pharynx and larynx, and on the other with croupous pneumonia.

The **ætiology** of the disease is as yet wholly unknown. From analogy with other well-known croupous inflammations of mucous membranes, we must look here for some noxious influence which destroys the epithelium, but up to this time we are entirely ignorant of its character. Individuals in youth and middle age, somewhere between ten and thirty years old, are the chief victims. Men are attacked somewhat more frequently than women. The disease comes on either in persons who were previously healthy—the essential fibrinous or croupous bronchitis—or in those who have already suffered from some other disease, especially some chronic pulmonary affection—the symptomatic, secondary croupous bronchitis. It is not certain whether the last-named cases have the same ætiological relations as the cases of genuine primary fibrinous bronchitis. Fibrinous bronchitis has been observed in the course of typhoid fever and other acute infectious diseases.

Symptoms and Course.—Primary fibrinous bronchitis occurs in two forms, acute and chronic. The acute form begins quite suddenly, with fever, cough, pain in the chest, and as a rule severe dyspnœa. The fibrinous coagula, which alone render the diagnosis possible, appear in the expectoration either at once, or after the existence for some days of what is apparently simple catarrhal bronchitis.

These coagula form complete casts of the bronchi, and are more or less branching. They are of a whitish color and of quite a dense, elastic consistency. The main stem may be a centimetre thick, and from it the further ramifications extend, dividing dichotomously. The largest casts are ten or fifteen centimetres long. On section, we usually find a free lumen within, and generally recognize a definite laminated structure in the membrane. In many places they are enlarged and swollen. Microscopically, we find white blood-corpuscles in and upon the hyaline ground-substance of the casts, and also red blood-corpuscles, sometimes epithelial cells, and quite often the peculiar pointed octahedral crystals which are also found in the expectoration in bronchial asthma (*vide infra*). The so-called "spirals" (*vide infra*) have also been found in the expectoration of fibrinous bronchitis. Chemically the casts apparently consist of coagulated albumen. It is doubtful whether they actually consist of fibrine. Weigert's fibrine stain does not affect them. Their solubility in alkalies, especially in lime-water, is of therapeutic importance.

On coughing, the patient usually raises a simple mucous or muco-purulent expectoration besides the casts, and in this sputum the casts are imbedded. They are often first discovered by pouring the whole amount of sputum into water, when they unfold and spread out. The expectoration also contains not infrequently a slight admixture of blood.

The subjective symptoms of the patient may be very violent. The dyspnœa sometimes attains a high and alarming degree. It ceases when a large cast is expectorated after a severe paroxysm of coughing. Such attacks may recur every day or two. In other cases, however, the subjective symptoms are comparatively slight.

Physical examination of the lungs reveals little that is characteristic. In uncomplicated cases percussion shows nothing abnormal, or at most the signs of an acute emphysema. Auscultation gives the ordinary signs of bronchitis, not characteristic in themselves, such as rhonchi, or moist râles. If a large bronchus is plugged, the respiratory excursions and the respiratory murmur are almost entirely absent in the corresponding portion of the lung, but after the expectoration of a cast the murmur becomes once more audible.

The duration of acute cases is sometimes only a few days, at most a few weeks. In favorable cases the fever, which at times is quite high, soon disappears, the respiratory symptoms grow milder, the expectoration of the casts ceases, and there is a complete and permanent recovery. In severe cases, however, death often ensues with all the symptoms of suffocation. The acute form sometimes becomes chronic, but this is rare.

The chronic form of fibrinous bronchitis may last for years. Usually the condition grows worse periodically, at varying intervals of time, and at each exacerbation casts are expectorated, while in the interval there is apparently merely a simple bronchial catarrh. Some observations are also recorded in medical literature of persons who have expectorated these casts at intervals for years without any special disturbance of their health or their nutrition. In some cases other chronic pulmonary affections, such as tuberculosis, finally develop.

The pathological anatomy of fibrinous bronchitis is not yet satisfactorily known on account of the rarity of the affection. The changes in the lungs found at the autopsy of fatal cases have usually been complications, such as pneumonia, pleurisy, or tuberculosis, which stood in no direct relation to the fibrinous bronchitis. A loss of epithelium has been discovered in some cases in the parts of the bronchial mucous membrane that were attacked.

Prognosis.—In all acute cases the prognosis should be guarded, for we know that about one fourth of the cases terminate fatally. The chronic cases, as has been said, are usually very protracted and are subject to frequent exacerbations, but they differ from the acute cases in being much less dangerous.

Treatment.—We make special use for inhalations of those remedies which, as we have said, have power to dissolve the casts. We usually employ a two- to five-per-cent. solution of carbonate or bicarbonate of sodium, and above all lime-water, either pure or diluted with an equal volume of water. The internal administration of iodide of potassium, in doses of twenty to forty-five grains (gramme 1.5–3.0) a day, proves of advantage in many cases. Energetic inunction with mercurial ointment is sometimes of service. Expectoration of the casts may be aided in many cases by such expectorants as senega and benzoic acid, or by the timely use of emetics. We do not know any remedies which can prevent a return of the attacks in the chronic form. The treatment, except at the time of the attacks, is the same as in ordinary chronic bronchial catarrh.

CHAPTER V

WHOOPING-COUGH

(*Pertussis. Tussis convulsiva*)

Ætiology.—By the name "whooping-cough" we mean a specific disease of the mucous membrane of the air-passages, which is seen chiefly in children, and is characterized by a peculiar violent and paroxysmal cough. Sporadic cases are of almost constant occurrence in large cities, but the disease often appears in epidemic outbreaks. Epidemics of whooping-cough follow epidemics of measles with remarkable frequency.

Whooping-cough is without doubt contagious, and therefore often attacks one child after another in the same family. Kindergartens, orphan asylums, and day-nurseries aid very much in extending the disease. The contagious element seems to be connected with the air expired by the patient, particularly with the secretion from the mucous membrane expectorated after coughing. Children are most subject to an attack up to the age of six years; from that age the liability to the disease decreases rapidly with increasing years. Whooping-cough is seen, indeed, in adults, but it is quite rare, and almost always without the pronounced features of tussis convulsiva.

The epidemic onset, the contagiousness, and the whole course of the disease favor the theory of its infectious nature. The presence of the organisms which are supposed to be the poison of the disease has not yet been certainly demonstrated, although many have claimed to discover characteristic bacteria in the sputa of patients. These assertions, however, do not harmonize with one another, and lack well-attested and methodical proof. If a patient has once had the disease, he is almost invariably safe from a second attack.

Symptoms and Course of the Disease.—Whooping-cough begins with the symptoms of a catarrh of the trachea and bronchi, which develops more or less rapidly, and which at first often shows nothing characteristic. We can at this period make a tolerably probable diagnosis only at a time when an epidemic is prevailing, or in case the child's associates have already been attacked with the disease. The cough is often very persistent, obstinate, and severe at the beginning, but it does not yet come on in distinct paroxysms. Examination of the chest shows nothing peculiar except a few rhonchi. There is often a coryza, with frequent sneezing, and there is sometimes a mild conjunctivitis. The child is restless and feverish, especially toward night. The temperature may repeatedly reach 103° or 104° (39°–40° C.) in this initial fever. The duration of this first so-called catarrhal stage varies, but it usually lasts a week or ten days.

The catarrhal stage gradually passes into the second, convulsive stage, without any sharp boundary. The cough becomes more violent, and comes on in the separate paroxysms of whooping-cough which are characteristic of the disease. We do not know the particular reason why the cough has this paroxysmal character, but a nervous factor probably plays the chief part in it.

The peculiarity of the attack consists in the violent, paroxysmal fits of coughing, which are from time to time interrupted by deep, long-drawn, loud, and shrill inspirations, due to the occurrence of a spasmodic contraction of the glottis. Exceptionally there are cases without this loud whistling inspiration. The child becomes markedly cyanotic during the attack, the veins in the neck swell, and tears come into the eyes. Hæmorrhage into the conjunctiva, nose-bleed, and in some cases hæmorrhages into other organs, as the ear and the skin, often come on as a result of this stasis. Vomiting very often occurs either during a paroxysm or at its close. Involuntary evacuations of urine and fæces may also follow from the violent contraction of the abdominal muscles. Exceptionally we observe still more severe symptoms with a paroxysm: a complete spasmodic cessation of respiration with imminent danger of suffocation, or sometimes general convulsions. In two cases we have seen hemiplegia in children which, according to the positive statements of their parents, came on suddenly during a severe attack of whooping-cough. It is still undetermined whether these "whooping-cough hemiplegias" are due to an actual cerebral hæmorrhage or whether they come from the enormous venous stasis in the brain.

The paroxysms vary with the severity of the disease, frequently appearing ten or fifteen times in twenty-four hours; sometimes with greater frequency—fifty times or more. They also occur at night as often or even oftener than in the daytime. They come on either spontaneously or from some special predisposing cause. The attacks which come on during eating cause the most disturbance, because the food taken is almost always vomited again. In children with whooping-cough we can often excite a spasm at any time, and this is important in diagnosis—by putting a spatula in the mouth, by pressing on the larynx, or by making the child cry. If there are several children with whooping-cough in the same room and a paroxysm attacks one of them, the others, as a rule, soon begin to cough too. Some prodromal symptoms often precede the peculiar paroxysm, such as general uneasiness, rapid respiration, or vomiting. At the end of a paroxysm many children are very feeble and exhausted, but others recover rapidly, and are playing again quite briskly a few minutes after.

In general the child feels quite well in the interval between the paroxysms, but the effects of the violent attacks of coughing may of course often be seen. Besides the occasional hæmorrhages into the conjunctiva, we find the eyelids somewhat swollen, their veins dilated and blue, and showing through the skin. Many children grow very thin from the repeated vomiting. A small ulcer is quite frequently formed on the frænum of the tongue, the origin of which is to be referred to mechanical causes. The tongue is violently protruded in the severe paroxysms of coughing, and the frænum is thus torn, or injured by the sharp lower incisors.

Physical examination of the lungs shows nothing abnormal in uncomplicated cases except a few moist râles or rhonchi. Sometimes the rhonchi are wanting, or are present in small numbers only a short time before a paroxysm, but in other cases an intense diffuse bronchitis is developed, which often leads to the development of a lobular pneumonia (*vide infra*). Sometimes, but not always, there is an acute catarrhal inflammation of the bronchi, and especially of the posterior wall of the larynx.

The fever, which is usually present in the first or catarrhal stage, is absent in **the convulsive stage.** The child is free from fever for the most part. We often find a slight rise of temperature up to 100° or 101° (38°–38.5° C.), but only toward night. Higher and more persistent fever points to the development of complications, especially on the part of the lungs.

The convulsive stage seldom lasts less than three or four weeks, and often much longer, up to three or four months. The paroxysms gradually become less frequent and less violent (*stadium decrementi*), until they finally disappear entirely; but relapses and fresh exacerbations also occur in this stage. A certain "irritability" of the bronchial mucous membrane remains for a long time after whooping-cough. Finally, however, the disease, in uncomplicated cases, goes on to a permanent and complete recovery.

Complications and Sequelæ.—The severe results which sometimes follow whooping-cough are probably due in part to the direct action of the specific causes of the disease, and in part to complications of a secondary nature whose development is merely favored by the whooping-cough. The most important are complications in the lungs. A lobular catarrhal pneumonia often develops after a severe bronchitis which involves the finer bronchi. In such cases the respiration becomes hurried and superficial, the fever higher, and the general condition bad even in the times between the paroxysms. On examination of the lungs, we hear numerous moist râles, especially over the lower lobes; and we can sometimes make out dullness on one or both sides, if there is extensive pneumonic infiltration. Such cases are always very protracted, and many children succumb, partly from the disturbance of respiration and partly from general weakness and inanition.

Complications in other organs are much rarer. Among the most frequent are attacks of diarrhœa which impair the child's nutrition. Many observers have also mentioned the quite frequent occurrence of a croupous or diphtheritic inflammation in the pharynx and larynx in the course of whooping-cough. Finally, a case under our own observation may here be mentioned, in which death occurred with severe nervous symptoms, convulsions, and coma. At the autopsy very numerous capillary hæmorrhages were found in the brain.

Pulmonary emphysema is the first thing to be mentioned among the sequelæ of whooping-cough. From the marked pressure which the severe and frequent outbursts of coughing exert from within upon the alveoli of the lungs, they gradually become dilated. An acute lobular emphysema ("acute pulmonary inflation") is set up, which sometimes passes into a typical chronic pulmonary emphysema (*vide infra*). Chronic bronchial catarrh may also remain for a long time after an attack of whooping-cough. We have already stated (see page 163) that many incurable cases of chronic bronchitis in adults are to be referred to an attack of whooping-cough in childhood.

A third important sequel of whooping-cough is pulmonary tuberculosis. The bronchitis and lobular pneumonia which occur during whooping-cough sometimes do not improve, especially in weak children with a tubercular tendency. The fever continues high, the child grows thin, and constantly becomes more and more miserable. At the autopsy we find cheesy nodules in the lungs, cheesy bronchial glands, and at times tuberculosis of other organs. These cases signify that when a tubercular infection is present, but is still latent, the whooping-cough acts as an exciting cause for the outbreak of the disease, or that a greater receptivity to infection with tubercular poison is created by the whooping-cough. Möbius has lately reported the occurrence, in a few cases, of paralysis as a sequel of whooping-cough. This usually begins in the lower and extends to the upper extremities, and is due apparently to neuritis.

The **diagnosis** of whooping-cough can not be made with certainty, as we have said, until the second or convulsive stage. It is easy then, however, since the characteristic attacks occur in no other affection of the lungs in like manner and with like frequency and duration. If we have no opportunity to observe the attack itself, and have to depend upon the description of the friends, the diagnosis is sometimes more uncertain; but the accounts of the occurrence of cough, in individual paroxysms associated with vomiting, are usually so characteristic that errors are, on the whole, rare. Furthermore, the child between the paroxysms usually presents certain signs: he has a bloated aspect, or we may find slight hæmorrhages into the conjunctiva, or ulcers on the frænum of the tongue, which make the diagnosis highly probable. Under some circumstances we may also make the attempt to bring on the paroxysm artificially (*vide supra*). In adults, as we have said, the attacks are rarely as characteristic as in children. There are usually the symptoms of a more or less severe bronchitis with obstinate paroxysmal cough, but without the characteristic attacks and usually without vomiting. The diagnosis of whooping-cough therefore rests mainly upon the existence of special ætiological conditions (the co-existence of the disease in children, etc.).

The **prognosis** is favorable with the majority of children if they are previously strong and healthy. Very young children are in more danger than older ones. [Under two years there is great danger, and over five scarcely any. It has been said that whooping-cough causes one fourth the total mortality of children in London.—V.] There is danger if secondary pneumonia develops, and if the general nutrition and strength of the child suffer. As soon as the diagnosis is certain we must call the attention of the parents to the probable long duration of the disease. Regard must also be paid to the possibility of the development of sequelæ, especially in weak children suspected of tuberculosis.

Treatment.—Since the disease is protracted and is not devoid of danger, it is our duty, when an epidemic of whooping-cough prevails, to guard children from it as far as possible. If one child in a family is taken ill, the other children must be rigorously kept away from him. If circumstances permit, we should prefer to send them away to another place free from whooping-cough.

With regard to the treatment of the disease, we must first endeavor to fulfill general dietetic and hygienic indications. The child should breathe good, pure air, and for this reason it is often advisable to transfer the patient to a larger room, with as much air and sunlight as possible. The atmosphere should not be too dry, and it is advisable to employ a spray of water (carbolic solution) frequently, or to hang up moist sheets in the room. In good weather the child should be out of doors a large part of the time, provided fever has ceased. City children are to be sent, if possible, into the country. The food should be good and nourishing, but dry and crumbly articles should be avoided, being apt to excite cough. Warm or lukewarm baths frequently prove very beneficial, particularly when there is considerable bronchitis, as they lessen the danger of a lobular pneumonia.

The medicinal treatment of whooping-cough has not yet shown brilliant results despite the large number of remedies recommended. During the catarrhal stage it is usually sufficient to give a simple expectorant (ipecac, etc.) and plenty of warm drink. In the convulsive stage the best remedies to try are quinine, antipyrine, belladonna, bromide of potassium, and bromoform. The latter has been much recommended of late. Quinine is given in powders of one and a half to eight grains (grm. 0.1 to 0.5) several times a day, either in capsules or, in the case of smaller children, with chocolate. The earlier this remedy is employed the more prompt is said to be its beneficial influence. Antipyrine is at present used more frequently than quinine, and in doses of four to eight grains (grm. 0.25–0.5) several times a day it often produces distinct improvement. Belladonna is prescribed in powders containing one twelfth to one sixth grain (grm. 0.005 to 0.01) of the extract of belladonna, giving three to five such powders a day. This remedy has often seemed to the author to diminish the number and violence of the paroxysms. Bromide of potassium is employed in an aqueous solution in the dose of fifteen to forty-five grains per diem (grm. 1 to 3). Its efficacy is probably due to its power to diminish reflex excitability. The same drug employed in an atomizer often has a palliative effect. Bromoform is the remedy most used of late; two to five drops or more are given several times a day in sweetened water or fruit juice. It is readily taken, and it seems to act favorably both on the severity of the individual attacks and on the whole course of the disease.

If the paroxysms are very violent we may cautiously administer small doses of morphine or codeia. Inhalations of chloroform and ether have also been recommended. The following mixture is a suitable one:

℞	Chloroformi	℥j	(grm. 30);	
	Ætheris	℥ij	(grm. 60);	
	Ol. terebinthinæ	ʒijss.	(grm. 10).	M.

Sig. One or two teaspoonfuls to be poured upon a pocket-handkerchief for inhalation.

Finally, efforts have been repeatedly made to lessen the frequency and the severity of the attacks by anæsthetizing the pharynx and larynx by painting with a ten- to fifteen-per-cent. solution of cocaine. Michael advocates the daily insufflation into the nostrils of powdered benzoin. Neither method of treatment has succeeded in establishing itself in practice.

Sprays of various antiseptic remedies have been frequently employed because of the infectious nature of the disease. The practitioner must not expect too much from them, however, although they sometimes act well. A one-per-cent. or two-per-cent. solution of carbolic acid is most frequently employed for inhalation. It may be given several times a day for periods of two or three minutes at a time. Next to this, turpentine and benzine are most to be recommended; of these, twenty or thirty drops are poured upon a sponge previously moistened with hot water. Turpentine and especially terpine hydrate (grains vij, gramme 0.5, thrice daily) are also given internally. [Farlow and others report marked success from spraying the upper air-passages with a two-per-cent. solution of resorcine.]

In very many cases we finally content ourselves with taking good general care of the child, and, if the weather permit, with keeping him as much as possible in the open air. For the treatment of the complications and sequelæ of whooping-cough the reader is referred to the appropriate chapters of this book.

CHAPTER VI

BRONCHIECTASIS

(Bronchial Dilatation)

Dilatation of the bronchi is not a separate disease, but it is a result of various affections of the lungs and bronchi. Nevertheless, we will speak of it briefly in this connection since many cases actually present the appearance of a very characteristic disease.

We distinguish anatomically the cylindrical and saccular bronchiectases.

Cylindrical bronchiectasis consists of a uniform dilatation of a bronchial tube, and occurs most frequently in the medium-sized, or rarely in the finer bronchi of one or more lobes of the lung. We usually find at the autopsy that, on slitting up the bronchi, the point of the scissors can easily be pushed through the dilated bronchial tube close up to the pleura. Cylindrical bronchiectasis is usually due to a long-continued bronchitis, and develops most frequently in cases of emphysema, and also in whooping-cough, measles, and sometimes in phthisis, etc. The primary process is probably always the atrophy which follows the catarrh, and the diminished resistance of the bronchial walls thus occasioned. The dilatation of the lumen of the bronchus is produced gradually, partly by the traction of the thorax during inspiration, and still more by the increased pressure in the bronchi due to the frequent and violent fits of coughing, and finally, perhaps, by the constant pressure of the stagnating secretion.

The diagnosis of cylindrical dilatation of the bronchi is only a probable one. We suspect that a bronchiectasis has formed if the conditions are fulfilled which we know lead to it. In the chronic bronchial catarrh of emphysema we judge that there is cylindrical dilatation of the bronchi if the secretion is very abundant and comparatively thin, and separates into layers on standing in a sputum-cup. The dilatation is usually emptied by a severe paroxysm of coughing, such as is apt to occur in the morning if the secretion collects in great quantity during the night. Physical examination usually gives numerous, low, fine, and medium moist râles, especially in the lower lobes. The respiratory murmur sometimes loses its vesicular character in marked cylindrical bronchiectasis, and has a more indefinite and tubular quality. Sometimes it is quite obscured in the lower part of the lung by the abundant râles.

Saccular bronchiectases are spherical or oval dilatations which are confined to a definite portion of the bronchial tube. They may attain a diameter of several centimetres. The bronchus passes suddenly or gradually into the dilatation, and it is often obliterated so that the bronchiectasis forms a completely closed cavity. The wall of a saccular bronchiectasis loses in great measure the character of a normal bronchial wall. As a rule it is atrophied to a high degree, the atrophy involving not only the mucous glands, but also the muscular fibers, the elastic elements, and even the cartilages, so that the bronchiectatic cavities seem lined with nothing but a thin membrane. The occurrence of many dilated vessels in the walls of a bronchiectasis is due perhaps to an atrophy of the vessel walls and is a symptom of great clinical importance (*vide infra*). In other cases, however, we find hypertrophic processes, which involve the connective tissue of the mucous membrane, and lead to band-like projections and swellings. Finally, ulcerative processes may develop on the inner surface of a bronchiectasis and attack the surrounding lung-tissue, and change the bronchiectatic cavity into a typical ulcerating cavity.

Only rarely, for example in emphysema, do we find a single saccular bronchiectasis surrounded by tolerably normal lung-tissue. Its origin, then, is to be referred to causes like those which have been given above for the much commoner cylindrical bronchiectases. In the great majority of cases we find saccular bronchiectases, singly or in large numbers, surrounded by indurated and contracted lung-tissue. They form one of the complications of "pulmonary contraction" [fibroid phthisis], which is almost always associated with contraction of the pleura. Since Corrigan's day we have with good reason looked upon this contraction as the chief cause for their origin. By the gradual shrinking and retraction of the lung, which as a rule has become adherent to the costal pleura, a traction is exerted upon the bronchial walls from without to which they gradually yield. Stagnation and pressure of the secretion work at the same time in like manner to dilate the bronchial tube. Thus arises the frequent combination of pulmonary contraction with the formation of bronchiectases. This combination is usually unilateral, and involves the whole lung or only one of the upper or lower lobes. Our knowledge is still very defective as to the cause of this "chronic interstitial pneumonia" leading to contraction. It often seems to be a quite insidious and gradual circumscribed chronic inflammatory process; in other cases by careful inquiry we can discover a previous acute process (pneumonia, severe acute bronchitis, etc.) as a starting-point for the disease. Inhalation of dust often plays a part in the causation.

We sometimes see the form of pulmonary contraction in question developing as a result of pleurisy. Laennec first advanced the opinion that in such cases the pleurisy was the primary trouble, and that from it an interstitial inflammatory process attacked the connective tissue of the underlying lung and led to contraction and then to the formation of bronchiectases. Unfortunately we lack further confirmatory evidence in this regard.

Most saccular bronchiectases are situated in the lower lobes, but genuine bronchiectasis is not very rare in the upper lobes. As a rule, the trouble is unilateral, or at least affects one side the most. Sometimes large portions of a lung may be completely transformed into a cicatricial tissue studded with many bronchiectatic cavities.

"Pure cases" of bronchiectasis have nothing to do with tuberculosis. Before the discovery of the tubercle bacillus there was, of course, confusion between bronchiectatic and chronic tubercular contraction of the lungs; but we must remember that, under some circumstances, chronic tuberculosis may lead to the formation of bronchiectasis, and that, on the other hand, in bronchiectatic processes there is not infrequently a secondary development of tuberculosis. There are cases in which it is hard to decide, even at the autopsy, whether an existing chronic contraction in a portion of the lung, with induration of tissue and the formation of bronchiectasis, was originally tubercular or of some other nature.

The symptoms caused by saccular bronchiectasis are derived in part from physical examination and in part from observation of the sputum and of the general course of the disease. If great bronchiectatic cavities lie near the chest-wall, they may give the same physical signs that we shall learn to recognize later in the description of tubercular cavities. Bronchiectases lying within the lung, however, are often devoid of definite physical signs, so that at most we may suspect them from other symptoms, such as the peculiarities of the sputum. The more abundant the formation of bronchiectases the more does the respiration lose its vesicular character and become harsh and finally bronchial. Inasmuch as there is usually a very considerable secretion of mucus, we generally find, upon auscultation, abundant medium and even coarse moist râles. The percussion note over the bronchiectatic portion of the lung is usually dull or dull-tympanitic, a result of the chronic interstitial pneumonia about the bronchiectasis. The whole thorax near an extensive bronchiectasis is often much retracted and distorted.

The expectoration is, as a rule, very abundant, and it is often raised by large mouthfuls. On standing, as a result of its thin fluid character, it exhibits a distinct division into an upper frothy, muco-purulent layer, a middle muco-serous layer, and a lower purulent layer. It usually has a peculiar stale, sweetish odor, but it may be fœtid. The latter is almost always associated with stagnation of the secretion. So long as the expectoration is loose and easily evacuated, it is not fœtid and the patient feels well. Then a cessation of the expectoration may ensue. The patient feels poorly, there may be a slight rise of temperature, and the expectoration becomes scanty and has a stinking fœtid character. Periods of improvement may thus alternate with exacerbations. It is a favorable sign when the amount of the expectoration decreases, provided that it does not become fœtid and that the patient does not feel worse. Since bronchiectasis may give rise to a permanent fœtid bronchitis, and since, on the other hand, as we have said, fœtid bronchitis itself often leads to the formation of bronchiectasis, we can understand the manifold inter-relations and transitions which the two forms of disease present.

The not infrequent pulmonary hæmorrhages in bronchiectasis are of great clinical importance. They are due to rupture of the dilated vessels (*vide supra*) in the walls of the bronchiectasis. They may also be due to ulcerative processes. Slight precursors may precede a severe hæmoptysis. The hæmorrhage may be very abundant, and it may be repeated during a long period—several weeks—so that the patient becomes extremely anæmic. Finally the hæmorrhages cease and the patient improves quite rapidly. Such attacks of hæmoptysis may be repeated very often in the course of the year. When we hear comparatively well-nourished patients with lung disease say that they have suffered for years from frequent and severe hæmoptyses, we can usually infer, from such statements, that there is a saccular bronchiectasis.

The whole course of the disease is very variable, but it usually lasts for many years. It is doubtful whether a saccular bronchiectasis can actually be cured; but if no more serious sequelæ occur, many patients may live for years in a tolerable condition, and may even reach an advanced age. The general nutrition often remains very good, although a certain very characteristic, anæmic, cyanotic tinge of the skin gives even to corpulent patients an unmistakable morbid appearance. A very characteristic sign is that the terminal phalanges of the fingers gradually become clubbed and thickened, and the nails are much bent. This peculiar trophic change, the cause of which is still unexplained, is much rarer in chronic tubercular processes (*vide infra*).

The diagnosis of extensive formation of bronchiectasis in the lungs is in many cases easy, but in other cases it may present great difficulties. In order to avoid confounding it with chronic tuberculosis the following should be especially considered: In bronchiectasis the patient does not present a true cachexia; he is usually somewhat cyanotic and pale at the same time. The layer of subcutaneous fat often remains well marked for a long time. The terminal phalanges of the fingers are often clubbed, as in fœtid bronchitis. Fever is usually absent, unless there are some special complications. The expectoration is more abundant and more distinctly stratified than is often seen in tuberculosis; and, most important of all, it contains, of course, no tubercle bacilli. Finally, bronchiectatic conditions ordinarily (though, of course, not always) develop in a lower lobe, while tuberculosis is apt to affect an upper lobe first.

We will refer to the appropriate chapters for the relations of bronchiectasis to fœtid bronchitis, pulmonary gangrene, etc.

The **treatment** is never directed against the bronchial dilatation as such, but toward its causes or sequelæ. The treatment of bronchiectasis, therefore, coincides with the treatment of chronic bronchitis, fœtid bronchitis, etc. Besides general hygienic measures (climatic resorts), balsamic remedies (turpentine, etc.) are chiefly used. Any pulmonary hæmorrhage is to be treated symptomatically in the same way as hæmoptysis in pulmonary tuberculosis (*q. v.*).

CHAPTER VII

STENOSIS OF THE TRACHEA AND BRONCHI

1. Tracheal Stenosis

Ætiology.—Stenosis of the trachea may be caused either by diseases in the vicinity of the trachea, or by diseases of the trachea itself. The first-named mode of origin is the more frequent. To this are due all the stenoses of the trachea from compression. Enlargements of the thyroid gland from simple struma and new growths, aneurisms of the arch of the aorta and of the innominate artery, tumors and abscesses in the anterior mediastinum, swelling of the lymph-glands at the bifurcation of the trachea, abscess on the anterior surface of the cervical vertebræ, etc., may exert so great a pressure on the trachea from without that its lumen is made narrower. Besides the direct action of pressure, in most cases, a gradual atrophy and a softening of the rings of cartilage, due to pressure, sometimes play an important part, according to Rose, in the occurrence of stenosis. A collapse of the trachea may arise from this "flaccid softening," which may come on quite suddenly, and may cause many of the cases of sudden "death due to goître."

Changes in the trachea itself leading to stenosis are quite rare. Cicatricial stenosis as a result of syphilitic ulcerations is relatively the most frequent. New growths in the trachea are also to be mentioned, such as polypi and cancer, the latter almost always having invaded the trachea from the adjacent parts. Very rarely acute and chronic inflammatory processes such as perichondritis lead to a swelling of the mucous membrane sufficient to cause stenosis. In conclusion, we may mention that stenosis of the trachea may be due to the presence of foreign bodies.

Symptoms.—If the stenosis is so extreme that there is a real hindrance to respiration, a very striking modification of the breathing occurs. It is difficult and labored, and is performed only by the help of the accessory muscles. Both expiration and inspiration are protracted, long drawn, and accompanied by a loud stridor. In many cases inspiration is more difficult than expiration, so that there is accordingly a preponderating inspiratory dyspnœa, and the number of respirations a minute is diminished. If the entrance of air into the lungs is incomplete in spite of the lengthening of the respirations, we see an inspiratory retraction of the lower part of the thorax, and sometimes of the throat and the supra-clavicular fossæ. In tracheal stenosis the larynx, however, shows little or no to-and-fro movement on respiration. This fact is of value in diagnosis in distinguishing tracheal from laryngeal stenosis, for in the latter the respiratory movements of the larynx are quite well marked.

We sometimes notice in the pulse during inspiration a marked fall in tension and in the height of the pulse-wave, the *pulsus paradoxus*. With the sphygmograph we can show still more plainly the changes in blood-pressure, which vary quite markedly with the respiration. The frequency of the pulse is usually a little increased, but sometimes it is diminished.

The symptoms of the disease just described may form so characteristic a picture that we can recognize it at the first glance. More precise information as to the seat of the stenosis, or the accurate differentiation of tracheal stenosis from the very similar picture presented by laryngeal stenosis, demands a direct laryngoscopic examination of the larynx and trachea, which, of course, is hardly practicable in a patient with a high degree of dyspnœa.

2. Bronchial Stenosis

Narrowing of a primary bronchus, which is the only form to be mentioned here, arises most frequently as a result of the presence of foreign bodies—e. g., bits of bone, plum-stones, buttons, etc. These may enter the air-passages by means of a deep inspiration, especially while eating, or during sleep. We know that foreign bodies get into the right bronchus, which is wider, somewhat more frequently than they do into the left. Stenosis of the main bronchi from pressure also arises from aneurisms of the aorta, mediastinal tumors, enlarged bronchial lymph-glands, etc. Stenosis of the left bronchus from the pressure of the greatly dilated left auricle has been observed in mitral stenosis.

The symptoms are not equally distinct in all cases, and they depend upon the shutting off of the corresponding part of the lung. The dyspnœa is usually very evident, especially in acute cases. The respiratory excursions are much less on the affected side than on the sound side. The percussion-note remains clear; sometimes it is very deep, because the lung beyond the narrowed bronchus is constantly inflated. Auscultation shows complete absence of the vesicular respiratory murmur. We either hear nothing or we sometimes hear over the whole side a loud whistling or humming sound, the vibration of which can in some cases be felt by the hand applied to the chest-wall. The vocal fremitus is diminished on the affected side. A vicarious emphysema soon develops in the other lung.

Lobular pneumonia frequently develops as a result of the entrance of foreign bodies into a bronchus, because the agents of inflammation have entered at the same time with these bodies, and, as the expectoration can be evacuated only with difficulty, and hence is more or less stagnant, these irritants can readily establish themselves in it. In stenosis from pressure the condition may of course be modified in many ways by the primary disease.

The **prognosis** and **treatment** of tracheal and bronchial stenosis depend entirely upon the nature of the primary disease. General statements as to treatment, therefore, need not be given here. A direct mechanical treatment of tracheal stenosis in appropriate cases, such as cicatricial stenosis, may be undertaken according to the different modes of dilatation above enumerated. The methods for removing foreign bodies from the larger air-passages belong to the domain of surgery. The employment of an emetic has met with distinct success in such cases, but it is not without danger, for the foreign body may wedge itself into the glottis during the act of vomiting and occasion danger of instant suffocation.

CHAPTER VIII

BRONCHIAL ASTHMA

(*Exudative Bronchiolitis. Asthmatic Bronchiolitis*)

Definition and Causes of the Disease.—Bronchial asthma is the name we give to a morbid condition whose chief symptom is a special form of severe dyspnœa. The characteristic features of the dyspnœa of bronchial asthma are, first, that it occurs in paroxysms, or at least that it grows worse paroxysmally, and, second, its peculiar manner of onset. The special character of the dyspnœa of bronchial asthma is explained by the fact that every true bronchial asthma is due to an extensive and quite rapid contraction of the smaller and smallest bronchial branches. This always gives rise to a form of distress for breath which differs materially from every other form of dyspnœa.

The question as to the origin of bronchial asthma coincides, then, with the question as to the origin of that extensive contraction of the bronchioles which is the root of the disturbance. There seems to us no doubt that the latter should not always be referred to the same cause. We must therefore distinguish several forms of bronchial asthma, which, as a result of the similar conditions of dyspnœa, have many points of clinical similarity.

Formerly the so-called nervous bronchial asthma was considered to be the commonest form. Asthma in many cases was regarded as a pure "neurosis," due to paroxysms of tonic spasm of the circular muscles in the small bronchi (Trousseau, Biermer). The sudden contraction of the bronchi and the consequent dyspnœa could, in fact, be very well explained in this way. The only thing left unexplained is the conditions causing the sudden onset of such a spasm.

Many recent observations (Voltolini, B. Fränkel, Hack, etc.) have rendered it extremely probable that, in at least a part of the cases, reflex processes play a great part, and especially reflex processes from the nasal mucous membrane. Many patients with asthma have been found to have marked changes in the nose, most frequently enlargement of the so-called erectile bodies on one or more turbinated bones, also nasal polypi, chronic catarrh of the mucous membrane, etc. The evidence of the close connection between the attacks of bronchial asthma and those morbid processes rest, in the first place, upon the clinical fact that the asthmatic attack usually begins with a marked increase of the nasal symptoms (a stuffed nose, greater secretion, etc.); and, in the second place, especially upon the important therapeutic observation that in such cases the asthma may be made permanently to disappear by removing the nasal trouble.

Accordingly there can hardly remain a doubt that in such instances it is proper to speak of a reflex bronchial asthma. Certain observations which have long been familiar are probably connected with these facts we have mentioned—namely, the cases in which persons are said to have genuine asthmatic attacks from particular odors (ipecac, burnt coffee, violets, etc.). This is probably a reflex process coming from the nose. Similar relations doubtless exist in the so-called hay asthma (see page 135).

Although we may not question the occurrence of a reflex bronchial asthma, yet we can not regard the theory that it is caused by a tonic spasm of the bronchial muscles as absolutely proved. So far as our own experience goes, even in "reflex asthma" there are usually during the attack plain signs of an abnormal secretion or exudation in the smaller bronchi (expectoration, bronchitic râles, etc.); but this condition requires further explanation than by the assumption of a muscular spasm. We are therefore of the opinion that we should not wholly reject the hypothesis advanced by many authors (Weber, Störk, Fräntzel). According to this hypothesis, acute stenosis of the bronchioles is caused much less by a muscular spasm than by an acute dilatation of the blood-vessels and acute swelling of the mucous membrane. Such a "vaso-motor" change may very well be excited by reflex causes, and it would much better explain the conditions in an asthmatic attack than would the assumption of a purely muscular spasm.

It is doubtful whether genuine bronchial asthma may be due to a reflex from any other organ than the nose. The most credible statements are those in regard to the connection between bronchial asthma and disease of the pharynx (hypertrophy of the tonsils), but the statements in regard to the occurrence of asthmatic attacks in diseases of the ear, stomach ("dyspeptic asthma"), intestines, female sexual organs, etc., are to be regarded with the greatest skepticism. In these cases there is usually confusion between true asthma and other conditions of dyspnœa (hysterical asthma, conditions of cardiac weakness, etc.).

Although, from what has been said, it follows that the occurrence of a nervous reflex asthma can not be questioned, our knowledge as to the onset of nervous asthma as a primary affection is still very vague. At any rate the cases which, after careful investigation, allow no other explanation are very rare. It is also doubtful whether we have to do with a primary bronchial spasm or with vasomotor conditions. The theory formerly maintained by many writers, that a tonic spasm of the diaphragm could be the cause of a true asthmatic attack, is at present almost universally abandoned. This theory is contradicted by the occurrence of manifest respiratory movements of the diaphragm during the asthmatic attack. These movements have also been demonstrated of late by the Röntgen rays.

Apart from the reflex asthmatic conditions, most cases of so-called "bronchial asthma" are undoubtedly due to a primary catarrhal inflammation of the smallest bronchi and bronchioles; but since not even catarrh of the finer bronchi causes typical asthmatic attacks, we must assume that in asthma we have to do with a special form of bronchiolitis or at least with special attendant conditions which cause the asthma. Curschmann, to whom we owe the first accurate clinical investigation of this most common form of bronchial asthma, termed the bronchial affection, which forms the basis of it, "exudative bronchiolitis," but we have ourselves for a good while usually employed the name of "asthmatic bronchiolitis." We would indicate by this term a peculiar disease of the smaller bronchi and bronchioles whose characteristic features are a peculiar sort of expectoration (*vide infra*) and characteristic "asthmatic" (*vide infra*) attacks of dyspnœa which grow worse paroxysmally. It is indeed possible, but not certain, that in the latter regard nervous reflex processes in the bronchioles also play a part, somewhat like the spasm of the glottis in whooping-cough. The causes of this asthmatic exudative bronchiolitis are not always evident. Sometimes, however, circumstances apparently play a part similar to those producing ordinary bronchitis, especially the continuous inhalation of vegetable dust (wool, meal, etc.). Many cases of bronchial asthma may also with great probability be referred to some severe bronchial disease in childhood (whooping-cough, severe bronchitis of measles, etc.).

Symptoms and Course of the Disease.—We will begin the account of the symptomatology by a description of the asthmatic "attack." In its purest form "nervous" bronchial asthma consists, in fact, of single attacks of dyspnœa, differing in frequency and duration, which occur at least without special cause or without any discoverable reason in persons otherwise apparently healthy. In the intervals between the attacks the patient is perfectly well, and, in particular, shows no signs of disease of the respiratory organs. In most cases of "bronchial asthma," however, the attacks, as we have said, are only more or less sudden exacerbations of a condition which in the intervals is not perfectly normal. While ordinarily only the signs of a chronic bronchitis are present, often associated with pulmonary emphysema, from time to time exacerbations occur, usually in the form of long-continued asthmatic dyspnœa, lasting for days or weeks. It is this latter form especially which can be explained only by the assumption of a true bronchiolitis.

The asthmatic attack either begins quite suddenly, or is preceded for a shorter or longer period by prodromata. These consist in a general feeling of discomfort, in abnormal sensations in the larynx or epigastrium, sometimes in remarkably frequent gaping, and often in a marked coryza associated with a good deal of secretion and frequent sneezing (compare the relation between many attacks of asthma and diseases of the nose, given above). The attack begins in most cases at night (before midnight). The patient wakes up with an intense feeling of pressure and anxiety. Sometimes he complains of a feeling of pain in the chest. He has to sit up straight, and in severe cases even to get out of bed. He often hurries to an open window in order to "get air." His expression is anxious; his skin becomes pale and cyanotic, and sometimes is covered with a cold sweat. On objective examination we are at once struck by the characteristic change in the respiration. Both inspiration and, especially, expiration are almost always accompanied by a high-pitched whistling sound, audible at a distance. Both respiratory acts are labored, requiring the aid of the accessory muscles. On inspiration, only the upper part of the thorax is elevated to any extent. We see in the neck the inspiratory contraction of the sterno-cleidomastoids, the scaleni, etc. Still more striking, however, is the labored, panting, long-protracted expiration, during which the abdominal muscles are contracted to a board-like hardness. We therefore recognize the disturbance of respiration in asthma as essentially an expiratory dyspnœa. The frequency of respiration is in many cases normal, or even somewhat diminished, yet we have repeatedly counted thirty or forty respirations a minute.

On physical examination of the lungs during the paroxysm, we find the percussion-note over them normal or even strikingly loud and deep—the "box-tone." The lower boundary of the lung is usually found one or two intercostal spaces lower than normal. Not only is this condition shown in the cases with a permanent pulmonary emphysema, but during the asthmatic attack itself there occurs an acute inflation of the lung. The latter is probably explained by the fact that the lung is much stretched by the strenuous inspirations which are made by aid of the accessory muscles, while the weaker expiratory force is not enough wholly to drive out the air again through the contracted bronchioles. Therefore it happens in bronchial asthma, as in every other bronchial disease, that the expiration is usually more labored and protracted than the inspiration. On auscultation, high-pitched whistling and creaking sounds, which quite obscure the vesicular murmur, are heard over most of the lung, especially during the long expirations. In many places, indeed, where the bronchioles are almost completely closed, the respiratory murmur is entirely absent, or we hear only a low whistle on expiration. Toward the end of the paroxysm the noises become deeper and more booming, and sometimes we hear a few moist râles.

In brief paroxysms there may be scarcely any cough or expectoration. In most, particularly in the tedious cases of true asthmatic bronchiolitis, there is, however, a scanty tough mucous expectoration. In this are found, beside the ordinary constituents of simple bronchitic sputum, larger or smaller numbers of very characteristic clumps. These may be yellow or greenish-yellow, or, on the other hand, gray. The yellowish masses, which are usually very tough, and often consist of a bunch of thready matter, represent swollen and fatty-degenerated pus corpuscles, between which are very frequently interspersed a considerable number of pointed octahedral crystals. These crystals were first described by Leyden in the sputum of asthmatic patients, and are usually termed asthma crystals (see Fig. 25). Chemically they are identical with "Charcot's crystals," which are found in the leukæmic spleen, the bone marrow, etc., and they probably represent the phosphoric-acid salt of an organic base (Schreiner's base, C_2H_5N), although this has lately been questioned. As soon as the paroxysms cease the number of crystals in the sputum usually begins to diminish rapidly, and it is often possible to observe in them evident signs of disintegration. Nothing is known as to the origin of these crystals. Often, also, numerous ciliated epithelial cells are found, in addition to the crystals, in the yellow masses. The gray plugs in the sputum of asthmatic patients consist mainly of clumps of thready mucus, and contain the peculiar "spirals" which were first described by Ungar and by Curschmann. Many of these spiral threads are visible to the naked eye, but others demand the microscope for their recognition, through which they are seen as brilliant forms composed entirely of various sized bands and threads collected in spirals (see Fig. 25). Sometimes a brilliant central thread of small diameter is seen in the midst of the spiral. Around the spirals are found round cells, drops of fat and myeline, and sometimes ciliated epithelium, and epithelial cells from the pulmonary alveoli. As to the precise way in which the spirals and their central thread develop, the question is not yet settled, but it is certain that the spirals represent casts of the minutest spirally twisted branches of the bronchi, and therefore clearly indicate the existence of a peculiar disease of the finest terminal bronchial twigs.

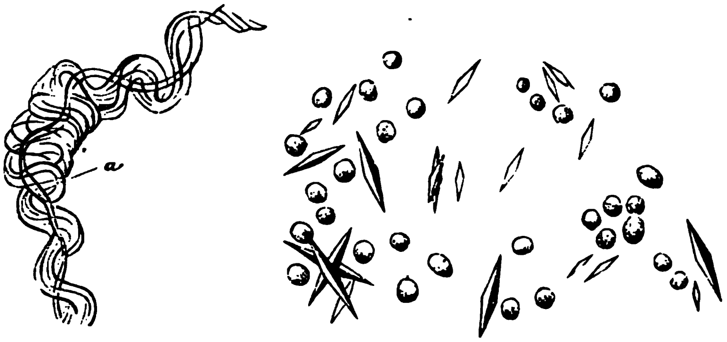

FIG. 25.—Asthma crystals and Curschmann's spirals (*a*, central fiber).

Of the other peculiarities of the sputum in bronchial asthma we may mention, first of all, the almost invariable occurrence of very many eosinophilous cells in the sputum, and also, apparently, in the blood. The significance of this fact is still unknown. We occasionally find in the sputum of asthmatics crystals of calcic oxalate and calcic phosphate.

The pulse is usually accelerated during the asthmatic paroxysm, the arteries contracted; the bodily temperature is normal, or sometimes even subnormal. In protracted attacks we have repeatedly seen a slight febrile movement up to about 102° (39° C.).

The duration of the asthmatic paroxysm is very different in individual cases, as has already been said. Sometimes it lasts only a few hours, but sometimes it lasts several days, and even weeks. Marked exacerbations and remissions of the disease usually alternate. The frequency of the paroxysms in ordinary asthma also varies exceedingly. Sometimes they come on almost every night, and then there are long pauses of months and years, so that we can not make any general statements as to the course of the disease. Many asthmatic patients make very remarkable statements as to the individual exciting causes of their attacks. Many patients claim, for example, that the attacks occur only in certain places, while in other places they are wholly free from the trouble, that they can live only in the upper storeys of the house, etc. Such statements should not remain unheeded, although it is certain that they are often due to imagination. Definite recoveries are quite rare, since long-continued asthma almost always leads to chronic pulmonary emphysema with all its consequences.

Diagnosis.—The diagnosis of the bronchial asthmatic condition as such is not difficult if we limit ourselves strictly to the characteristic type of the disease: the strenuous respiration with wheezing that can be heard at a distance, the labored, prolonged expiration, the characteristic physical signs in the lungs, and the peculiar sputum. Usually we can easily distinguish bronchial asthmatic dyspnœa from cardiac asthma (*q. v.*), spasm of the glottis (*q. v.*), and also from hysterical dyspnœa with its superficial and very rapid respiration and normal conditions in the lungs. This latter distinction of true bronchial asthma from "hysterical asthma" is very important both in prognosis and treatment, since the two are frequently confounded. If we are sure that we are dealing with true asthmatic attacks, the next question is whether it is a "purely nervous" reflex asthma or an asthmatic exudative bronchiolitis. Here, of course, we can decide only after a careful and thorough examination of the patient (especially of the nasal cavities) and after observing the course of the disease. Finally, we must also consider the possibility of a purely symptomatic asthma in chronic pulmonary emphysema, in the chronic bronchitis of renal disease or arthritis, etc. We must, however, confine our use of the term "symptomatic bronchial asthma" to those cases in which we actually have a dyspnœa with all the characteristic peculiarities of true bronchial asthma.

Treatment.—In every case of asthma the first thing to be thought of is whether there is not a definite cause whose removal may cure the disease. In this connection we should examine the nose carefully, since numerous recent observations have shown that a previously existing asthma may wholly disappear or at least be materially relieved after the treatment of some nasal disease which may be present, such as the removal of polypi, the destruction of the erectile bodies by the galvano-cautery, etc. Of course these results should not be over-estimated. It has repeatedly been claimed by nasal specialists that in many asthmatic patients we can find a definite spot in the nose and excite an asthmatic attack by touching it with a probe. This spot must then be treated first of all. We will not wholly reject these statements, but we are somewhat skeptical in regard to them. At any rate, the nose should be treated, in our opinion, only when it presents actual morbid changes.

If we can not satisfy the causal indication in this way, we should always try next a remedy which must pass for a direct specific against certain forms of asthma—iodide of potassium. In doses of twenty to forty-five grains a day (grm. 1.5–3.0), or even more if necessary, this usually causes a rapid improvement, which of course is not always, although it is frequently, permanent. In asthmatic bronchiolitis, especially, iodide of potassium in large doses often acts excellently. It seems to make the tough secretion more fluid, to ease the expectoration, and in this way to lessen the stenosis of the bronchioles. Of course a permanent and complete cure is only rarely obtained from iodide of potassium. If iodide of potassium has been used in vain, we must turn to the other remedies which have been employed against asthma, although their action is often quite uncertain. We may mention here the nitrite of sodium (two parts in one hundred and twenty of water, two to three teaspoonfuls a day), and nitro-glycerine, which has an analogous action (twenty drops of a one-per-cent. alcoholic solution in six and a half ounces [grm. 200] of water, a tablespoonful two or three times a day); also bromide of potassium, belladonna, atropine, etc. In some cases pneumatic treatment, such as the inhalation of compressed air, has been successful, and sometimes, too, electricity (galvanization and faradization of the neck), or hydrotherapy, has been claimed to give relief. Change of climate may sometimes be of distinct service. Many patients bear the sea-air well, while with others a mountain residence exerts a favorable influence. Special resorts (Marienbad, Kissingen, ferruginous baths, etc.) may often be recommended with advantage if appropriate to the patient's general constitution.

In severe cases a special symptomatic treatment of the attack itself is often necessary. Narcotics are without doubt the most effective, especially chloral and morphine. In severe attacks we can not avoid injections of morphine, but we must always be cautious in order that the patient may not form the habit of using this to excess. Chloral hydrate (gr. xv–xxx, grm. 1.0–2.0) often relieves the individual attack. Among other symptomatic remedies we may mention mustard-plasters to the chest and calves, putting the hands and feet into hot water, inhalations of nitrite of amyl, turpentine, chloroform, pyridine, etc. Fumigation with saltpeter paper—unsized paper dipped in a concentrated solution of nitrate of potassium and dried—and the stramonium cigarettes to be had in most drug-stores, are much praised. The patient may also smoke with benefit stramonium or belladonna leaves which have previously been dipped in a solution of saltpeter and then dried. Many patients praise highly the different asthma cigarettes and smoke-producing powders or pastilles, which are sold as secret remedies, especially in America. Such apparent success is certainly due in great measure to the psychical suggestion wrought by these remedies.

[Potassic iodide is more likely to prevent recurrence if it is given continuously, for several months at least, and it should not be thrown aside as useless until it has been pushed to the limit of toleration without avail. A convenient form of administration is in saturated aqueous solution, a minim of which represents about a grain of the drug.

The syrup of hydriodic acid may be substituted for potassic iodide in cases of intolerance of the latter. Grindelia robusta, a drachm of the fluid extract three or four times a day, serves sometimes to prevent recurrence of attacks. Marked alleviation of the paroxysms is often obtained from the inhalation of fifteen to thirty drops of the iodide of ethyl.]

SECTION IV

DISEASES OF THE LUNGS

CHAPTER I

PULMONARY EMPHYSEMA

(Alveolar Ectasis. Pneumatosis pulmonum)

Nature and Ætiology of the Disease.—Pulmonary emphysema, the abnormal inflation and dilatation of the lungs, is one of the commonest pulmonary affections. It either develops in separate parts of the lung, in which case it is subordinate to other pathological changes which co-exist in the lungs, or it involves almost the whole extent of both lungs, and then presents the symptoms of a characteristic affection, which it is usually easy to recognize.

The essence of pulmonary emphysema, the condition from which most symptoms are immediately derived, is the loss of elasticity in the lungs. If we compare the sound lung, with its normal elastic force, to a new and very tense rubber band, the emphysematous lung must be compared to an old and lax band that is stretched and pulled out. We therefore see why the emphysematous lung takes up a greater space than the sound one, for, on account of its lack of elasticity, it can no longer contract to its former volume. We may therefore call emphysema a permanent inspiratory distention of the lung from which it can no longer return to its expiratory condition. If we open the thorax of a subject with normal lungs, they contract, as is well known, but the emphysematous lungs remain in their inflated condition after the thorax has been opened.

If we inquire into the factors which cause this loss of elasticity in the lung, we find that they are the same kind of influences which tend to diminish the elasticity of any other elastic body. As a rubber band, by much pulling and stretching, gradually gets longer and less elastic, so the lungs, as a result of their abnormally frequent and severe distention, gradually become inelastic and emphysematous. Emphysema in many cases is a true wearing out of the lung. The normal traction of inspiration, which is continually making new demands on the elastic powers of the lungs, finally leads to a loss of elasticity in them. In advanced age most lungs become more or less inelastic. The lungs of an old man are like an elastic band which has done its work for years but which has finally given out. We therefore class emphysema of the lungs in old age rather among the states of involution, such as develop in almost all organs in advanced life, than among special pathological changes. We distinguish, moreover, most lungs with senile emphysema from other emphysematous lungs by the fact that their volume as a whole is not increased, but is rather diminished below that of the healthy lung, since we find in them the extensive atrophic processes of old age.

The condition becomes pathological, however, if the elasticity of the lung is deficient in earlier years and independently of the action of the special injurious influences which will soon be mentioned. In such cases of emphysema, developing in middle life or even in youth, the idea of a congenital weakness of the elastic elements in the lungs can not be set aside. It probably consists in a quantitative or a qualitative defect of the elastic tissue. Some observations seem to corroborate the statement that a disposition to emphysema may be present in several members of the same family.

If a lung whose elasticity is previously subnormal can not persistently satisfy the ordinary demands upon it, a normal lung, on the other hand, also loses its elasticity if the demands made upon it are greater than it can perform. This is the reason why pulmonary emphysema is in some instances to be regarded as a disease arising from the occupation. We mean here not only those influences which lead to chronic bronchitis and thus later to emphysema (*vide infra*), but more especially the abnormal demands upon the lungs in all those callings which necessitate severe physical labor. We must not only regard the deeper and more rapid respirations, but also the increased pressure during expiration to which the lungs are often exposed in the raising of heavy weights, etc. This explains the common occurrence of emphysema in the laboring classes, and also its greater frequency in men than in women. Besides this, we must add that in certain callings, such as glass-blowing and horn-blowing, the over-straining of the lungs is much more direct. In all such cases emphysema may be termed simply a premature exhaustion of the lungs.

In very many cases emphysema develops as a result of other diseases of the lung, and especially as a result of chronic bronchitis. Dry catarrh of the medium-sized and finer bronchi when of long duration leads, as a rule, to pulmonary emphysema. The abnormal mechanical influences to which the lungs are thus exposed act both in inspiration and in expiration. Since the entrance of air to the alveoli is rendered more difficult by the swelling of the mucous membrane in the smaller bronchi, abnormally deep and strong inspirations are necessary, with a marked expansion of the alveoli, in order to draw a sufficient quantity of air into the alveoli. The alveolar walls are therefore exposed to an abnormal traction at each inspiration. On expiration, a pressure from within, which is perhaps even more injurious, acts on the alveoli. The ordinary expiration, which usually needs only the elastic power of the lungs, is not sufficient in chronic bronchitis to drive the air out of the alveoli through the narrowed bronchi. Thus arise the difficulty and delay in expiration which are present in chronic bronchitis, and which lead to the active participation of the muscles of expiration, the abdominal group of muscles. On forced expiration, however, the pressure does not act simply upon the contents of the alveoli, but much more upon the smaller bronchi themselves. The channel of exit for the air from the alveoli, therefore, becomes still narrower. Since the air can not at once escape, the pressure within the alveoli is raised by the efforts at expiration, and the alveolar wall is thus again abnormally expanded. The cough, which is often present in chronic bronchitis, is a further factor, which acts in a precisely similar injurious fashion. The attacks of coughing begin with a forced contraction of the muscles of expiration, which follows the closure of the glottis. Until the glottis opens, therefore, the lower parts of the lung especially are put under strong pressure. The air in them, which can not escape outward, is driven into the upper parts of the lung, and there leads to expansion of the alveoli, and finally to emphysema.

We accordingly see that a number of injurious influences co-operate in the gradual development of emphysema from chronic bronchitis, and that, sooner or later, these influences have as their result the gradual dilatation of the lungs. Here, too, we must bear in mind the individual differences in the resisting power of the lungs.

Conditions precisely similar to those in chronic bronchitis occur in other diseases, and lead in like manner to pulmonary emphysema. We very often see the development of emphysema in severe and persistent whooping-cough. The worst factor here, besides the existing bronchitis, is the frequent paroxysms of coughing. Many cases of pulmonary emphysema and chronic bronchitis may be ultimately referred to such a severe bronchial disease occurring in childhood. We have already mentioned, in the description of bronchial asthma, both the acute emphysema, which occurs during the attacks, and the final development of a permanent emphysema.

In conclusion, we must here consider a theory advanced by Freund, which would make the development of an emphysema dependent upon a "primary rigid dilatation of the thorax." It is indeed conceivable that from certain pathological changes in the costal cartilages, as Freund claims, a thorax, which had become fixed in the position of inspiration, might exert a constant abnormal traction on the lungs and so give rise to an emphysema. The occurrence of this hypothetical primary disease of the cartilages, however, has up to the present time not been established. It is rather considered by the majority of authors as a secondary change, developing as a result of emphysema or else simultaneously with it. On the other hand, it is certainly remarkable that we sometimes observe in children the "emphysematous habit" of the thorax and neck, which will be more fully described further on, and that in fact we can often discover in such children a beginning emphysema early in life. We might perhaps imagine a congenital failure of harmony between the size of the thorax and the size of the lungs, whereby the latter are from the start in constant overtension.

Besides the already described essential or substantial emphysema, which is a special disease attacking both lungs uniformly, we distinguish a so-called vicarious or complementary emphysema. If, by any disease, certain portions of the lungs are incapacitated in their functions, the parts which remain healthy must then assume the whole business of respiration. They become excessively expanded on inspiration, and as a result they become emphysematous. Thus we see emphysema of the upper lobes in affections of the lower lobes. Emphysema of one lung is most frequently observed clinically when the other lung is extensively diseased, especially in unilateral chronic contractions of the lungs and pleuræ, usually seen in tuberculosis. Vicarious emphysema may also be confined to quite small portions of the lung, but then it is merely of pathological and not of clinical interest.

Pathological Anatomy.—As we have seen, the actual abnormality of the lung in emphysema is not due to a pathological change, but to a change in its physical conditions. The loss of elasticity of the lung is shown in its greater volume, in its lack of contractility, and in its persistence in a position of inspiration.

The single alveoli are of course just as much expanded as the whole lung, but their walls show at first no histological changes. We have here, then, a condition which Traube has called "increased volume of the lungs," and has distinguished from the "pulmonary emphysema" proper. This distinction is without doubt justified anatomically, but clinically it can not well be maintained. As the distention is constant, the alveolar walls can not withstand the constant traction and pressure. This leads to progressive atrophy of their tissue from pressure—that is, it leads to a real disappearance of the elastic elements of the lung. The atrophy begins quite gradually. The partition-walls of the alveoli are first perforated, and then they partly or wholly break down. The neighboring alveoli run more and more into one another, and thus finally arise alveolar ectasis and infundibular ectasis, which can be made out with the naked eye, and which may attain a diameter of five or ten millimetres or more. If single air-bubbles enter the interlobular, interstitial, or subpleural connective tissue, which may happen perhaps in severe fits of coughing, we speak of an interstitial or interlobular emphysema, in distinction from the ordinary vesicular or alveolar emphysema.

The tissue atrophy in the septa of the alveoli affects not only the elastic tissue, however, but also the branches of the pulmonary capillaries in the alveolar walls. The affection of the elastic tissue adds no new conditions to the disturbed functions of the emphysematous lung, which we have just described. The destruction and final atrophy of the pulmonary capillaries, however, is the second important factor in the pathology of pulmonary emphysema, for, with the destruction of so great a part of the vascular area in the lungs, the outflow from the right side of the heart is considerably impeded. There must therefore necessarily be a stasis in the pulmonary arteries and the right side of the heart, and the right side of the heart can overcome the increased resistance only by increased work, and thus in every chronic pulmonary emphysema there finally arise a dilatation and consecutive hypertrophy of the right ventricle with their further consequences.

Symptoms and Course of the Disease

General Course of the Disease.—Although a pulmonary emphysema may sometimes, as in whooping-cough, develop in a comparatively short time, still its course is always very chronic. In most cases the origin of the disease is quite gradual, as in all those instances in which emphysema develops from chronic bronchitis or asthma, or as a result of some injurious occupation. The symptoms gradually and insidiously associate themselves with those of the chronic bronchitis.

The symptoms of emphysema usually begin in middle or advanced life, but marked emphysema may occur in youth and childhood. The disease always lasts for years, unless some fatal intercurrent disease arises.

The objective and subjective symptoms are due either to the chronic bronchitis, which very often co-exists, or to the emphysema itself. Not only is the bronchitis, as we have seen above, very often the cause of emphysema, but, on the other hand, the development of chronic bronchitis is greatly favored by the circulatory disturbances in the lung associated with emphysema. Thus the two diseases, emphysema and chronic bronchitis, are closely connected clinically.

Bronchitis causes its well-known symptoms—cough, expectoration, moderate dyspnœa, and a feeling of pressure in the chest. The bronchiectases, which are often gradually formed, especially in the lower lobes, may lend a peculiar stamp to the cough and expectoration (see page 178). Emphysema increases the patient's dyspnœa to a degree which can never be caused by chronic bronchitis alone. The emphysematous lungs soon become incapable of satisfying any extraordinary demands of respiration. Many patients are only slightly conscious of the difficulty in breathing so long as they keep quiet, but whenever they make a trifling physical exertion, go up-stairs, or take a little longer walk than usual, the dyspnœa comes on.

The variations in the intensity and extent of the bronchitis correspond to the frequent and quite marked variations in the patient's feelings. These variations depend upon the condition of the patient, his circumstances, and the possibility of his taking care of himself ; the change of seasons, too, has an influence on him. In pleasant weather many patients live in tolerable comfort, but autumn and winter bring an aggravation of all their symptoms with the increase in their bronchitis.

The last stage of the disease is characterized by the appearance of disturbance of compensation in the heart. We have seen above that the cause of the impairment of the pulmonary circulation, and of the resulting hypertrophy of the right ventricle, is the closure of numerous pulmonary capillaries. A further reason for the impairment of the circulation comes from the disturbance of respiration itself, since the influence of the respiratory movements on the circulation is well known. The appearance of a marked disturbance of the circulation may be deferred for some time by the increased efforts of the right ventricle. The cyanosis of most patients, however, is due not only to incomplete oxidation but to the blood-stasis which extends backward from the right side of the heart into the veins of the body. Finally, the right ventricle becomes more and more feeble, the stasis in the veins increases, œdema of the extremities and transudation into the various cavities of the body ensue, and after long suffering the patient succumbs to dropsy.

Emphysema is frequently combined in its later stages with other chronic diseases. Pulmonary emphysema with its sequelæ is seldom found at the autopsy as a single lesion, but we discover in the cadaver co-existing disease of the heart, the blood-vessels, or the kidneys, all a genuine wearing out of the aging body. Pulmonary tuberculosis is often a final development in emphysema, but it is usually of the chronic indurated form, and is not very extensive.

Physical Examination. 1. *Inspection.*—In many patients we can detect the disease with considerable confidence at the first glance; we are therefore justified in speaking of an emphysematous habit. The patients are usually quite well nourished, at least in the early stages of the disease, and are often rather corpulent. They appear plump or even somewhat bloated, and their faces are more or less markedly cyanotic. The configuration of the neck and thorax is especially characteristic. The neck is usually short and thick; the sterno-cleido-mastoid muscles, which have to act as auxiliaries in inspiration, are tense and hypertrophied. The inspiratory contraction of the scaleni may also be seen and felt. The veins in the neck are visibly dilated, and in severe cases are swollen to thick blue cords, and we sometimes see in them evident undulating or pulsating movements. The thorax is rather short, but broad and strikingly deep—the "barrel-shaped thorax." The intercostal spaces are narrow, and the lower ribs incline only a little downward. The epigastric angle is therefore obtuse, and sometimes becomes almost a straight line. The respiratory movements are almost always accelerated in severe cases. Inspiration becomes short and labored. The excursions of single ribs are therefore slight, and the thorax is raised rigidly and more as a whole. Expiration is visibly prolonged. There may be a noticeable retraction of the intercostal spaces on inspiration, in the lower and lateral portions of the thorax.

This characteristic form of the thorax in emphysema is regarded as a constant inspiratory position of the ribs, and corresponds to the permanent inspiratory dilatation of the lungs. The peculiar rigidity of the thorax is probably due to the changes in the costal cartilages already described, which, according to Freund, are primary. In many cases the emphysematous form of the thorax gradually develops in the course of the disease, but in other cases it seems to depend on some original predisposition to the disease (*vide supra*).

In conclusion, we must state that the above description corresponds to the typical form of emphysema, from which we may have many deviations. In the paralyzed thorax, for instance, we may meet with a high degree of essential emphysema of the lungs, which has often given rise to errors in diagnosis.

2. *Percussion.*—Percussion gives very decided results in the diagnosis of pulmonary emphysema. We find the inferior border of the lungs one or two intercostal spaces lower than under normal conditions, corresponding to their permanent inspiratory inflation. Clear pulmonary resonance on the right front in the line of the nipple extends to the lower border of the seventh, and sometimes of the eighth rib. On the left front it extends to the fifth or sixth rib, so that the cardiac dullness is lessened. The area of cardiac dullness can often not be made out at all; or at most, on strong percussion, it is made out in a limited extent as relative dullness. The pulmonary resonance extends on both sides in the back to the first or second lumbar vertebra. These results of percussion in emphysema, however, are frequently altered, because other conditions, such as passive congestion of the liver, meteorism, and ascites, may be present at the same time, and push up the diaphragm. Thus the detection of emphysema by percussion is made decidedly difficult.

Qualitative changes in the percussion-note may be entirely wanting in emphysema. The pitch is sometimes remarkably loud and deep—the "box-tone" [tympanitic resonance]; but in other cases, especially in the back, we find it somewhat raised. This may depend in part upon the poor vibratory conditions in the rigid chest-walls, but in other cases it is caused by the retention of an abundant secretion in the lower lobes.

On inspiration, the lower edge of the lung moves downward very little or not at all. This is an important sign in diagnosis. Since the lung is always in a state of abnormal inspiratory distention, and since the entrance of air is impeded by the accompanying catarrh of the bronchi, the difference between the inspiratory and expiratory expansion of the lung is much diminished. The difference caused by respiration in the lower limit of the lung affords a good measure of the respiratory disturbance in the lower lobes.

The detection of dilatation and hypertrophy of the right ventricle by percussion is in many cases difficult, because the lungs cover the heart. A positive result can be obtained only by carefully defining the relative cardiac dullness [by very light percussion]. The epigastric pulsations frequently seen in emphysema, and also the marked undulating and pulsating movements in the jugular veins, are to be regarded as quite certain signs of dilatation of the right side of the heart.

3. *Auscultation.*—The characteristic auscultatory sign of emphysema is the prolonged expiration. As a flabby rubber band, when it is stretched and then let loose, no longer snaps back quickly and strongly, so the emphysematous lung, when it has been stretched in inspiration, comes back again only slowly. We hear with it a somewhat aspirated, sonorous sound, which plainly exceeds the vesicular inspiratory sound in duration. The vesicular murmur itself often undergoes a modification in pulmonary emphysema. It sounds exaggerated, and shuffling, or in other cases it is rougher and more indefinite. In a high degree of emphysema the vesicular respiration is sometimes very faint and obscure, because the inspiratory current of air is reduced to a small amount in the lungs, which are already excessively dilated. In many cases we hear rhonchi beside the respiratory murmur, dry whistling, buzzing, and creaking sounds on inspiration and expiration. If cylindrical bronchiectases have already formed, we hear, especially over the lower lobes, numerous fine and medium moist râles, but no sonorous rhonchi. The adventitious sounds may wholly conceal the respiratory murmur. With a marked retention of secretion we sometimes hear nothing but a low, suppressed rattling.

In the heart the sounds are usually rather feeble, because it is covered by the lung. The "functional systolic murmur of emphysema" at the apex, described by some writers, we have heard much less frequently than we should expect after the statements relating to it. If present, it is probably due to co-existing valvular changes. The pulmonic second sound in emphysema is, as a rule, markedly accentuated, as a result of the stasis in the pulmonary circulation.

The diminution of the expiratory pressure in emphysema may be measured with the manometer, or with Waldenburg's "pneumatometer." The normal expiratory pressure of 110 to 130 millimetres sinks in emphysema to 100 or 80 millimetres. As we should expect, the spirometer shows a diminution of the vital lung capacity, which can be readily explained. The normal lung capacity of about 3,500 cubic centimetres falls to 2,000 or 1,000 cubic centimetres.

OTHER SYMPTOMS IN THE LUNGS AND IN OTHER ORGANS

In regard to the other symptoms in the lungs we have only a little to add to what has already been said. The intensity of the cough naturally varies in individual cases according to the degree of the existing bronchial catarrh. Many patients are troubled by a dry cough, while others have abundant expectoration. There is nothing characteristic of emphysema in the composition of the latter. All the kinds of sputa which are found in the different forms of chronic bronchitis are also found in pulmonary emphysema. The dyspnœa, whose predominant expiratory character we have already mentioned, increases in advanced cases to a most marked degree. Sometimes the increase shows itself by the appearance of distinctly paroxysmal dyspnœa. This is often really to be regarded as a symptomatic bronchial asthma, but, on the other hand, we must not overlook the fact that a temporary increase of the bronchitis, retention of secretion, and cardiac failure, may also excite attacks of dyspnœa, which can not properly be termed asthma.

The important changes in the heart resulting from emphysema have already been described. The exhausted right ventricle can no longer overcome the increased resistance in the pulmonary circulation. The difficulty of respiration is still greater, from the passive congestion of the pulmonary vessels. The skin becomes still more cyanotic, and finally œdema and general dropsy develop. The failure of compensation is indicated by the smallness of the pulse, its increased frequency, and sometimes by its irregularity. The difficulty of an objective examination of the heart in emphysema has been spoken of above.

The appearances of blood-stasis in the internal organs are shown especially in the liver and kidneys. The liver is swollen, and its increase in size (the liver of passive congestion) can frequently be made out by percussion or palpation. The pains in the hepatic region, of which many patients complain, are perhaps due sometimes to the stretching of the capsule of the liver, but they are probably more often muscular pains excited by the frequent coughing.

In the kidneys the effect of stasis is first shown by a diminished excretion of urine. The urine is more scanty in amount, more concentrated, of a higher specific gravity, and of a darker color. It generally gives an abundant sediment of urates, and it may contain a small amount of albumen. Microscopically we may find a few hyaline casts, and a few red and white blood-corpuscles. It is evident that this diminished activity of the kidneys favors the development of dropsy.

The spleen is not infrequently found congested at the autopsy. The evidence of this, however, is often uncertain during life, for percussion of the spleen is difficult on account of the emphysema, and palpation is difficult from the anasarca.

Gastro-intestinal symptoms may be present in emphysema. The appetite seldom remains good throughout the disease. Many patients suffer from chronic constipation; and more rarely there is a tendency to diarrhœa.

Fever is not present in simple pulmonary emphysema. Whenever fever exists for a long time it depends on other complications, such as severe bronchitis, pneumonia, or tuberculosis.

Complications of emphysema with other chronic diseases are frequent. The old opinion that emphysema and tuberculosis, and emphysema and chronic heart disease, were antagonistic to each other is entirely false. These complications are not very rare. We may also mention the complication with general arteriosclerosis and with chronic nephritis, especially the contracted kidney. Among acute diseases we must mention particularly the acute accidental pulmonary complications, to which emphysematous patients, particularly of the lower classes, are much exposed as a result of over-exertion, exposure to cold, etc. Acute febrile bronchitis and lobular pneumonia cause marked exacerbations, and attacks of true influenza and croupous pneumonia in aged patients are not infrequently dangerous to life.

The **diagnosis** of emphysema can be made directly from the results of the physical examination, and usually presents no difficulties. We think it important merely to say that too much stress should not be laid upon a low position of the lower edge of the lung alone. Many men apparently have dilated lungs but no symptoms at all. The chief thing, therefore (besides the dilatation of the lungs), is the presence of delayed and difficult expiration and lessened power of inspiration. The diagnosis is difficult when a patient with emphysema is not examined until the final, dropsical stage. In such cases it is often very difficult to avoid confusion with heart disease (primary hypertrophy, myocarditis, mitral stenosis), contracted kidney, etc. It is often difficult, also, to come to a decision in those cases in which there are evident signs of co-existing cardiac or renal disease besides the emphysema. In such cases it is often hardly possible to decide upon which of the different organic changes the chief stress is to be laid. In all such cases we must place especial confidence in an accurate history as well as a careful physical examination. The special nature and the sequence in development of the individual symptoms often afford valuable data for a correct appreciation of the whole clinicial picture.

Prognosis.—Pulmonary emphysema of acute origin—that is, acute inflation of the lung—like that resulting from whooping-cough and analogous affections, may be recovered from in many cases. Otherwise, as regards the final curability of the disease, the prognosis is wholly bad. The duration of the disease and the intensity of the symptoms are of course very different in individual cases. Here almost everything depends upon the circumstances in which the patient is placed. With sufficient care the disease may be tolerably well borne for many years, but without it the first symptoms of respiratory and cardiac insufficiency appear much earlier. The final termination is usually due to complications (*vide supra*).

Treatment.—Since emphysema itself is only slightly amenable to treatment, most of our therapeutic remedies are directed to that accompanying condition upon which the greater part of the symptoms depend—the chronic bronchitis. If we succeed in improving this, or even in wholly removing it, we always obtain a decided improvement in all the patient's symptoms. The therapeutic agents mentioned in the description of chronic bronchitis are therefore of frequent use in emphysema.

In the first place, we must seek the best hygienic conditions for the patient, and remove him from all injurious influences, such as dust, bad air, and great physical exertion. In dry catarrh we should use the alkaline mineral waters, and when there is abundant mucous secretion the balsams, such as turpentine internally and by inhalation. The most valuable expectorants are iodide of potassium, apomorphine, and ipecac when the expectoration is tough, and liquor ammonii anisatus and senega when it is abundant. Their action, of course, too often fails of the desired result, so that we frequently have to change our remedies. When there is a troublesome cough, disturbing the sleep, we can not dispense with narcotics, such as morphine, codeine, or Dover's powder. If severe dyspnœa comes on, we may try to obtain relief by mustard plasters to the chest, or by immersing the hands and feet in hot water. With asthmatic attacks we may try iodide of potassium, besides the other remedies mentioned for asthma. Here, too, we must finally resort to narcotics.

We must carefully watch the condition of the heart, and use digitalis when there are signs of beginning disturbance of compensation and the pulse grows small and irregular, and this drug may prove very useful. If symptoms of dropsy set in, we may sometimes prescribe diuretic remedies, such juniper-tea, acetate of potassium, diuretine, calomel, etc., besides digitalis. When there is persistent weakness of the heart we should also use digitalis and other stimulants (strophanthus, camphor, wine, etc.).

Besides the purely symptomatic treatment thus described, the attempt has been made to meet the causal indications in emphysema, and especially to aid the patient in expiration, and thus to improve the power of the lung to contract, where it is possible. To this end Gerhardt has recommended assisting expiration mechanically by compression of the thorax. This compression must be done methodically by another person,* about five or ten minutes every day, by the aid of both hands laid flat on the lower lateral portions of the thorax. The effect of this manipulation in diminishing the dyspnœa and making expectoration easier is in many cases very satisfactory. The "breathing chair" made by Rossbach has a similar mechanical action.

The employment of the pneumatic treatment has also become quite general, especially since the introduction of Waldenburg's portable apparatus. The expiration into rarefied air, which meets the causal indication, may procure great relief for the patient in many cases, and sometimes, too, may result in an improvement of the emphysema which can be demonstrated on physical examination. Inhalations of compressed air are also employed when there is severe bronchial catarrh. Still, too much must not be anticipated from pneumatic treatment.

CHAPTER II

PULMONARY ATELECTASIS

(Compression of the Lungs. Aplasia of the Lungs in Cases of Kyphoscoliosis)

Ætiology.—Atelectasis of the lungs is a condition the direct opposite of emphysema. While in the latter the lungs are abnormally inflated, in the former they are abnormally collapsed. The air has disappeared from the alveoli and lesser bronchi, and in the most advanced cases even from the larger bronchi. The atelectatic portions of the lung are not altered histologically, but are changed to a firm tissue, deprived of air—so-called splenization or carnification.

The atelectasis of the new-born is due simply to deficient respiration and to the consequent imperfect entrance of air into the lungs. In weak children, who die soon after birth, we often find the lower lobes wholly or in part in a fœtal, uninflated condition—that is, atelectatic. By artificial inflation we can readily expand the lungs to their normal extent. In many cases of weak, newly-born infants there is at first an atelectasis of parts of the lung, which gradually disappears entirely and is replaced by normal conditions.

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* One of the author's patients at the policlinique in Leipsic made himself a very simple but very effective apparatus for producing this compression of the thorax on himself by the aid of two narrow boards, which are fastened together at one end by a cord of suitable length. These boards, which are furnished with a pad at this end fitted to the wall of the chest, are laid flat on the two sides of the thorax so that their free ends project forward some six inches or a foot, and serve as a one-armed lever. By pressing them together the patient himself can thus, without any strain, exert a considerable pressure on his thorax with each expiration.

Acquired atelectasis occurs in two ways. We may mention, as the first and most frequent ætiological factor, the plugging of the smaller bronchi. If a complete closure of a bronchus arises from the accumulation of secretion, as may easily happen in the narrow bronchi of children, the air can no longer enter, on inspiration, into that portion of lung supplied by the plugged bronchus. The air which is shut up in it is gradually absorbed by the blood. The adjacent parts of the lung expand, and the portion that is excluded from respiration collapses, leaving a circumscribed pulmonary atelectasis, usually rich in blood but devoid of air. Such atelectases, in greater or less number and extent, are very often found in the bodies of children who have suffered from severe bronchitis, especially after measles, whooping-cough, or diphtheria. Besides the direct action of the plugging of the bronchus, the weakness of the respiratory movements and the cough, conditional upon the general state of the disease, play a significant part.

The second very frequent and important cause of pulmonary atelectasis is compression of the lung. In all the diseases which diminish the space for the expansion of the lungs, the lungs are compressed to a greater or less extent, whereby the air is squeezed out of them. Thus arise the atelectases from pressure in pleuritic effusion, hydrothorax, pneumothorax, in marked cardiac hypertrophy, pericardial effusion, and aneurism of the aorta. Atelectasis of the lower lobes also arises in the same way from great upward pressure on the diaphragm by ascites, meteorism, abdominal tumors, etc.

That form of pulmonary atelectasis which arises from deformities of the thorax is of great practical importance. In severe kyphoscoliosis, the half of the thorax corresponding to the convexity of the vertebral column is much narrowed. The lungs are materially hindered in their expansion, and even in their growth, if the deformity occurs in youth. This is called "aplasia of the lungs," a condition which may give rise to grave results (*vide infra*).

Symptoms.—In the majority of cases the symptoms of atelectasis are subordinate to the disturbance caused by the primary disease. This is especially the case in most of the atelectases from pressure, although the most dangerous factor lies in the compression of the lung.

The atelectasis of the lungs developing as a result of diffuse capillary bronchitis, especially in children, can of course not be detected by physical examination until it is of great extent. The respiration, in extensive formation of atelectasis, often shows a very striking and characterstic deviation from the ordinary type, especially when the atelectasis develops in the lower lobes. It is accelerated and labored, and is performed chiefly by the upper and anterior portions of the thorax. In the lower portions we see marked inspiratory retractions, which are caused in part by the external pressure of the air, and in part by the exaggerated contraction of the diaphragm.

Physical examination can, of course, reveal abnormal conditions, especially dullness on percussion, only when the atelectasis is extensive. Dullness, however, is usually hard to make out in children. Auscultation gives signs of existing bronchitis; and sometimes, too, with more extensive consolidation, there is bronchial respiration. In other cases, as may be easily seen, the respiratory murmur is much diminished or wholly absent. Thus, the physical signs of atelectases are not easily distinguishable from those of pneumonia, especially of lobular pneumonia. In fact, a sharp distinction between atelectatic nodules and nodules of lobular pneumonia in the lung can not be drawn clinically.

Aplasia of the lungs in kyphoscoliosis demands a special description, because it is of great practical significance. Many patients with kyphoscoliosis may live for years without special respiratory disturbance. Careful observation, of course, usually shows a somewhat labored and hurried respiration, but the patients have not paid much attention to it. In other cases the difficulty in breathing is more noticeable. The person affected is incapable of any severe physical exertion; he always feels short of breath, and often suffers from cough and expectoration. In the cases first mentioned, however, which for years have had little or no trouble, disturbances in respiration sometimes come on quite suddenly. They may also frequently arise without any special cause, and may attain a very threatening degree. The condition may improve, or it may lead to comparatively or even absolutely sudden death. Examination of the lungs during life usually shows nothing but the signs of an extensive bronchitis. By careful percussion we may quite frequently detect an increase of cardiac dullness to the right. Sometimes a moderate œdema develops. In such cases the autopsy shows nothing as the cause of death but the changes in the chest. The lungs are deficient in air, small, and compressed, but in circumscribed portions, on the contrary, emphysematous and expanded. The right side of the heart in the great majority of cases is dilated and hypertrophied. There can scarcely be a doubt, therefore, that the cause of the onset of severe symptoms and the final cause of death is to be sought in the cardiac failure.

Finally, it is worthy of mention that there is a frequent form of mild atelectasis in the lower lobes, which occurs in very sick and bed-ridden patients who usually keep in one position—on the back—as in typhoid fever. On making such patients sit up we hear during the first inspirations exquisite crepitant râles over the lower lobes, which sometimes disappear after a few deep inspirations. Here we have to do with a mild atelectatic condition, with a temporary collapse of the alveoli and smallest bronchi.

The **treatment** of atelectasis coincides in great measure with the treatment of the primary disease, and is therefore to be looked for in the corresponding chapters. The prophylaxis of atelectasis, by constant attention to the respiration, is of great practical importance. We should try to keep the patient from lying continually on his back, and we should make him take deep inspirations. The timely use of tepid baths, with shower-baths, is a special preventive of the development of atelectasis, and it may bring about a recovery when atelectasis is already present.

Tepid baths may also be used with care in the treatment of dyspnœa caused by kyphoscoliosis. The condition of the heart, however, deserves especial attention (stimulants and digitalis). The reader is referred to the consideration of the general treatment of circulatory disturbances in the chapters on diseases of the heart. In other respects the symptomatic treatment by expectorants, etc., is the same as in other chronic pulmonary affections.

CHAPTER III

PULMONARY ŒDEMA

Ætiology and General Pathology.—We have in pulmonary œdema the exudation of a highly albuminous fluid, usually somewhat hæmorrhagic, not only into the interstitial tissue, but also into the alveoli themselves. The danger of the condition is easily understood from the high degree of dyspnœa which immediately ensues from it. In fact, pulmonary œdema is in many cases a terminal symptom, which comes on in all forms of acute and chronic disease. Many patients are said to die with the signs of pulmonary œdema, especially patients with heart disease, pulmonary and renal disease, and also with other affections of the most different kinds.

In rare cases pulmonary œdema is a transitory symptom. Repeated attacks of it may occur, especially in heart disease and chronic renal disease, and, for a time at least, the patient recover from them.

In spite of much clinical and experimental work, the special causes of pulmonary œdema are still quite obscure. For one class of cases the work of Cohnheim and Welch shows that pulmonary œdema is to be considered as purely the result of stasis. Pulmonary œdema takes place when the outflow of venous blood in the lung meets an obstacle which can no longer be overcome by the mechanical force of the right ventricle. The obstacle which plays the most significant part here, and which may occur in all possible forms of disease—of course more readily in those mentioned above than in others—is paralysis of the left ventricle. If the further progress of the blood is much hindered by this, the overfilling of the pulmonary circulation and a consequent pulmonary œdema will necessarily follow, in spite of the most vigorous action of the right ventricle. Many cases of terminal pulmonary œdema seem to depend upon this fact, that the left ventricle is paralyzed in its action sooner than the right.

The paralysis of the left ventricle, however, is certainly not the only factor to be considered in the origin of pulmonary œdema. We must also consider the condition of the walls of the blood-vessels in the lungs. In many cases, especially in renal disease, pulmonary œdema seems to depend upon local changes in the vessel walls (Sahli). This form of pulmonary œdema is somewhat akin to true inflammatory œdema. The latter is found chiefly in the vicinity of portions of lung infiltrated with pneumonia; it is usually of limited extent, and therefore it is of subordinate importance as a cause of disturbances in respiration compared with the diffuse œdema of stasis.

In very rare cases, as we have ourselves seen, an apparently primary acute pulmonary œdema, with a speedily fatal termination, develops in men who are apparently perfectly healthy, and the autopsy gives no explanation of its origin. We perhaps have to do in these cases with the sudden failure of the left ventricle, but probably with acute vascular changes as well.

Symptoms.—Marked dyspnœa is the most striking symptom in pulmonary œdema. It is subordinate only when the patient is found in the death agony and is no longer fully conscious.

In pulmonary œdema the respiration is hurried, labored, and rattling. All the accessory muscles of respiration are called into play. The patient usually sits upright in bed. We see on his lips and cheeks a gradually and constantly increasing cyanosis, and we often hear at a distance the moist râles originating in the larger bronchi.

On examination of the lungs, the percussion is essentially normal, if there is no other disease of the lungs. Sometimes the percussion-note is a little higher in pitch, and often it is slightly tympanitic. On auscultation, we hear everywhere many fine and medium moist râles. If the patient can still expectorate, he raises a large amount of frothy, sero-sanguineous sputum. The whole picture of the disease is so characteristic that the condition can scarcely be mistaken.

Treatment.—Since in most cases pulmonary œdema is not so much the cause as a symptom of approaching death, our remedies against it are apt to prove powerless, but it must always be our duty, at least in all cases that are not absolutely hopeless, to try to relieve the pulmonary circulation. From the pathogenesis of pulmonary œdema it follows that we must pay particular attention to the condition of the heart, especially of the left ventricle. Hence we should use energetic stimulants, especially subcutaneous injections of camphor or ether, every half hour or hour. Internally we give first strophanthus (ten drops of the tincture every hour) and also camphor, wine, and very strong *café noir*. Besides that, we apply strong irritants to the chest, such as large mustard plasters or hot sponges. Sometimes an actual improvement of the respiration, when it has nearly stopped, may be obtained by a bath with cold douching. When there is marked general cyanosis, if the patient is on the whole strong and well nourished, venesection is sometimes of manifest benefit. Emetics, however, accomplish little, and are even dangerous on account of the collapse which is apt to come on after them. Expectorants (benzoin, liquor ammonii anisatus) are more frequently prescribed, and an energetic "derivation to the intestines," by senna, calomel, or enemata of vinegar, seems sometimes to be really of service. Acetate of lead in large doses, one or two grains (gramme 0.05–0.10), in powder, every hour, employed empirically by Traube, is deserving of trial.

In this way, especially in acute diseases like typhoid and pneumonia, we in fact sometimes succeed in averting the danger of pulmonary œdema by rapid and energetic action. In the cases of œdema occurring in incurable chronic diseases of the heart and kidneys, the remedies employed are of course unfortunately incapable of preventing death.

CHAPTER IV

CATARRHAL PNEUMONIA

(*Broncho-pneumonia. Lobular Pneumonia*)

Ætiology.—Catarrhal pneumonia is neither ætiologically nor anatomically one single, absolutely independent disease, but from a clinical standpoint we are perfectly justified in grouping together the catarrhal, lobular pneumonias, which are usually secondary to other diseases, and especially to a previous bronchitis, and in contrasting them with croupous, lobar, "genuine" pneumonia. In the great majority of catarrhal pneumonias the belief is certainly correct, that the agents of inflammation do not enter the pulmonary alveoli directly from without, but that the inflammatory process is situated originally in the bronchi, and from this point extends downward to the special respiratory parenchyma of the lung. This extension of the inflammation may take place entirely by continuity, or it may skip some part, as the germs are often inhaled from the bronchi directly into the infundibula and alveoli. The last-mentioned parts must, however, possess considerable power of resisting germs, since the danger of an extension of bronchitis to the alveoli generally exists only in the severer and more extensive cases of bronchitis or under other peculiar conditions. This extension of the process is not uniform in all parts, but it occurs first in the distribution of some small branches of the bronchi, and this is the reason why the pneumonic infiltration affects first only a few bronchial areas—that is, individual lobules.

In opposition to this generally accepted theory of the method of origin of the true "lobular" or "broncho-pneumonic" foci, the attempt has recently been made to show that the inflammatory process can pass through the walls of a small bronchus directly into the neighboring parenchyma of the lung, and that it may then extend further along the lymph-channels. Clinically, however, we can not yet distinguish this form of focal pneumonia from the ordinary catarrhal pneumonia.

If we ask what are the conditions under which we are most apt to observe the development of lobular pneumonia, we must first mention a number of acute infectious diseases, in which the air-passages are primarily affected, or in which they may very readily be involved sympathetically. Chief among these are measles and whooping-cough, and next diphtheria, influenza, small-pox, etc. In all these diseases there is either a bronchitis at the beginning, or else it can develop very easily in them. It is in these same diseases, also, that simple bronchitis comparatively often develops into lobular pneumonia.

Furthermore, in almost all severe acute and in many chronic diseases the conditions favor the development of secondary bronchitis and subsequently, at times, of lobular pneumonia. Everywhere in the air-passages, as well as in the cavities of the mouth and pharynx, saliva, mucus, etc., readily collect if the patient is very ill. Expectoration is imperfect, and the constant dorsal decubitus favors the accumulation of secretion, especially in the lower lobes. The mouth and pharynx are harder to keep clean than under normal conditions. Fungi and bacteria collect in the secretion itself, as well as in the epithelium and particles of food which are left in the mouth, and these excite and keep up processes of decomposition. The inflammatory agents, which are carried into the air-passages with the inspired air, find everywhere favorable conditions for settling and further development. From the upper portions they are drawn farther downward. From the larger bronchi the process invades the alveoli, and leads to catarrhal pneumonia. It is probably of significance, too, that in such very sick persons the vital resistance of the tissues has suffered, and that the development of such secondary inflammation is consequently facilitated. We must also bear in mind that many patients who are very ill have difficulty in swallowing. They get choked, and particles of food, with the germs of inflammation clinging to them, are carried into the air-passages. These particles, which a healthy person could easily cough up again, remain there, are decomposed, and give rise to bronchitis and lobular pneumonia.

This is the explanation of the frequent development of lobular pneumonia in the course of diseases which are entirely dissimilar. We observe it especially in severe bed-ridden cases, in all patients with stupor, in severe infectious diseases, in meningitis, and also in cases of nervous disease, in which coughing and deglutition are impaired as a result of bulbar affections. In all such cases lobular pneumonia is to be considered a complication, and with reference to its origin deserves the name of inhalation pneumonia or deglutition pneumonia. We shall soon see that this form, under some circumstances, may pass into circumscribed gangrene.

Although in the preceding we have always spoken of "agents of inflammation" in general, the precise variety is not necessarily the same in all cases. In the lobular pneumonias occurring in the course of measles, whooping-cough, influenza, etc., it is possible that the original specific germs may penetrate as far as the alveoli, and there cause an inflammatory exudation; but this is certainly not always the case, and there are many reasons why in these affections, as well as in all the others mentioned above, catarrhal pneumonia should be regarded as a secondary complication, dependent upon the secondary invasion of other varieties of agents which cause inflammation. Different micro-organisms may probably act as such agents. So far as our present investigations go, streptococci seem most frequently to be the special cause of broncho-pneumonia, and sometimes in other cases staphylococci, diplococci, etc. Clinically, we can not at present make a complete separation on a strictly ætiological basis.

The development of lobular pneumonia from bronchitis is most frequent, as we know, in children and old people. The frequency of catarrhal pneumonia in childhood is due in part to the limited dimensions of the bronchi. Besides that, however, the diseases in which it is especially frequent—namely, measles and whooping-cough—are children's diseases. In old people its comparatively easy development is due to imperfect expectoration, and perhaps to the diminished resistance of the tissues.

The mild cases of primary bronchitis scarcely ever lead to lobular pneumonia, but sometimes in children, and less often in adults, a severe febrile bronchitis may occasion the formation of pneumonic foci. In Erlangen, the author has seen a good many cases which can not be regarded otherwise than as primary catarrhal pneumonia. We may also state that the inhalation of irritating chemicals may occasion lobular pneumonia as well as bronchitis.

Pathological Anatomy.—It is characteristic of catarrhal pneumonia that the inflammation is usually plainly circumscribed (*vide supra*), being limited to the territory of a small bronchus. Hence the name of "lobular" pneumonia or broncho-pneumonia, in distinction from croupous lobar pneumonia. An atelectasis (*vide supra*) of the affected lobule, arising from the plugging of the bronchus leading to it, often, but not always, precedes the inflammation. The atelectasis of course becomes pneumonia only when germs enter the atelectatic spot. The inflammatory process itself consists of the exudation of a scanty fluid, which usually does not coagulate, and of numerous pus-corpuscles (white blood-corpuscles) into the lumen of the alveoli. With this is usually associated more or less marked desquamation of the alveolar epithelium, in which we often find necrosis or fatty degeneration. The alveolar cavities are completely filled with leucocytes and epithelium; few if any red blood-corpuscles are to be seen, although in some cases they may be more abundant. The vessels of the alveolar walls are hyperæmic, and the connective tissue also contains a few wandering leucocytes.

The inflamed lobules are readily apparent to the eye and the touch by their firm consistence, being devoid of air. Their color at first, from the blood contained in the inflamed part, is a dark red ("splenization"), but later it becomes more grayish. A bit of such an inflamed spot, cut out with the scissors, does not float in water but sinks, because it contains no air. The lobular boundary of the individual nodules is usually easily distinguished from the neighboring healthy tissue, but, by confluence of adjacent nodules, large portions of the lung, and even whole lobes, may become infiltrated throughout—generalized lobular pneumonia.

Symptoms.—1. **Primary Catarrhal Pneumonia of Adults.**—The primary catarrhal pneumonia which infrequently occurs in adults usually begins with the same phenomena as a severe attack of acute bronchitis. The patient feels prostrated, and has cough, dyspnœa, and pain upon the side which is chiefly affected. There is almost never a pronounced initial chill, as in croupous pneumonia. The fever is usually not very high, from 101° to 103° (38.5° to 39.5° C.), but higher temperatures sometimes occur, especially at the beginning of the disease. The expectoration is simply catarrhal or muco-purulent, never muco-sanguineous, as in croupous pneumonia. The physical examination gives almost invariably in one lower lobe many moist râles and a slight tympanitic or dull tympanitic percussion-note. Marked dullness and definite bronchial breathing are rare. In the lower lobe on the other, unaffected side, we often find signs of a slight bronchitis; but in general the unilateral character of the symptoms is characteristic of primary catarrhal pneumonia, in distinction from ordinary bronchitis and secondary broncho-pneumonia. In mild cases the fever lasts from four to eight days, but the disease may last two or three weeks or more. There is never a crisis; the fever ends gradually by lysis.

The ætiology of primary catarrhal pneumonia has been little studied. Many cases may be streptococcus pneumonia; we must also consider the influenza bacilli, since catarrhal pneumonia is especially common at the time of an influenza epidemic.

2. **Secondary Catarrhal Pneumonia.**—Most of the cases of catarrhal pneumonia develop, as we have already said, secondarily in the course of other affections, hence the symptoms are frequently overshadowed by those of the other diseases. There are often found at autopsy a few foci of lobular pneumonia in the lower lobes which gave rise to no clinical symptoms whatever.

In other cases, however, the development of extensive lobular pneumonia is of the greatest clinical significance. In many cases of chronic bronchitis, pulmonary emphysema, pulmonary tuberculosis, etc., in which the patient suddenly grows worse with a rise of temperature, the cause is certainly the development of foci of lobular pneumonia. Such complications may after a time completely disappear, or may cause a permanent aggravation of the original condition (e. g., tuberculosis). The onset of lobular pneumonia in other acute diseases is very important. The disturbance of respiration, during the patient's life, forms the most striking symptom of the disease, and lobular pneumonia is shown at the autopsy to be the immediate cause of death. The largest part of the fatal cases of measles and whooping-cough, and no very small part of those of diphtheria, scarlet fever, typhoid, small-pox, influenza, etc., are due, in the last instance, to the disturbance of respiration dependent upon lobular pneumonia.

Since a diffuse bronchitis, extending into the finer bronchi, almost always precedes the development of lobular pneumonia, and since it may also give rise in itself to marked disturbance in respiration, there is no sharp boundary to be drawn clinically between diffuse capillary bronchitis and lobular pneumonia. Only the experience, a hundred times repeated, that extensive capillary bronchitis is apt to lead to lobular pneumonia, permits us to suspect the latter, with considerable certainty, even if there is no direct clinical evidence of it. The most important objective physical sign of broncho-pneumonia is the not very loud, medium moist râles, almost always to be heard in the lower lobes. These râles are often high-pitched. Dullness of the percussion-note develops only when many lobular nodules coalesce and form a more extensive infiltration of the lung. Then we also find bronchial respiration.

Catarrhal pneumonia is almost always associated with fever, which is moderately high and generally has a remitting character. We must, of course, judge of the fever in connection with the primary disease and any other complications.

No general statements can be made as to the duration of secondary catarrhal pneumonia. Sometimes the acute symptoms last only a few days, in other cases they continue for weeks. Catarrhal pneumonia is not infrequently followed by pleuritic effusion, when the lobular infiltration reaches the pleura. Foci of lobular pneumonia may develop into abscess or gangrene, but this is rare.

3. **Catarrhal Pneumonia in Childhood.**—The catarrhal pneumonia of childhood is very characteristic and clinically important. It is observed most frequently in measles and whooping-cough, and also in weak, atrophic, and rachitic children. The increased frequency of respiration is most striking. The breathing is superficial, but labored, as is shown by the contraction of the auxiliary muscles of inspiration and the play of the nostrils. We also notice inspiratory retraction of the lower lateral portions of the thorax as a result of the incomplete entrance of air. The number of respirations in a minute increases in children to sixty or eighty, or even more. In most cases the child has a frequent and apparently painful cough. Expectoration is entirely absent in small children. When it is present it shows no characteristic peculiarities different from ordinary catarrhal sputum. The general condition is always bad. The child is restless, apathetic, and more or less stupid. Its face is usually pale, but often quite cyanotic. The pulse is very rapid, and in small children may attain a frequency of 140 to 180 a minute. Fever is almost always present. It shows no typical course, it is now remitting and now intermitting, and toward evening

it perhaps rises to 104° or 105° (39.5°–40.5° C.). The occurrence of such a rise in temperature is not without value in the diagnosis of catarrhal pneumonia. If in diffuse capillary bronchitis a high fever is present for some time, we may assume with considerable certainty that the lobular infiltration has already begun.

Physical examination furnishes direct evidence of the affection of the lungs, but its results are for the most part to be referred to the diffuse bronchitis and not to the lobular infiltration. Auscultation gives the most valuable signs. We hear over the lungs, in a greater or less extent, numerous fine and medium moist râles, often quite high-pitched. From these signs, strictly interpreted, we can diagnosticate merely bronchitis, but we may suspect pneumonia with the greatest probability. With very confluent broncho-pneumonia, auscultation sometimes gives bronchial breathing and bronchophony, besides the râles.

It goes without saying that little lobular foci, surrounded by normal lung-tissue containing air, give no special signs on percussion. With numerous nodules running into one another, the percussion-note is duller, and there is sometimes tympanitic resonance. The dullness is often first to be made out over a stripe extending along the vertebral column—so-called "stripe-pneumonia."

An attack of extensive lobular pneumonia is usually quite protracted. Even in favorable cases the disease rarely lasts less than two or three weeks, and it may persist much longer. The course of the disease is apt to be irregular, relapses succeeding improvement. The chief danger of the disease lies in this tendency to a protracted course, extending over weeks and months. Many children finally die, not of the lobular pneumonia itself, but from the general weakness and emaciation following the tedious febrile disease. We must remember, however, that complete recovery may sometimes take place quite late in the disease.

The "transition of catarrhal pneumonia to caseation and tuberculosis" is a clinical fact with which physicians have long been conversant. In fact, we often find true tubercular changes in the lungs of children who have died after a tedious illness, as a result of measles, whooping-cough, etc. There can, of course, be no real question, however, of an actual transition from one disease to the other. In such cases either we have to do with an acquired tubercular infection, which has found a favorable soil in an already diseased lung, or (what is probably more frequently the case) the disease of the lung has promoted the development of a previously existing tuberculosis. It is usually weak children, with a hereditary predisposition to tubercle, who succumb to tuberculosis as a result of the above-named diseases. The diagnosis of a developing tuberculosis is not always easy, since it is only rarely that marked phthisical changes—like dullness at the apex, cavities, etc., which can be made out by a physical examination—are found in the lungs. We can usually suspect tuberculosis only from the general conditions (emaciation, persistent hectic fever, hereditary predisposition, or some secondary tubercular disease such as meningitis, etc.), especially as absolute proof, from the detection of tubercle bacilli in the sputum, is only rarely possible in children.

Treatment.—Since we have already mentioned the proper treatment, in our description of the various diseases in which secondary pneumonia is especially prone to develop, we can now be brief. We have also laid repeated stress upon the possibility and the great practical importance of prophylaxis, which is self-evident from a just comprehension of the origin of lobular pneumonia. Besides keeping the nose, the mouth, and the pharynx as clean as possible, tepid baths, perhaps with cool douching, are the best means of preventing the development of lobular pneumonia, or of checking its further extension. Wet cold packs are often used with advantage (*vide infra*). It is an advantage, which is indeed to be considered in the second rank in comparison with the improvement in respiration, that by both the bath and the pack the febrile temperature is at the same time reduced.

In the treatment of the lobular pneumonia of children a wet pack including the whole body is the best remedy. A sheet is dipped in water, wrung out, and wrapped around the whole of the patient except his head and arms. Outside of this is to be placed a dry woolen blanket or a layer of oiled muslin. The temperature of the water employed should be 68° to 77° (16° to 20° R.). The higher the fever the colder should the water be, and the oftener, say every hour or two, must the pack be renewed. In milder cases and at night it may be allowed to remain for three or four hours. The beneficial influence of the pack is shown not only by the temperature, but still more by the respiration. It is often striking to see how much quieter the child becomes in the pack. If the breathing, despite this remedy, remains unsatisfactory, and the patient becomes more and more stuporous, the treatment must be changed to lukewarm baths of a temperature of 77° to 86° (20° to 24° R.), with douchings of colder water. It is sometimes advisable in severe cases to add to the water employed for bathing or for the wet pack a few handfuls of mustard. The stimulation thus exerted upon the skin is quite marked.

Among external applications to the chest, besides mustard plasters and poultices, dry cups are to be mentioned, which often do very good service in strong, older children, and especially in adults. We never need to use local blood-lettings, however, in catarrhal pneumonia.

Of internal remedies, expectorants are most used. Chief among these are ipecac, apomorphine, senega, and benzoic acid. This last is particularly useful in the lobular pneumonia of children. In strong children the abundant collection of mucus in the bronchi may sometimes be relieved by the administration of an emetic, but we seldom need to resort to this. We should also be cautious in the use of narcotics. Stimulants (camphor, wine) must be used in severe cases. Antipyretics may be given to reduce the temperature, but if cool packs be used antipyretics can be wholly dispensed with. Inhalations or sprays are quite valueless in lobular pneumonia, yet it is recommended to keep the air in the sick-chamber constantly moist by hanging up wet towels, or by sprinkling with water. The room should also be as large and as well ventilated as possible. The general hygienic treatment is of the greatest importance. One of the most important duties, of which the physician must always be mindful, is to keep up the patient's strength by sufficient and proper food. When convalescence sets in, complete restoration to health may be materially furthered by going to a suitable place in the country.

CHAPTER V

CROUPOUS PNEUMONIA

(Lung Fever. Lobar Pneumonia. Fibrinous Pneumonia. Pleuro-pneumonia)

CROUPOUS pneumonia is a sharply defined febrile disease of the lungs, which, in the great majority of cases, displays a distinct individuality in its clinical, anatomical, and usually, also, its ætiological relations. Among the severe acute diseases it is decidedly one of the most important and frequent, and it is universally familiar even to the laity under the names of pneumonia or inflammation of the lungs. In most cases this disease appears quite suddenly, and often, apparently without any special cause, in persons up to that time in perfect health. Such cases are described as primary, genuine, or frank pneumonia. On the other hand, a case of croupous pneumonia will sometimes occur as a complication of all sorts of other diseased conditions (secondary pneumonia). The clinical picture of pneumonia in these latter cases, however, is usually obscure and not well characterized, and consequently the following description is especially applicable to the primary form of the disease:

Ætiology.—The thought that croupous pneumonia might be an acute infectious disease had impressed the majority of physicians for a considerable length of time, but this suspicion did not receive satisfactory substantiation until the more recent investigations in bacteriology had been made. For instance, Friedländer demonstrated a special form of bacillus in lungs affected with pneumonia. Then, later, A. Fränkel, and, soon after him, Weichselbaum, proved that while this "pneumonia bacillus of Friedländer" is perhaps to be regarded as the cause of croupous inflammation of the lungs in a few cases, yet in the overwhelming majority of instances the particular ætiological factor is the so-called diplococcus pneumoniæ (*diplococcus lanceolatus capsulatus*).

The pneumonia diplococcus is characterized by its lancet-shaped outline ("like the flame of a candle"), and its frequent arrangement in pairs, the individuals of each pair having usually their broad ends apposed. Very often there are short chain-forms. The pairs of diplococci are usually surrounded by a delicate capsule. This is particularly common in preparations of the sputum. We have not space to describe particulars as to the pure cultures of the pneumococci upon agar and similar media.

Pneumonia diplococci are among the most widely distributed of pathogenic organisms. They produce severe disease not only in the lungs, but in many other organs. With regard to the development of the pneumonic infection, it is a particularly interesting fact that these diplococci have been not infrequently found in the mouths of healthy persons. This suggests the thought that the germs are drawn into the lungs with the inspired air, and there settle and proliferate when the conditions are especially favorable—for instance, if the resisting powers of the organism have been impaired, or perhaps if the diplococci are especially virulent. In animals the injection of diplococci into the lungs almost always excites pneumonia, but yet it is doubtful whether the incidence of the disease in man always bears out the surmise above mentioned. The severity of the constitutional disturbance at the very onset of many cases certainly justifies the supposition that at least often the infection takes place through the blood, and that the pathogenic organisms are carried to the lungs by way of the blood-vessels. Diplococci have been repeatedly found in the blood of pneumonia patients, and the injection of diplococci into the subcutaneous cellular tissue of animals, particularly rabbits and mice, invariably excites a violent and fatal constitutional infection (so-called sputum-septicæmia). Certain clinical facts suggest that perhaps in individual cases there may be other modes of infection; for instance, by way of the intestine in cases with well-marked intestinal symptoms. Starting from the lungs, the diplococci may invade the pleura, less often the pericardium, the meninges, and other organs, so that they are invariably demonstrable in the pus of cases of meta-pneumonic empyema, pericarditis, and meningitis. From a clinical point of view their almost invariable presence in pneumonic expectoration (Fig. 26) is of the greatest importance. They can be easily demonstrated by the staining of a dried preparation with gentian-violet, although for absolute certainty of diagnosis further investigation is required. Supposing the infectious nature of pneumonia to be certain, all the other alleged causes may of course be regarded as at most "predisposing causes." The old opinion, which is yet current, that pneumonia is due to catching cold, is to be received with great limitations, for croupous pneumonia is very frequently seen independently of any such influence. In many cases it will be found that an exposure to cold immediately preceded the commencement of the disease; but in these instances the cold is probably to be regarded merely as that circumstance which promoted the occurrence of the infection, possibly because of the resultant injury to the bronchial and pulmonary epithelium. This explains the fact that pneumonia is especially frequent in certain classes, for instance, among day-laborers and soldiers. With regard to the so-called "traumatic pneumonia," the state of the case is similar to that of pneumonia due to cold. Patients from the laboring classes sometimes assert that they were taken ill as a result of heavy lifting or of a blow on the chest, but in such cases the subsequent stitch in the side was probably not the result of the injury, but a symptom of the disease which had previously begun to develop. But still, in some few cases it may be that a preceding trauma injures the pulmonary tissue in such a way as to promote the occurrence of the infection.

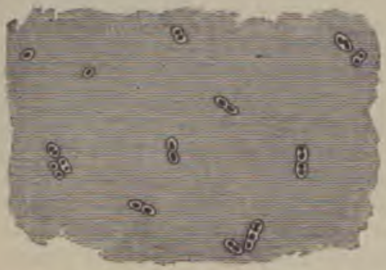


FIG. 26.—Pneumonia diplococci. (After Vierordt.)

It is a strong argument in favor of our conception of pneumonia as an acute infectious disease that it may rarely be endemic. Extensive endemics of pneumonia, usually of quite a malignant character, have been repeatedly observed in single buildings, especially in barracks or prisons, as well as in tenement houses and other localities. It is possible that precisely these severe cases of endemic pneumonia are ætiologically different from the ordinary croupous form and occasioned by some other pathogenic organism; but it is also true that the diplococcus of pneumonia itself appears to vary greatly in its virulence.

Pneumonia does not, as a rule, show a decided epidemic character. In a large population sporadic cases occur at any season. It has been observed, however, that the disease may become very frequent or almost completely disappear, and then again for a time be decidedly prevalent. In a limited way, therefore, it is quite proper to say that there are actual epidemics of pneumonia, and these, again, differ from one another in special peculiarities, particularly in their comparatively benign or malignant character. Here again, no doubt, an important part is played by the varying virulence of the diplococci which we have already referred to, which is also very evident under the influence of different modes of artificial cultivation of the organism. Most attacks occur in the winter or spring months, without any necessary relation, however, between the frequency of pneumonia and the occurrence of especially bad, wet, or cold weather.

Individual predisposition plays an unmistakable part in the disease, as we must suppose that it does in all infectious diseases. Like facial erysipelas and acute articular rheumatism, pneumonia is one of those diseases which is prone to attack the same individual several times. There are persons who have had acute pneumonia four or five or even more times in their lives.

We can not affirm with certainty that the liability to pneumonia is due to a special bodily constitution. The strongest and most robust often fall ill with it, and, on the other hand, weak and delicate people, with a tendency to phthisis, are frequently attacked. Drunkards seem to have a special predisposition to the disease, but of course it is exceedingly hard to give any definite statistics upon this point.

Pneumonia occurs at any time of life, most frequently in youth or middle age; but it is by no means rare in early childhood, and also in more advanced years up to sixty or seventy. In general it is observed rather more often in men than in women.

[Defective house drainage seems to be a predisposing cause of pneumonia in some cases. A careful inspection of the local sanitary conditions is desirable, especially where more than one case occurs in a house.]

Pathological Anatomy.—The anatomical process in croupous pneumonia consists in the formation of a hæmorrhagic, coagulable "fibrinous" or "croupous" exudation into the pulmonary alveoli and the smallest bronchi. The development of the exudation usually extends over one or more lobes to their whole extent, and, as the alveoli and finer bronchi are completely filled by the tough exudation, the spongy lung, filled with air, is changed to a firm tissue, devoid of air, except as it is penetrated by the large bronchi.

Since Laennec's day we distinguish three stages in the development of the process. In the first stage (stage of inflammatory engorgement, *engouement*) the lung is very hyperæmic, dark red, and the air contained in it is even now much diminished, but not entirely absent. The alveoli are filled with an abundant exudation, already hæmorrhagic, but still fluid and not coagulated.

In the second stage (stage of red hepatization) the coagulation of the exudation is complete, and the lung has become throughout of the consistence of liver. The hepatized lung shows a somewhat increased volume, and is strikingly hard. The surface of the section has a red and manifestly granular appearance, which is due to the projection of the numerous little fibrinous plugs situated in the alveoli. With the knife we can scrape off a tenacious, creamy, grayish-red fluid from the surface of the section. In the small bronchi, divided by the knife, we find characteristic tubular bronchial casts.

In the third stage (stage of yellow or gray hepatization), which gradually develops from the second, the red surface of the section changes to a yellowish-gray color, often mottled, while the lung grows anæmic and the exudation poor in red but rich in white blood-corpuscles. The consistency of the lung is still dense but more friable. The fluid scraped from the surface of the section is more abundant, milky, and puriform.

The recovery from the process begins as the exudation becomes fluid. The fluid is in part absorbed and in part coughed up.

It is not necessary for every pneumonia to go through all three stages completely. In mild cases the process may stop sooner and recovery begin.

Concerning the finer histological processes in croupous pneumonia, the primary change is probably to be found in the injury and partial destruction of the epithelium in the alveoli and smallest bronchi, produced by inflammation due to the specific causes of the disease. As in every croupous inflammation of a mucous membrane (see the chapter on diphtheria), a coagulable exudation is formed on the surface of the alveoli and smaller bronchi after the destruction of the epithelium. With the microscope we see the fibrinous net-work of the exudation filling the alveoli. Between its meshes lie numerous red blood-corpuscles—red hepatization. Where there is any of the alveolar epithelium left, we often notice active proliferation—increase and growth of cells. Later on the white blood-corpuscles increase, migrating from the vessels into the exudation—yellow hepatization. The red blood-corpuscles are dissolved unless they are removed by expectoration. The fibrinous exudation is also gradually dissolved as the result of chemical changes not yet clearly understood (peptonization of the albuminous substances?), and is absorbed like the cells. The regeneration of the missing epithelium comes from the epithelium that has remained intact, and with that follows a gradual and complete *restitutio ad integrum*.

The whole process is comparatively brief, usually running its course in a week or ten days. The most frequent termination is in complete recovery. The other methods of termination, as well as the complications in other organs, will be spoken of in connection with the clinical symptoms. We may here mention simply that the pleura over the affected portion of the lung takes part in the inflammation, without exception, as soon as the disease reaches the periphery, and a fibrinous pleurisy, which is not very intense, may then be recognized; hence the former use of the terms "pleuro-pneumonia" and "peripneumonia."

Croupous pneumonia usually spreads rapidly over a great part of the lung. It is very often quite sharply limited to a single lobe—"lobar pneumonia"—so that the septum of connective tissue between two lobes also forms a strict boundary between pneumonic infiltration and healthy lung tissue; but this boundary is by no means insurmountable, and quite frequently several lobes are wholly or in part attacked by pneumonia. According to all statistics, the lower lobes are more frequently affected than the upper. Isolated disease of the right middle lobe may occur, but it is much rarer than pneumonia of the upper lobes. Of the two lungs, the right is attacked with decidedly greater frequency than the left. We have ourselves seen, in 244 cases, 137 on the right, 86 on the left, and 21 in which both lungs were attacked to a great extent. Simultaneous affection of the lower lobe on one side and the upper lobe on the other—quite a rare occurrence—is termed "crossed pneumonia."

General Course of the Disease.—In spite of the numerous modifications which the course of pneumonia may undergo in individual instances, we can still call pneumonia a typical disease, considering the great majority of cases. The subjective and objective symptoms dependent upon the local affection of the lung usually, but not always, take the chief place among the clinical phenomena. In this, pneumonia differs from many other infectious diseases, such as typhoid, in which the local organic disease is subordinated to the general infection.

Pneumonia usually begins quite suddenly. In the majority of cases it starts with a pronounced chill of half an hour to an hour's duration, or at least with a marked and prolonged chilliness. The initial chill may attack the patient while in the best of health. Many patients are able to tell almost the very hour when, having been previously in perfect health, they were attacked by disease. The chill comes on in the daytime, in the evening, or even in the middle of the night, after a previously quiet sleep. At the same time the patient almost always feels as if a severe illness were beginning. Almost at once he is obliged to give up work, has violent headache, and loss of appetite. Not infrequently there is a single initial act of vomiting. Sometimes there are at once pulmonary symptoms, such as a stitch in the side and cough. Usually, however, these phenomena do not develop until later (*vide infra*).

In other and somewhat rarer cases the beginning of pneumonia is more gradual. A prodromal stage of a few days, or even longer, precedes the severe illness. The symptoms are either of quite a general and indefinite nature, consisting of malaise, dullness, loss of appetite, and headache, or the prodromal symptoms point more strongly to a pulmonary affection. The patient complains several days, or even weeks, before the onset of the severe disease, of cough, thoracic discomfort, slight dyspnœa, and similar symptoms. At the same time it is not possible to determine certainly whether these prodromata are caused by an already existing pneumonia or not. It is undoubtedly true that in most cases we have merely a simple bronchitis which furnishes a favorable soil for the development of pneumonia; but perhaps the initiatory bronchitis may, in some cases, itself be an effect of the diplococcus infection, already begun, but not yet completely developed.

The subjective symptoms in the chest begin shortly after the onset of the disease, often on the very first day, but in other cases later. The patient has a stabbing pain in his side whenever he draws a deep breath; the respiration, therefore, becomes superficial and accelerated, and often somewhat irregular; his speech is interrupted by frequent pauses. In the further progress of a severe case the dyspnœa becomes extreme and the respirations very frequent. With the stitch in the side is associated a desire to cough. The cough is painful, and hence short, half suppressed, and quite frequent and troublesome. From the second day the expectoration may assume its characteristic viscid, rusty, hæmorrhagic appearance. Physical examination gives on percussion and auscultation the signs to be described more fully below. These are rarely to be found on the first day, but more frequently on the second, and sometimes not till later.

In well-marked cases the severe constitutional symptoms persist or grow worse. We observe general weakness, headache, and complete loss of appetite. Sometimes there are marked nervous symptoms, such as restlessness, wakefulness, stupor, and delirium; herpes appears on the lips or nose; the bowels are sluggish, or, again, they may be loose; the urine is concentrated, and very often it contains a small amount of albumen.

Almost always the pneumonia is associated with high fever. The typical character of the disease and the peculiarities of the individual case are always well shown by the temperature chart. As the bodily temperature rises there is a corresponding increase in the frequency of the pulse.

The course varies greatly according to the previous individual circumstances, the severity of the disease, and the existence of complications. In the majority of cases, after a comparatively short duration, the disease takes a favorable turn. The beginning of improvement is often sudden, like the onset of the disease. After the symptoms have lasted for some five to seven days, or in rarer cases a shorter or a longer time, at a constant height or with increasing intensity, there occurs in the regular course of the disease a critical decline of the fever—frequently associated with quite a copious perspiration—and with that a very rapid improvement of all the other symptoms. In a short time complete recovery follows.

In other cases, however, the course is not so favorable. The disease may have a fatal termination. In a third small class of cases the disease finally takes a protracted course, which is usually due to the occurrence of sequelæ in the lungs or pleura.

To this brief sketch of the disease we must append a description of the special symptoms.

Description of Single Symptoms and Complications

1. **Symptoms on the Part of the Lungs.**—First among the subjective symptoms comes the characteristic painful feeling or "stitch" in the side. This probably always has its origin in the dry pleurisy which accompanies the pneumonia. It is therefore absent in the cases of central pneumonia (*vide infra*). In pneumonia of the lower and right middle lobes the pain is usually more severe than in pneumonia of the upper lobes. One result of the stitch in the side is the difficulty, or even the impossibility, of deep inspiration. Hence the patient's dyspnœa is considerably increased, and this explains the incongruity between the shortness of breath and the relatively slight extent of the pneumonia in many cases. If the pneumonic infiltration of the lung is extensive, of course the dyspnœa of the patient is also referable in part to the diminution of the respiratory surface. The subjective feeling of difficulty of breathing is prominent in the majority of cases, and it may become most distressing.

Cough is one of the most constant symptoms in pneumonia, and is usually very painful; hence the patient often tries to suppress it. Expectoration is apt to be very difficult at the onset of the disease, from the viscidity and scanty amount of the sputum; hence severe and distressing paroxysms of coughing are sometimes observed. The cause of the cough is probably not to be found in the affection of the alveoli, but in the co-existing bronchitis. The irritation of the pleura may also set up a reflex cough. In rare cases cough is entirely absent in pneumonia. Except in the cases of limited or late localization (*vide infra*), we observe this absence of cough chiefly in the pneumonia of old or very weak people, and also, what is of practical importance, in the drunkard's pneumonia associated with delirium tremens.

The pneumonic expectoration is so characteristic that we can often make the diagnosis of croupous pneumonia from this alone. It consists of a very tough viscid mucus, which sticks fast to the bottom of the cup even when inverted, and contains an intimate admixture of blood, which gives it a more or less pronounced red or yellow hæmorrhagic color. In individual cases there are numerous gradations. We usually call the pneumonic sputum "rusty," or "brick-red," or of a "prune-juice color," etc. Sometimes it has only a slight reddish or yellowish tint, and sometimes it consists almost entirely of blood. Often the sputum is very frothy. In some cases it assumes a peculiar grass-green ["green-gage"] color, which is due to a change in the blood coloring-matter, or to a mixture with bile pigment in "bilious pneumonia."

The red color of the sputum, as microscopic examination shows, is due to numerous red blood-corpuscles, many of them still well preserved, mixed with it. They are, however, in part dissolved, and hence cause the uniform red color of the sputum. Separate spots containing much blood are often seen in it. Besides the red blood-corpuscles, the microscope shows numerous partly swollen or fatty-degenerated pus-corpuscles. We also see long threads of mucus; sometimes large, round, pigmented cells (alveolar epithelium?); and finally, in rare cases, ciliated epithelium and crystals of hæmatoidin.

Fränkel's diplococci are, as we have said, almost always easily demonstrable in the expectoration, and of course numerous other bacteria besides.

We have still to mention the bronchial casts as important constituents of pneumonic sputum. Since they are usually rolled up together, we may not find them except by spreading out the sputum in water. They consist of the most beautiful casts of the small bronchi, with many dichotomous divisions, and are a product of the croupous inflammation extending into the bronchi. The casts of the smallest bronchi are sometimes found in the form of "spirals," like those in asthmatic bronchitis (see page 184).

The amount of the pneumonic sputum is, as a rule, not very considerable, but it differs a good deal in different cases. The chemical examination of the sputum has so far given no remarkable results. The amount of common salt contained in it is quite considerable.

In many cases the pneumonic expectoration is absent, either because there is no cough or because the exudation is firmly coagulated and is absorbed without ever liquefying. Sometimes it is very tough and slimy, but without any admixture of blood; in other cases the sputum is simply catarrhal, when present at all, and then, of course, it comes not from the parts infiltrated with pneumonia, but from the catarrh of the larger bronchi. In many severe cases the hæmorrhagic expectoration soon assumes more of a purulent character. We often find simple catarrhal sputum, too, besides the characteristic pneumonic sputum.

The pneumonic sputum is sometimes seen in the first or second day of pneumonia, but it may not appear until later. With the beginning of resolution it gradually loses its characteristic appearance. The expectoration then becomes less tenacious and simply muco-purulent, and finally disappears entirely.

Physical Examination.—Inspection shows no especial anomaly in the general contour of the thorax. A marked bulging of the affected side occurs only when there is also abundant effusion into the pleural cavity. The action of the thorax in respiration is very important. With a limited pneumonia we often notice a very marked delay and limitation of motion of the affected side on inspiration. This is due in part to the pain in the side, which comes on with every deep inspiration, and also, in extensive pneumonia, of course, to the physical conditions resulting from the anatomical changes. The unaffected portions of the lung act all the more forcibly.

The acceleration of respiration is very striking, its frequency increasing to thirty or forty, or even more, a minute. We have repeatedly counted sixty respirations in adults, even in cases that finally resulted favorably. The breathing is shallow, but yet, in all severe cases, labored, and often also irregular, as a result of pleuritic pain or cough. We see the inspiratory contraction of the sterno-cleido-mastoids and scaleni in the neck, and often in the face a marked dilatation of the nostrils on inspiration. If there is marked dyspnœa the patient sometimes reclines in bed with the upper half of the body raised. The cheeks and lips are cyanotic. There is often a sharp contrast between the pale portions of the face near the corners of the mouth and the striking feverish and slightly cyanotic flush of the cheeks.

The results of percussion are directly dependent upon the changed physical condition in the lung, due to the anatomical processes. In the beginning of pneumonia, so long as the total amount of air in the lung remains but little altered, the percussion-note is clear, but when the elasticity and tension of the tissue in the diseased portion of the lung diminish, the resonance often becomes quite tympanitic. With increased exudation into the alveoli and smallest bronchi the amount of air in the lung constantly grows less, and therefore the percussion resonance becomes very dull, but it usually retains its tympanitic timbre. Since the pneumonic lung is rarely absolutely deprived of air—for a certain amount is always left in the larger bronchi—the percussion resonance seldom becomes so completely dull or flat, as it does, for example, with a large pleuritic effusion. The sensation of resistance upon the percussion of a pneumonic lung is likewise much less marked than over a pleuritic exudation. A marked sense of resistance implies, therefore, an unusual degree of involvement of the pleura in the inflammation. As soon as the absorption of the exudation begins, the volume of air in the lung increases, and the percussion-note becomes clearer, but remains for some time still markedly tympanitic, until the lung has regained its normal tension and elasticity. We have also to note that the intensity of the dullness in croupous pneumonia is sometimes subject to quite marked variations, since the secretion retained in the bronchi is at one time abundant and at another, after expectoration, scanty.

The extent of the dullness or of the tympanitic resonance is naturally dependent upon the extent of the anatomical process. Small and central infiltrations may entirely escape detection by percussion.

Auscultation is of almost greater importance than percussion in the detection of a beginning or limited pneumonic infiltration. The auscultatory signs depend upon the presence of the pneumonic exudation, and upon the consequent transformation of the lung into a firm tissue devoid of air except in the larger bronchi. In the beginning of the disease we hear over the affected portions coarse or fine râles, and very often, too, the characteristic crepitant râle on inspiration discovered by Laennec. This arises because the walls of the alveoli and smallest bronchi, which are glued together by the viscid exudation, are torn apart at each inspiration. The crepitation, however, is neither pathognomonic of pneumonia, nor heard in every case of pneumonia. With increasing infiltration, bronchial breathing replaces the vesicular. The bronchial breathing in pneumonia is usually very loud, sharp, and close to the ear. When the infiltration is very complete there is often to be heard a pure and loud bronchial respiration, without any adventitious sounds; but of course there are often heard, besides the bronchial breathing, high-pitched, moist râles in greater or less abundance. It is especially true that with the commencement of " resolution "—that is, as soon as the exudation becomes more fluid, abundant moist râles reappear, enough to obscure more or less the bronchial respiration. These râles are, for the most part, rather coarse, moist, and have a musical character. Often we hear at this time the characteristic crepitant râle again (*crepitus redux*). The râles gradually disappear, the respiratory murmur loses its bronchial character, becomes harsh and indefinite, and finally is normally vesicular once more.

We often hear a few rhonchi over the unaffected portions of the lungs. On the diseased side, the respiratory murmur is often more or less diminished because of the diminished respiratory movement. Otherwise respiration in the unaffected portions of the lungs is usually completely normal.

The auscultatory signs just described undergo an important change if the larger bronchi leading to the affected portion of the lung are completely plugged by the secretion, as they are quite liable to be. The respiratory murmur may then almost entirely disappear, and we hear, perhaps, only here and there a few obscure râles. Since such a plugging may be very transitory, we understand why in one day, over the same portion of the lung, we hear first loud bronchial breathing and râles, and then quite obscure and diminished breathing.

Wherever there is bronchial breathing, we hear marked bronchophony. Sometimes one can discover a beginning pneumonic infiltration by bronchophony sooner than by any other physical sign. The vocal fremitus persists or is somewhat increased over a pneumonic lung so long as the large bronchi are open; but when they become plugged, as they are quite liable to be, the vocal fremitus is weakened or wholly abolished; and again, the voice sounds are weakened whenever there is a considerable amount of pleurisy accompanying the pneumonia.

We have yet to add a few remarks about the parts of the lung in which we may expect first to perceive the physical signs of pneumonia, especially the auscultatory signs.

In the first place, we should never neglect to examine carefully the lateral portions of the thorax and the axillary region when we suspect a developing pneumonia. We often find the first râles here in pneumonia of the lower lobes. The first signs of infiltration may be found in the posterior middle portion of the thorax—that is, in the upper part of the lower pulmonary lobes—and thence they extend downward. Pneumonia of the upper lobes begins just as frequently behind in the apices as in front in the infra-clavicular fossæ. Isolated pneumonia of the right middle lobe also occurs, to be made out in front, on the right, between the fourth and sixth ribs. This may remain confined to the middle lobe, or extend to the neighboring lobes.

Few general statements can be made about the nature or the rapidity of the extension of pneumonia, since in these respects the greatest differences are observed. The infiltration may remain confined to a small portion of the lung, or again it may spread over a whole lobe or more in a short time, even in one or two days. We call the pneumonia, whose constant extension by contiguity we can follow from day to day, wandering pneumonia (*pneumonia migrans*), or, from a purely superficial resemblance, which has given rise to many wrong ideas, "erysipelatous pneumonia." In these cases all the signs of resolution are present in the parts first attacked, while the parts affected later are found still at the height of the disease, or in the beginning of infiltration; but we may also find in the autopsies of wandering pneumonia the parts of the lung affected later in a more advanced stage (gray hepatization) than the parts first attacked, which are still in the stage of red hepatization—that is, the inflammatory process in such cases seems to go through a more rapid evolution in the portions of lung later affected. Wandering pneumonia is almost always severe and quite protracted.

Pneumonia in rare cases progresses by leaps. Such cases have been termed erratic pneumonia. In severe cases it is not unusual for both lungs to be affected. We then find the pneumonia either in both lower lobes, or in the lower lobe on one side and the upper lobe on the other side.

2. **Symptoms on the Part of the Pleura.**—As we have already mentioned, every case of pneumonia which reaches to the surface of the lung is associated with a fibrinous pleurisy, and, furthermore, it is not impossible that there should be an infection of the pleura without any direct extension from the neighboring pulmonary tissue. In many instances, the mild pleurisy which attends a pneumonia causes no physical signs. On the other hand, the pain in the side in pneumonia is probably always referable to the involvement of the pleura. In other cases the dry pleurisy attracts attention by the clearly audible friction, which may often be very loud, and is sometimes appreciable to the touch, if the hand is laid upon the side. We rarely hear the pleuritic friction sound in the beginning of pneumonia, but more frequently in the later stages, and perhaps not till many days after the crisis has taken place.

The cases in which pleurisy with effusion complicates pneumonia are more important. This may occur quite early. The abnormality of the clinical course is shown in these cases, as a rule, by the irregular behavior of the fever. There will be no typical crisis, but instead a slow lysis; or after the crisis occurs, the temperature will rise again. In most instances the exudation is serous, but it may be purulent (metapneumonic empyema). Long-continued fever should arouse suspicion of an empyema. In the pus of such cases of empyema the pneumonic diplococcus above mentioned (see page 205) has been repeatedly found. In two fatal cases the author has seen a hæmorrhagic pleurisy with a large amount of coagulated blood in the pleural cavity.

The diagnosis of pleurisy with effusion complicating pneumonia is seldom difficult. The percussion resonance is duller, and the sense of resistance is more marked than in pure pneumonia (*vide supra*). The respiratory murmur and the vocal fremitus are constantly diminished and finally entirely absent. The symptoms of pressure on the neighboring organs and cavities, the heart, the liver, and the semilunar space (see page 287), are especially important because they are most unequivocal. An exploratory puncture with a Pravaz's [hypodermic] syringe, that has been carefully cleansed and disinfected, gives a certain and safe method of recognizing pleurisy in doubtful cases. When there is suspicion that an empyema has begun, an exploratory puncture is imperative.

A moderate degree of pleurisy may somewhat delay the course of the disease, but it has no special significance. Large effusions, however, may decidedly increase the difficulty in respiration and the duration of the illness. Again, the pneumonia may recover, leaving the pleuritic effusion quite undisturbed. In pneumonia of an upper lobe, too, the pleurisy may develop below and lead to an effusion there, while the lower lobe itself remains quite free from pneumonia. Metapneumonic empyema invariably requires surgical intervention, but after operation it almost always pursues a rapid course to recovery.

3. **Circulatory Apparatus. Blood.**—The pulse is accelerated from the beginning of the disease. In cases of moderate severity its frequency reaches 100 or 120; and, in very severe cases, a still higher rate up to 140 or 160 is seen, and is always a dangerous symptom. This high rate of the pulse does not have as bad a significance in children as it does in adults. The consideration of the quality of the pulse is important. Smallness, weakness, and irregularity of the pulse are of bad omen as symptoms of the onset of cardiac weakness. The attacks of collapse, which sometimes come on quite suddenly in severe cases of pneumonia as

in other acute diseases, are especially dangerous. They occasion sudden weakness of the heart with a very small and frequent pulse. The temperature sinks to subnormal, 95° to 93° (35°–34° C.). The peripheral parts, the nose and extremities, become cool, pale, and somewhat cyanotic. The general weakness and prostration become extreme. The collapse may be recovered from, especially with timely assistance, but patients may die in it.

Pericarditis with fibrinous or sero-fibrinous exudation is one of the most serious cardiac complications. This can always be explained by a direct conduction of the inflammatory process from the neighboring pleura, and is therefore somewhat more frequent in left-sided pneumonia than in right. It is a serious matter. Its diagnosis is seldom difficult if we make a careful physical examination of the heart, but with very severe and extensive symptoms in the lungs a complicating pericarditis may be overlooked.

A slight fresh endocarditis is sometimes found at the autopsy, but it has no clinical significance. Diseases of the cardiac muscle, especially fatty and parenchymatous degeneration, may be discovered *post mortem*, but they are by no means frequent. In very weak persons, drunkards, etc., who die of pneumonia, we sometimes, indeed, find the heart remarkably flabby, with the right ventricle dilated, but in many cases of pneumonia we find the muscle of the heart at the autopsy perfectly normal. Here we almost always have to do with conditions of the heart which existed previously to the pneumonia, and merely became prominent during its course. When persons of vigorous and healthy constitution die of pneumonia, as, indeed, seldom occurs, the myocardium is found at the autopsy essentially healthy. It must be emphatically stated that our present knowledge does not enable us to establish before death any positive relation between the histological condition of the cardiac muscle and its functional ability. Frequent experience has made us certain of this fact.

With regard to the blood, there is usually a well-marked leucocytosis in pneumonia. The exact count shows, not infrequently, twenty to twenty-five thousand or more of leucocytes in a cubic millimetre. With the crisis of the fever the number of leucocytes also falls abruptly, while in case of a pseudo-crisis their number remains high. In some instances the leucocytosis is not marked. This is particularly frequent in severe and fatal cases, so that the absence of leucocytosis is regarded with some justice as an unfavorable element in prognosis.

4. **Digestive Apparatus.**—In severe cases of pneumonia the tongue is dry, coated, and quite like the tongue in typhoid. The appetite is also almost wholly lost from the beginning. Vomiting is not infrequent, especially in the beginning of pneumonia, and it also occurs later. It is observed with especial frequency in the pneumonia of children. Severe symptoms on the part of the intestinal canal are rare. As a rule the bowels are constipated, but there are also cases in which diarrhœa is so troublesome that we must believe that the mucous membrane of the intestine is considerably involved in the morbid process (*vide infra*).

The complication of pneumonia with jaundice has a certain significance, but its causes are not always very clear. It is apparently sometimes due to an accompanying catarrh of the duodenum. In other cases the veins of the liver, dilated from stasis, may exert a pressure on the bile-ducts. Slight jaundice has no special significance, and is frequent, even in mild cases; a marked jaundice, however, is seen only in severe cases, especially in drunkard's pneumonia. We call such cases, associated with jaundice, "bilious pneumonia." They have often other severe gastro-intestinal symptoms, such as vomiting, diarrhœa, and meteorism, and severe nervous symptoms, such as stupor and delirium.

The liver may be somewhat enlarged, usually because of passive congestion. The spleen is often moderately swollen, particularly in severe cases, just as in other infectious diseases (acute splenic tumor).

5. **Kidneys and Urine.**—The infectious character of pneumonia is also shown by frequent involvement of the kidneys. Careful examination of the urine almost always shows a trace, or even a considerable amount, of albumen. However, this is very seldom of serious import, and vanishes promptly after the crisis. In many instances the changes in the urine are so considerable as to show an acute nephritis. There is a large amount of albumen, with casts, epithelium, and blood, in the urine. But even these cases of genuine pneumonic nephritis, which usually develop about the third to the sixth day of the illness, seldom prove to be serious, and scarcely ever result in œdema, uræmia, or other complications. Usually they get well rapidly. In one single case the author has observed a transition into chronic nephritis. The way in which pneumonia causes albuminuria and nephritis—between which no sharp dividing line can be drawn—is probably by the production and excretion of toxines.

Great weight was formerly laid upon the diminution of the chlorides in the urine in pneumonia. In fact, the precipitate of chloride of silver, when we put a drop of solution of nitrate of silver into the urine, may be very slight or entirely absent. The chief cause of this diminution of the chlorides is the small amount of nourishment taken by the patient, but we must also bear in mind the large amount of chloride of sodium contained in the pneumonic exudation, and the retention of the chlorides in the body.

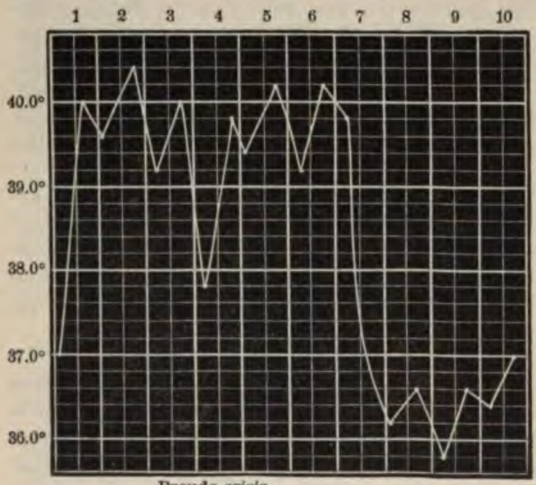
Great significance was also formerly ascribed to the abundant sediment of sodium urate (exceptionally, uric acid) which is often noticed on the day of the crisis. This is known as brick-dust sediment (*sedimentum lateritium*), and is perhaps due in part to a material increase in uric acid (query: through destruction of the large number of leucocytes in the blood?), but certainly in greater part to the fact that the conditions for the deposition of sediment are especially favorable on the day of the crisis. The urine is scanty in amount because perspiration is so excessive, and hence it is concentrated and relatively very acid. It is, therefore, natural for the urates contained in it to be deposited in the form of a sediment.

Pneumonia, in common with most of the other acute febrile diseases, is attended with an increased secretion of urea during the disease. The fact is of theoretic interest that during the resolution of pneumonia the urine often contains a demonstrable quantity of peptone, which is, in all probability, due to the destruction of the cells in the pneumonic exudation and their absorption into the blood.

6. **Nervous System.**—As in every severe febrile disease, nervous symptoms of a mild type are very rarely absent in any case of pneumonia. Among the nervous symptoms are general weakness and dullness, and especially headache, which is often very intense, and is usually increased by coughing. The onset of more severe cerebral symptoms, particularly delirium, is of great importance. Delirium may appear in any case of severe pneumonia, but it is most marked and has peculiar characteristics in alcoholic subjects. This delirium gives the pneumonia of drunkards (*vide infra*) its characteristic stamp.

The usual cerebral symptoms in pneumonia do not correspond with macroscopic changes in the brain, but result from the poisoning of the body with the toxines of the pneumonia diplococcus; and yet there is also a true cerebral disease which has beyond a doubt a special relation to pneumonia, although it is an infrequent complication. We refer to purulent cerebro-spinal meningitis. This complication is particularly apt to appear at times when an epidemic of cerebrospinal meningitis prevails, but it has been repeatedly observed at other times. The diagnosis of pneumonic meningitis may be obscured by the severe constitutional disturbances. Factors of importance are: the stiffness of the back and the neck; the pain in the head and in the nape of the neck; the stupor, changing to deep coma; and, in many cases, optic neuritis, demonstrable by the ophthalmoscope. The termination of a well-marked case of meningitis is probably invariably fatal, but we may have milder meningeal symptoms in pneumonia, such as pain and stiffness in the neck, followed by complete recovery.—With regard to the development of this meningitis, it is probably to be regarded as a true metastatic inflammation, inasmuch as the pneumonia diplococci have been repeatedly found in the pus of the meningitis. As to the path which the pathogenic germs take to reach the meninges, we are not yet certain, but suppose that they travel along the lymph-channels of the intercostal nerves into the meninges surrounding the spinal cord, and thence further to the membranes of the brain.

7. **Skin.**—The frequent appearance of herpes in the course of pneumonia is characteristic, and is of diagnostic importance. It appears from the second to the fourth day of the disease, or sometimes later. Its ordinary seat is on the lips, especially at the corners of the mouth, also on the alæ of the nose, and more rarely on the cheeks or the ear (*herpes labialis, nasalis,* etc.). It has been seen only very rarely on other portions of the body besides the face, for example, on the forearm and the buttock, and in some cases on the cornea and on the mucous membrane of the tongue or gums. The herpes does not always come out all at once, but in fresh crops on successive days. We have several times seen two eruptions of herpes separated by an interval of several days. In repeated instances, under our own observation, herpes labialis, with a fresh rise of temperature, appeared some days after the crisis had taken place. Herpes may be extensive in the mildest cases, while it is particularly apt to be scanty or absent in severe cases. We are, therefore, on the whole, inclined to regard a well-marked eruption of herpes as of favorable prognosis. The true cause of the herpetic eruption is unknown. One might think of referring it to the action of toxines, just as in the herpes of other infectious diseases, such as intermittent and relapsing fevers, and epidemic meningitis. Other affections of the skin are of rare occurrence. We have seen urticaria in some cases. The jaundice occurring in pneumonia has already been described.



Pseudo-crisis.

FIG. 27.—Example of the temperature-curve in croupous pneumonia. (Personal observation.)

8. **Course of the Fever** (see Figs. 27 and 28).—Pneumonia is, almost without exception, accompanied by a more or less high fever with a very typical course. In the beginning of the fever the temperature rises very rapidly to a high point. Even during the initial chill the bodily heat increases from normal to about 104° (40° C.) and over. There are at present no observations to show whether, in the cases of pneumonia that begin gradually, there is also a gradual increase of the fever. During the course of the disease the fever shows on the whole a continuous or remitting character, but there is with this a decided tendency to single deep falls of temperature. Since these at first may easily be taken for the actual occurrence of crises, although later they are proved by the renewed rise in temperature to be a mere

temporary decline in the bodily heat, they are termed pseudo-crises. Pseudo-crises are usually seen in the first days of the disease, but in some cases they appear later and, what is remarkable, they are more apt to appear on those days, such as the fifth or seventh, on which the true crisis is apt to occur. They may be repeated one or more times, giving the fever a decidedly intermitting character. These intermitting pneumonias, so called from the course of the fever, have nothing at all to do with malaria, which fact must be especially noted because of the frequency of erroneous statements.

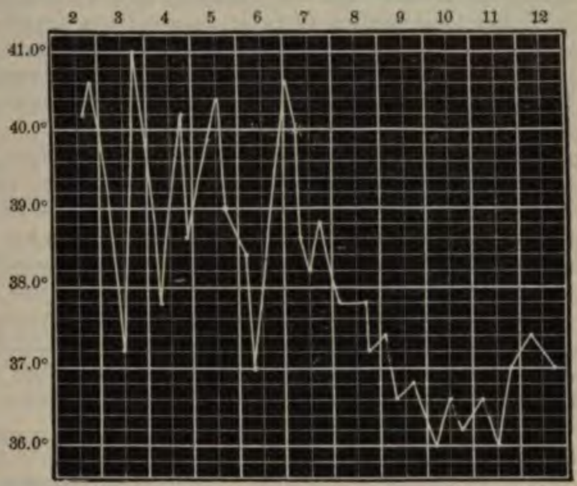


FIG. 28.—Example of the temperature-curve in "intermitting" pneumonia. (Personal observation.)

The fever may be decidedly high in pneumonia, often reaching 104° or 106° (40°–41° C.). The highest temperature observed by us was 107.8° (42.1° C.). This was temporary. There is a certain parallelism between the height of the fever and its severity to this extent, that severe cases are often associated with persistent and especially high fever. But sometimes the most severe and even fatal cases have a comparatively low temperature—between 101° and 103° (38.5–39.5 C.). The highest temperatures are especially common in the first days of the illness, and yet, in spite of very high fever at the onset, the general course of the disease may turn out to be favorable, for the crisis may occur on the second or third day (*vide infra*, rudimentary and abortive pneumonia). In severe cases the progress of the disease often stamps itself plainly on the temperature. The pseudo-crises correspond to temporary improvements, and the fresh exacerbations of temperature to the invasion of a fresh lobe of the lung. We have certainly not seen a special rise immediately before the crisis—the so-called *perturbatio critica*—so often as many statements would lead us to expect. We have seen a gradual decline in temperature quite frequently in the closing days in fatal cases, but the opposite condition also obtains. A marked rise before death is not peculiar to pneumonia, but it does occur when there is a complicating meningitis.

The decline of the fever is the most characteristic portion of the pneumonia curve. The fall in temperature usually comes on in the form of a decided crisis. Generally in the night there is a sinking of the temperature with a more or less abundant perspiration, in which as a rule the temperature may reach a subnormal point—96° to 95° (36°–35° C.). The critical decline is often broken by new and slight elevations of temperature, so that on the morning of the next day there may be a definite increase of fever, the so-called protracted crisis. Only in a comparatively small number of cases does the fever end by lysis, in which the temperature goes down like steps. The duration of lysis is seldom more than three or four days at most. A decline of temperature by lysis is most frequent in severe and protracted cases, in so-called typhoid pneumonia (*vide infra*), and also particularly in *pneumonia migrans*.

After the final crisis has occurred, the active pneumonic process ceases. The day of the crisis is therefore reckoned as the last day of actual illness. The pneumonia makes no advance after that, but resolution and absorption of the exudation and the restoration of the patient's strength still take time. Hippocrates knew when the time of the crisis occurs, and that the odd days, especially the fifth and the seventh, have a special significance in regard to it. In an infectious disease that has a typical course there can be nothing strange in the fact that the cessation of fever, to a certain degree, is associated with a definite period of time; but Hippocrates's rule has frequent exceptions. The crisis sometimes occurs on the ninth, the twelfth, or the thirteenth day, and even later, and, on the other hand, there are quite short pneumonias of but one or two days' duration (*vide infra*).

In the days following the crisis the temperature, which, as we have said, falls to subnormal, regains its normal height. The pulse, which usually sinks to fifty or sixty during the crisis, when it often shows a slight irregularity, reaches its normal frequency again in a few days. We are quite apt to see, in the days immediately following the crisis, a slight temporary increase of temperature again, 100° to 102° at most (38°–39° C.), but this has no special significance.

In cases which run their course regularly, the signs in the lungs upon auscultation and percussion become normal again in about six or eight days after the crisis. Often the time is even shorter than this, or it may be longer. Abnormally delayed resolution will be mentioned below.

Special Peculiarities and Anomalies in the Course of Pneumonia

1. *Pneumonia in Children.*—Besides the common lobular pneumonia there is also a genuine, lobar, croupous pneumonia in children, which is by no means so rare as some authors formerly supposed. An initial chill is seen only in older children; initial vomiting, however, is very common in children. In many cases severe cerebral symptoms, like convulsions, drowsiness, or delirium, obscure the pulmonary symptoms at first. The further course, the development of physical signs, the fever, and the complications, are quite analogous to the appearances in adults. The pneumonic sputum is only exceptionally obtained for observation in children under eight years of age. In previously healthy children the prognosis of croupous pneumonia is almost invariably favorable.

2. *Pneumonia in old people* is, on the other hand, always dangerous. It may begin suddenly, as in people of middle age, but often it begins more slowly and insidiously. Its course is marked by the speedy onset of great weakness and debility. Nervous symptoms, like delirium, are not infrequent. Often there is fatal weakness of the heart.

3. *Drunkard's Pneumonia.*—We see croupous pneumonia in drunkards with remarkable frequency. The usually severe and dangerous course of the disease is due to the feeble resisting powers of their impaired organs. It is characterized by delirium tremens, which usually develops in the first days of the disease. The patient's mind is disturbed, he is very restless, constantly tries to get out of bed, and he fumbles night and day with his bed-clothes or night-gown. The alcoholic character of the delirium is shown by the patient's whole manner, the tremor of the hands and tongue, and the cast of his thoughts, which are usually happy but exceptionally anxious and terrified. His mind wanders to his former occupation or his usual boon companions, and the like. He becomes tearful or raving only when forcibly restrained. He may think himself involved in the tavern brawls. The alcoholic delirium is almost always associated with hallucinations. The hallucinations of little moving black figures are especially characteristic. They are either animals, rats or beetles, or little black men and similar weird shapes, and they give him much trouble. The subjective symptoms of pneumonia are wholly in the background. No delirious patient with pneu-

monia complains of cough, pain in the chest, or dyspnœa. Careful objective examination is the only thing that confirms the diagnosis. Very often patients with a happy delirium serve to entertain those about them, until suddenly very severe symptoms arise, and they become somnolent and succumb, with the symptoms of pulmonary œdema. The prognosis of every case of drunkard's pneumonia, therefore, is to be regarded as very unfavorable.

4. *Pneumonia in Pre-existing Chronic Diseases.*—Croupous pneumonia is occasionally seen in all forms of chronic disease. It is especially dangerous in persons who are already enfeebled, or afflicted with chronic cardiac or pulmonary disease, such as phthisis or emphysema. The pneumonia which often attacks patients with emphysema is clinically important, since emphysema may render the objective evidence of pneumonia very obscure. The croupous exudation does not completely fill the dilated alveoli; hence decided dullness and bronchial breathing are absent.

5. *Pneumonia with Late Localization—Central Pneumonia.*—Cases are quite often seen whose beginning, course, and subjective symptoms correspond throughout to a croupous pneumonia, but in which the objective evidence of pneumonic infiltration evades the most careful examination. The disease begins with a chill, the fever is high, the patient complains of pain in the chest, which is usually slight, there is perhaps herpes, but not till the fourth, fifth, or sixth day can we make out anywhere any bronchial breathing or crepitant râles. In other cases even the crisis may set in before we are able to localize the pneumonia with certainty. In most of these cases we probably have to do less with an actual late localization than with a central infiltration which nowhere approaches the periphery, and hence is made out objectively only late or not at all. A careful examination of the sputum is of the greatest diagnostic importance, since it sometimes has a perfectly characteristic appearance in spite of the absence or the indefinite character of the physical signs. If there is no sputum, the diagnosis may of course remain very uncertain. The appearance of herpes and a critical fall in the temperature make the diagnosis of a pneumonic infection probable even in these cases. In one such case the author observed the development of a pleuritic friction rub upon the first day after the crisis, confirming the diagnosis of pneumonia after the disease had run its course.

6. *Rudimentary and Abortive Forms of Pneumonia—Unusual Localizations of the Pneumonic Infection.*—Particularly at times of a pneumonia epidemic, but also at other times, the author has observed illnesses of short duration but often with high temperature which did not seem like clear cases of pneumonia, although they still were, in all probability, to be regarded as pneumonic—i. e., due to infection with the specific diplococci. Cases of this sort usually begin suddenly with a chill, headache, and high temperature. Generally there is a cough and pain in the chest. Sometimes, however, there are no thoracic symptoms at all. The physician expects pneumonia to develop, but instead, on the first, second, or third day, the fever ceases abruptly, no changes in the lung having been discovered. Very often there will be a *herpes facialis* in such cases, and we doubt not that many instances of so-called *herpes febrilis*, or *febris herpetica*, are really cases of pneumonic infection, without any other localization than the herpes. In other instances, upon careful examination there will be found, at some place in the lungs, a slight crepitation, or limited bronchial breathing; but the process does not extend; and in a very brief time, after a day or two, the fever ceases (rudimentary pneumonia; abortive pneumonia).

In this connection we may remark that the diplococcus infection may be localized in still other places. Thus, for example, we regard it as very probable that many cases of sore throat or acute enteritis, associated with the sudden onset of high fever and with herpes, and especially when seen at the time of an epidemic of pneumonia, are due to diplococcus infection. Associated with these attacks there may also be a late development of pulmonary symptoms.

7. *Typhoid Pneumonia—Asthenic Pneumonia.*—By typhoid pneumonia we mean those cases in which, beside the local pulmonary symptoms, which may be either slight or well marked, there are remarkably severe general symptoms. The cases do not often begin as suddenly as ordinary pneumonia, but more gradually, like typhoid. Even at first the general symptoms, such as great dullness, loss of appetite, or headache, predominate over the thoracic symptoms. At the height of the disease there is a decided typhoidal state, stupor, delirium, a very dry tongue, great general weakness, and also enlargement of the spleen, and frequently mild jaundice, albuminuria, etc. Such cases are to be regarded as pneumonia with an unusually severe general infection (or intoxication). They sometimes occur in epidemics. It is said that pneumonia of the upper lobes shows a somewhat more frequent tendency to severe nervous symptoms than pneumonia of the lower lobes. Recovery from this typhoid or asthenic pneumonia, which may last two weeks or more, often follows by lysis. Typhoid pneumonia is by no means a sharply defined disease. The term serves merely as a short name for the grave constitutional disturbance. Clinically it is impossible to distinguish it sharply from *pneumonia migrans*, bilious pneumonia, and other forms. We must await further bacteriological investigations to determine whether there may not be some other special pathogenic organism in many cases of severe character. For instance, Finkler is inclined to regard certain cases of especially severe pneumonia that seem to be endemic or contagious, as referable to streptococcus infection. In this connection may be briefly mentioned, also, the peculiar and severe pneumonia which is contracted from diseased parrots. On the other hand, it is not improbable that another important factor may be a variation in the virulence of the ordinary pneumococcus.

Pneumo-typhoid is to be carefully distinguished from typhoid pneumonia, although from a clinical point of view the diagnosis between them is often no easy matter. By pneumo-typhoid is meant typhoid fever with a localization of the typhoid bacilli in the lungs (see page 14), but it is also true that ordinary croupous pneumonia may occasionally appear as a complication of typhoid fever.

8. *Pneumonia with Delayed Resolution.*—While the resolution of pneumonia is complete, as a rule, in three days to a week after the occurrence of the crisis, there are cases in which this process demands a much longer time. Not infrequently, and particularly in severe cases of pneumonia, one sees after the crisis a surprisingly rapid disappearance of all the physical signs, while, on the other hand, recovery is sometimes remarkably slow in apparently mild cases; but this rule is of course not without exceptions. The course of the disease is often enough precisely the opposite. Just what are the conditions upon which the rapidity or the slowness of resolution depends we do not know. Sometimes unfavorable constitutional conditions, such as anæmia, debility, phthisical tendencies, and kyphoscoliosis, appear to delay resolution. Sometimes, on the other hand, no such explanation can be found. It seems to us that at certain times all the cases of pneumonia exhibit more of a tendency to delayed resolution than at others, so that it is not impossible that there are variations in the pathological process itself. In many cases of delayed resolution it is our opinion that we have to do with genuine secondary diseases, or a mixed infection in the lungs for the development of which the precedent croupous pneumonia had prepared a favorable soil.

With regard to the symptoms of delayed resolution, there are various forms. In the first place, we see cases where the crisis takes place in the usual way, and the temperature thereafter remains permanently normal. The patients perhaps feel quite well, and are troubled little by thoracic symptoms; nevertheless, the dullness upon percussion remains unchanged, or diminishes at best very gradually, and the bronchial breathing and moist râles can still be heard. All the signs diminish very slowly, sometimes occupying several weeks in their disappearance, and then complete recovery ensues. In a few cases it is striking that, after the crisis, bronchial respiration and dullness persist, while there are scarcely any râles and no expectoration. In these cases it seems as if the pneumonic exudation does not really liquefy at all, and is very slowly absorbed. In other cases there is no distinct crisis, but the fever continues, although lower than at first, and the physical signs remain to a greater or less extent. At the end of two or three weeks, or even still later, the fever slowly ceases, and thereupon normal resonance and vesicular breathing gradually return.

In still other cases the patients remain free from fever for the first few days after the crisis has taken place, although the pneumonia is not completely resolved. Then there appears again a rise of temperature which is mostly moderate, 100.5°–103.5° (38°–39.5° C.), while the dullness continues and there are an abundance of moist râles and catarrhal expectoration. After two or three weeks the fever gradually ceases and the abnormal pulmonary signs also slowly disappear. In such cases we may indeed suppose that some secondary infection, a sort of secondary catarrhal pneumonia, has developed upon the seat of the croupous pneumonia. The disease may take still another course somewhat different from those thus far described, and of this we have seen repeated instances much resembling one another. After the occurrence of the crisis the patient remains for about a week without fever. During this time the dullness and the not very loud bronchial breathing remain unchanged. Then appears a moderate intermitting fever, with elevations to about 102° or 103° (39.5° C.). This fever may last two to four weeks, or even longer. Over the affected portion of the lungs are heard either no moist râles or only a very few. Gradually there appears a moderate but distinct contraction of the side involved, then the resonance gradually grows clear, the respiratory sounds louder and vesicular, the fever ceases, and finally health is completely restored. In many other cases of delayed resolution, as we have already said, we see this same striking absence of moist râles and the development of a moderate degree of contraction. Under such circumstances it is often very difficult to exclude a secondary pleurisy, and we can not make sure except by repeated exploratory puncture. Moreover, it is not exceptional to find delayed resolution and secondary pleurisy both present in the same patient.

9. *Termination of Pneumonia in Phthisis, Contraction of the Lungs, Pulmonary Gangrene, or Pulmonary Abscess.*—Four terminations of pneumonia are ordinarily mentioned as unusual and anomalous—the termination in "chronic pneumonia," in tuberculosis, in gangrene, and in abscess.

Concerning the termination in chronic pneumonia, we have already mentioned a process belonging here, the termination in contraction with ultimate recovery. In rare cases the contraction is permanent. The anatomical process consists in the development of pulmonary cirrhosis, with the formation of a large amount of connective tissue. This proliferation, it should be said, takes place not only in the interstitial tissue, but also in the interior of the alveoli by extension from the alveolar walls. Few clinical observations of the further course of these cases, provided death does not shortly ensue, have as yet been published.

When croupous pneumonia is said to terminate in pulmonary tuberculosis, of course the statement can be understood to mean only that the symptoms of tuberculosis follow immediately upon an attack of pneumonia. When this is the case—it does not occur often—it is probable that the pneumonia attacked a person already suffering from tuberculosis, in whom, however, the symptoms did not become evident until after the pneumonia had run its course. It may also be that exceptionally the pneumonia furnishes a soil for a secondary infection with tubercle bacilli.

Pneumonia results in pulmonary gangrene in rare instances, when the patient is elderly, delicate, or diabetic. Here, too, in our opinion, a new infection, with a foul and putrid substance, must always take place, and this excites the gangrene. The previous pneumonia furnishes only the occasion for the development of gangrene, and perhaps prepares the soil for the agents of decomposition. The development of gangrene is appreciated clinically (see the appropriate chapter) by the change in the sputum and the persistent fever.

The transition from pneumonia to pulmonary abscess is very rare. We can not decide whether a further specific cause is also needed for this, or whether the pneumonic process may exceptionally go on into the formation of abscess. We know that the pneumococci sometimes excite purulent inflammation in the pleura and in the meninges, and so it would not seem impossible that, under peculiar circumstances, they should also cause the development of an abscess in the lungs. The transition to an abscess may be recognized by the character of the sputum, which contains fragments of pulmonary tissue, such as elastic fibers, besides abundant pus. Moreover, we sometimes find, on microscopic examination of the sputum in abscess, scales of cholesterine (Fig. 29) and hæmatoidine crystals; the latter may be so abundant as to give the expectoration a brownish color. Sometimes we have seen a peculiar greenish color of the sputum. The signs of a pulmonary cavity are found if the abscess bursts.

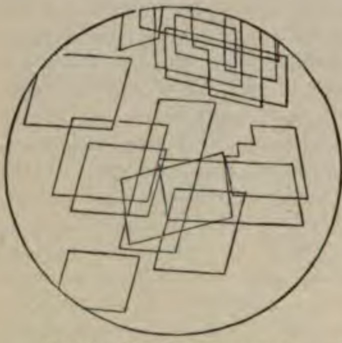


FIG. 29.—Cholesterine crystals.

Diagnosis.—No special remarks on diagnosis need to be added to the description we have given of all the important symptoms which may occur in croupous pneumonia. In the first place, we must consider the sudden onset with a chill and high fever, shortly followed by subjective thoracic symptoms, such as cough and pain in the side; also the characteristic sputum and the objective physical signs, the appearance in many cases of herpes on the face, and finally the general course of the disease, particularly the temperature-curve with its final sudden drop. We will discuss the differential diagnosis between pneumonia and pleurisy with effusion more fully in the description of the latter affection.

Prognosis.—Croupous pneumonia belongs in general to the benignant infectious diseases. The great majority of cases, in previously strong and healthy individuals, run a favorable course, and end in complete recovery. On the other hand, pneumonia brings a number of perils with it, the knowledge of which should always make us cautious in giving a prognosis.

One grave danger lies in the extension of the process. If the advance of the pneumonia can not be stopped, if the whole of one lung is involved, and, besides that, a great portion of the other lung, the diminution of the respiratory surfaces may of itself occasion a fatal termination.

A further danger lies in the onset of certain complications. An intense pleurisy, with effusion, especially if purulent, causes greater difficulty in respiration, and thus increases the danger. Still worse is a sero-fibrinous or purulent pericarditis, which, in not very rare cases, is revealed at the autopsy as the special cause of death. We must note, however, that recovery sometimes finally takes place in spite of an empyema or of a purulent pericarditis. The complication with a purulent meningitis, which is fortunately very rare, is probably invariably fatal.

The dangers of constitutional infection and constitutional intoxication are, on the whole, much smaller in pneumonia than in other infectious diseases; e. g., typhoid fever; but yet, this possibility deserves some consideration, particularly in certain forms of pneumonia already referred to and termed "typhoid" or "asthenic." Such particularly severe and malignant forms of pneumonia, with a high rate of mortality, sometimes appear as endemics and epidemics; but it should be added that these cases are also often marked by the extent of the local process and the development of the dangerous complications above enumerated.

The individuality of the patient plays the most important part in the prognosis of pneumonia. While a constitution that was previously healthy and uninjured usually survives the disease, one that was previously weak and diseased readily succumbs. In this fact lies the danger of pneumonia in old, weak, badly nourished persons, and in persons with a pre-existing emphysema, kyphoscoliosis, heart disease, etc. In this, too, lies the great danger of every pneumonia in drunkards. Since the nervous system is much affected by chronic alcoholism, we very often see outbreaks of delirium tremens in pneumonia. In like manner the other nerve-centers are weakened and incapable of resistance, especially the regulatory centers for the heart and respiration. Hence we can understand why even moderate drinkers, though previously strong and well to all appearances, succumb to pneumonia from failure of the heart and impairment of respiration.

If we ask upon what symptoms our prognosis in any given case should depend, we must reply that no single factor can be given especial prominence. Chief stress must always be laid upon the state of the lungs and the respiration, but attention must also be given to the general condition, the heart's action, the height of the fever, etc. The worst dangers of pneumonia have just been mentioned.

Of the abnormal terminations of pneumonia, contraction gives the best prognosis; but recovery, or at least a marked subsidence of all the symptoms, may sometimes take place after gangrene and abscess.

Treatment.—Many of the milder cases of typical pneumonia need no special active treatment when the disease takes a favorable course. Most cases get well under, or, we can almost say, in spite of any treatment. From the now obsolete method of treatment by large bleedings, and from the use of certain drugs (veratrine, tartar emetic), which are even now sometimes employed, we should expect harm rather than any benefit, yet under such treatment many patients have recovered.

We do not know of any certain means favorably to influence the pneumonic process. Whether we are destined to learn of some specific mode of treatment, perhaps by means of some serum, after the analogy of other infectious diseases, as diphtheria, we can not say. A few beginnings have already been made in this direction, but without any practical results as yet. We are at present obliged, therefore, to fall back upon a purely symptomatic and constitutional treatment of pneumonia.

The symptoms which are usually prominent in pneumonia, even in the milder cases, and of which the patient is especially desirous to be relieved, are the pain in the side, the troublesome cough, and the difficulty and distress in breathing. Since the respiratory symptoms, as we have seen, are partly due to the pain, as this improves the patient's breathing often undergoes a decided improvement. For the pain, we may first mention a number of external applications to the skin on the affected side. An ice-bag sometimes gives marked relief. Many patients can not bear this, but prefer warm poultices or cold wet compresses. The application of mustard plasters or dry cups to the skin may be of advantage. Subcutaneous injections of morphine, however, are the most effective remedy, and are often indispensable. There is no reason why we should not use this remedy, with care and in moderate doses, for the relief of pain; and, as the disease is of short duration, we need not particularly fear the morphine habit. Small doses of morphine, subcutaneously or by the mouth, may also be required to alleviate the cough.

Local blood-letting is a remedy the action of which can not be explained physiologically, and yet experience has shown that it is of undoubted advantage. The relief which many patients feel after the application of eight or twelve leeches to the affected side is very striking; but we should prescribe them only when there are severe symptoms at the beginning of the disease, and in persons who were strong and healthy before the attack. Wet cups accomplish the same thing, but their effect is somewhat more powerful, and hence they should be reserved for strong and robust persons, such as laborers.

The tepid or cold bath serves as the most effective means of improving the respiration, of aiding expectoration, and of stimulating and refreshing the whole system. We hold it useless, if not injurious, to give a patient baths if the disease is progressing favorably, for almost every bath has some disagreeable feature. These disadvantages, however, are always less, in severe cases, than the benefit and improvement which baths give the patient, and which most patients recognize with gratitude. The main point is that the patient should make no physical exertion while in the bath, that he should be lifted into it, held and supported while in it, and lifted into bed again after it. Since the baths are given primarily not on account of the fever, but to improve the respiration, and because of their favorable influence on the nervous system, their temperature need not be especially low. We give them from 82° to 86° (22°–24° R.); somewhat warmer with sensitive and weak people, and cooler, down to 77° or even 72.5° (20°–18° R.), with strong persons, or with very high fever or severe nervous symptoms. We need not employ more than two or three baths a day, and at night we employ them only when there are threatening symptoms. The favorable action of the baths is seen especially in the great relief and refreshment that the patient feels. The respiration is quieter and slower, but deeper. The patient often falls into a quiet sleep after the bath. Of late years we have often replaced the baths by a wet pack, even in adults. This has been almost always very well received, and indeed seemed to make the patients quieter, with easier respiration and less discomfort. We would particularly and strongly recommend the pack for private patients, in whose case the employment of baths is associated with many difficulties.

Among internal remedies for pneumonia, antipyretics are frequently employed. We do not believe they are capable of exerting any decided influence upon the general course of the disease, although we admit that antipyrine and, under some circumstances, also phenacetine and antifebrine have a good effect, since they not only modify the fever, but improve the nervous symptoms and the general condition. Antipyrine is prescribed in doses of fifteen to thirty grains (grammes 1–2), particularly toward night.

To make the cough somewhat more effective, expectorants are prescribed. We ourselves most frequently employ infusion of ipecac, apomorphine, infusion of senega, liquor ammonii anisatus, and benzoin. The last two remedies are especially favorable if the heart is feeble. It also seems to us of some importance, with regard to expectoration, to prescribe an abundance of liquid in the form of water, tea, lemonade, and similar drinks.

The behavior of the heart should always be watched with vigilance, especially in elderly and delicate persons, and in the obese and alcoholic. If the pulse becomes very frequent, an ice-bag is placed upon the heart. If the pulse-rate is remarkably rapid from the start, we may order digitalis at once, either in infusion, or, still better, in powders containing 1½ to 3 grains (gramme 0.1–0.2), repeated several times a day; and as digitalis acts slowly, requiring twelve to twenty-four hours to make its influence felt, if the weakness of the heart is dangerous, stimulants which act more promptly must be employed. As such, tincture of strophanthus, repeated several times a day, deserves to be recommended, and, above all, subcutaneous injections of oleum camphoratum (1 to 4), of which three or four syringefuls (♏ xv), and more, may be employed (ʒss. to ʒj). Again, ether injected subcutaneously is a powerful cardiac stimulant, but it should be employed cautiously because of its marked local effect (paralysis of the nerves), and the place of injection should be the skin of the abdomen or the thigh.

We have yet to make some remarks upon the very extensive use of large amounts of alcohol in pneumonia. Without doubt a free use of alcohol is necessary in drunkards, especially when delirium tremens is beginning or is already pronounced. Since the withdrawal of any poison that is taken habitually, like nicotine or morphine, may excite the severest symptoms, the sudden withdrawal of alcohol from drunkards may have the worst results, while, if we give an abundant supply of the stimulant to which the nervous system is accustomed, we sometimes succeed in avoiding the onset of severe nervous symptoms, such as collapse and failure of the heart and respiration. Likewise wine should certainly be given to persons who are accustomed to it and themselves desire it. It is quite a different matter with patients who before their illness have not been accustomed to take alcohol at all, or who took it only in small amounts. It may be true that in these cases moderate amounts of wine may have a stimulating and exciting action, although we never could satisfy ourselves of the often praised influence of alcohol upon the action of the heart. We hold it, however, unjustifiable to force large amounts of alcohol indiscriminately upon every patient with pneumonia, perhaps in spite of great resistance on his part. Why should we expect sick persons to bear doses of alcohol which have only bad results on healthy men unaccustomed to them? The assertion that fever patients "bear" alcohol better than healthy persons lacks proof. It should be admitted that perhaps alcohol is more rapidly consumed in fever than in health, but it should also be considered that the toxic effects of alcohol are less easily noticeable in comatose patients than in persons possessing normal consciousness.

[Few American physicians of any experience will accept the reasoning of the author on the employment of alcoholic stimulants in those not accustomed to their use.

The toxic effects of alcohol are as undesirable in pneumonia as in any other disease, but there are few affections in which so great tolerance is shown for this agent. The chief indications for its exhibition are derived from the pulse and the first cardiac sound at the apex. A flagging heart calls for alcohol, the effect of which on the symptoms and on the circulation is to be carefully watched; the quantity is to be diminished, increased, maintained, or the agent is to be omitted entirely, according to the conditions present in the individual case. I am no advocate of indiscriminate alcoholic stimulation; but I believe that lives have been frequently saved in the past, and will be saved in the future, by the judicious and sometimes extremely free use of this class of remedies in acute pneumonia. Inhalation of oxygen has won a prominent place in the treatment of severe pneumonia of late years, and cases are reported in which life seems unquestionably to have been saved by it. In a considerable number of cases the editor has seen only one which could come under this class. He has seen, however, marked temporary improvement in pulse and respiration, with diminution of cyanosis and induction of relatively quiet sleep. But the oxygen must be given in large quantities, sometimes continuously. If its inhalation fatigues the patient, the gas may be allowed to escape before the mouth or under the nose of the patient. The objection to the efficient use of oxygen in some cases is the expense of perhaps some hundreds of gallons a day at five cents a gallon.]

We scarcely need to lay special emphasis on the fact that the physican should endeavor, so far as possible, to maintain the bodily strength by means of appropriate and sufficient nourishment. Soups, bouillon with toast or rusk, milk, and eggs are the most suitable articles of diet, and it is sometimes proper to give small amounts of finely minced meat. Care should be taken that the patient has an abundance of refreshing beverages. Wine and also moderate amounts of good beer may be allowed without hesitation.

The treatment of complications follows the ordinary rules which have been given under the individual affections. We must also mention that, in delirium tremens, tepid baths with cold douches sometimes have a very good effect. Besides this, we may try subcutaneous injections of strychnine, seven to fifteen minims of a one-per-cent. solution, once or twice a day. We can not wholly dispense with narcotics, such as morphine and chloral, but we must warn against the imprudent use of large doses of chloral, above thirty-five grains (grammes 2.5).

CHAPTER VI

TUBERCULOSIS OF THE LUNGS

(*Pulmonary Phthisis. Pulmonary Consumption*)

General Pathology and Ætiology of Tuberculosis

Ever since Bayle, in 1810, demonstrated the extensive distribution of peculiar nodules in the various organs, and their relation to pulmonary consumption, few questions have so taxed clinical observers and pathologists as those relating to the cause and nature of tuberculosis. Harmony could not be reached, however, so long as the criterion for the decision of the questions was sought in the presence of definite anatomical changes, which were regarded as specific of tuberculosis. Laennec considered the peculiar change in the tubercular products, which later was named caseation by Virchow, to be characteristic, and called everything tubercular in which it was found. He distinguished the isolated tubercle from diffuse, tubercular, cheesy infiltration. Thus Laennec recognized that many processes were allied whose affinity was often disputed afterward, and which has only recently been established, such as the affinity between "scrofulous" enlargement of the glands and tuberculosis. Another opinion became quite prevalent, after Virchow discovered that precisely the same anatomical process as tubercular caseation was also found in inflammatory products, which were certainly not tubercular, and in cancerous ulcerations. Hence Virchow made a sharp distinction between tubercle and those new growths and inflammatory processes which had become cheesy. The anatomical criterion of tuberculosis was, in his view, the presence of the miliary tubercle, a gray nodule, the size of a millet-seed at the largest, made up of cells like lymph-corpuscles. The study of the finer structure of the miliary tubercle was now pushed most eagerly by Wagner, Schüppel, Langhans, and others, but they were unable to reach perfect harmony regarding its origin and significance.

As long ago as 1865, however, a discovery was made which pointed unequivocally to the only way which could lead to a correct knowledge of tuberculosis. It was the fact, discovered by Villemin, that tuberculosis can be produced artificially by inoculating healthy animals with small amounts of tubercular and cheesy substances. Although doubted and misinterpreted at first in various quarters, the fact that tuberculosis can be transmitted, and consequently the fact of its infectious character, must now be regarded as proved beyond a doubt. In the general change which our opinions upon the nature of infectious diseases have undergone, especially in the last few years, the existence of a specific, organized cause of tuberculosis, too, had to be assumed. Klebs, and later Cohnheim, had already without reserve defined tuberculosis as a specific, infectious disease and, sooner than we dared to hope, R. Koch discovered the special carriers of the disease in the shape of the tubercle bacilli, in the year 1881. The definition of tuberculosis no longer rests upon any external anatomical character. Every disease is tubercular which is excited by the pathogenic action of a specific kind of bacteria, the tubercle bacilli discovered by Koch.

The pathogenic bacteria of tuberculosis belong to the group of bacilli. The tubercle bacilli are rod-like, of small diameter, slightly rounded at their extremities, and either straight or somewhat bent. Their length is perhaps a fourth or a half that of the diameter of a red blood-corpuscle. In the interior of these rods it is not infrequently possible to distinguish very minute colorless spots which are probably to be regarded as endogenous spores. The tubercle bacilli have no independent motion whatever. Their reaction to certain coloring matters is very characteristic, and of the highest importance with regard to their recognition (*vide infra*). We know with absolute certainty that the tubercle bacilli are always present in all the different forms of pulmonary tuberculosis, both in the lung itself and in the expectoration (*vide infra*), and also in tubercular diseases of other organs, the brain, the intestines, the spleen, the liver, and the kidneys, and also in "scrofulous lymph-glands," in "fungous" diseases of the bones and joints, and in the so-called lupus, which is nothing but a local tuberculosis of the skin. Precisely the same bacilli are also found in the "spontaneous" tuberculosis of animals, such as monkeys, puppies, and guinea-pigs, and in every tuberculosis that is artificially produced in animals by inoculation. Finally, by the discovery of tubercle bacilli in the "pearly distemper" of cattle, the identity of this disease with tuberculosis—an identity which had already been established by experiments in inoculation—was confirmed anew.

Koch, by his successful "pure cultures" and inoculations with the cultivated bacilli, has established the fact that these bodies, known as tubercle bacilli, are to be regarded as organized, and as the special cause of tuberculosis. Bacilli coming from any fresh product of tubercular disease may be cultivated at a constant temperature of 98° to 100° (37°–38° C.) upon blood-serum which has been stiffened by heating, and upon several other artificially prepared soils. In this cultivation they show certain characteristic properties in their growth, which can not be fully described here, and they multiply to an unlimited extent. In this way we can keep up perfectly pure cultures of tubercle bacilli. Inoculation experiments tried with them on all sorts of creatures always give a positive result. The animals fall ill, lose flesh, and finally die, and at the autopsy we find undoubted tubercular disease of the internal organs, which may be more or less extensive. The most instructive inoculations are those into the anterior chamber of the eye in puppies or guinea-pigs, which were first tried by Cohnheim and Salomonsen. After an incubation of two or three weeks we see here very plainly an eruption of the first nodules of tubercle in the iris, and the tuberculosis spreads to the other organs of the body later.

By these experiments it was first demonstrated that tuberculosis is always, at the start, a purely local disease, and that its further extension depends upon the spread of its germs.

Ætiology of Tuberculosis in Man

The distribution of tubercle bacilli must be remarkably extensive, for tubercular diseases occur in almost every country on earth. The predisposition of mankind to the disease is also very great, and thus we understand the frightful fact, which statistics show, that nearly one seventh of all deaths are from tuberculosis! It has neither been proved, nor is it probable, that tubercle bacilli multiply outside of the human body, like the bacilli of splenic fever, since they can develop only in a constant and uniformly warm temperature between 85° and 104° (30°–40° C.). We must therefore regard them as true parasites, which can live—that is, which can propagate and multiply—only in the bodies of animals, but they (or their spores) seem to preserve their virulence and their ability to multiply outside of the body for a long time. Phthisical sputum may be used for inoculation with success, even if it has been dried for several weeks. The tubercle bacilli also resist most chemical reagents, such as nitric acid, very decidedly.

If the body becomes infected, then, with tubercle bacilli, it is always probable that they have come from some other individual—man or beast—with tubercular disease. We need not mention how numerous the opportunities for infection may be, considering the present general distribution of tuberculosis. The chief stress in this respect is to be laid upon the expectoration of phthisical patients, which contains the bacilli. This dries on the floor, on the linen, and on other objects, and then the small particles which contain the germs of infection are carried off by the air. The extensive investigations of Cornet have well shown how frequently it is possible to obtain tuberculous material, which is capable of causing infection, from the dust in the neighborhood of phthisical patients who carelessly deposit their expectoration upon the floor and in handkerchiefs and elsewhere. On the other hand, Flügge has lately emphasized the fact that, as shown by experiments, the inhalation of dry sputum in the form of dust is far less dangerous than the breathing-in of fine particles of liquid expectoration. He points out that the consumptive with a cough can be shown to defile the surrounding air with numerous minute drops of sputum containing tubercle bacilli. These hover for a time in the air, and may be inhaled by other persons. This mode of infection may actually be important as causing the direct transmission of tuberculosis, and that such direct transmission is possible is shown by the cases of tuberculosis in married couples, in nurses, and in patients who for a long time occupy a ward in which are many consumptives. Still, the experience of physicians proves that such direct infection with tuberculosis is of very infrequent occurrence (*vide infra*). There are very many who fall sick with tuberculosis who have never been in close contact with other consumptives. In such cases it must be that the tubercle bacilli enter the body indirectly.

The material which contains the bacilli or spores is taken into the body, in the majority of cases, along with the inspired air. This is probable, because, in most cases, tuberculosis has its starting-point in the air-passages, the lungs, or the larynx. Inoculation experiments show that the first extension of the process depends upon the point of inoculation. If it be in the anterior chamber of the eye, the first nodules appear in the iris, as we have said; if it be in the abdominal cavity, we have first a tuberculosis of the peritoneum; if the infectious matter be inhaled, we have first a tuberculosis of the lungs. Hence it seems very probable that, in tuberculosis in man, the infectious matter is taken directly into the air-passages by the breath. In this way it sometimes, though rarely, attacks the upper air-passages, as in primary tuberculosis of the nose, the pharynx, and the larynx, but more frequently it affects the deeper portions of the respiratory apparatus, as in primary tuberculosis of the lungs and bronchi.

We must also consider other modes of infection, first among which is the possibility of infection of the intestinal canal, from swallowing the infectious material. The transmission of tuberculosis from domestic animals to man plays a part in this connection which perhaps is not unimportant. Since the pearly distemper of cattle is identical with tuberculosis in man, the use of the flesh of such animals as food furnishes a possible means of infection. It is a still more important circumstance that, when pearly nodules are present on the udder, the milk of the diseased animal may be polluted by tubercle bacilli, and that the use of such milk as food, when it is uncooked, certainly involves the danger of the transmission of tuberculosis. Primary tuberculosis of the intestines, however, is not very frequent, probably because the tubercle bacilli which have been swallowed are usually destroyed in the stomach. Still, it is to be noticed that primary tuberculosis of the intestine and of the abdominal lymph-glands (*vide infra*) is far more frequent in children than in adults, and that it is children who drink the most milk.

In some cases the tubercular infection may probably arise from little fissures and excoriations of the skin. In this way either we get a local tuberculosis of the skin, such as lupus, or the bacilli are carried by the lymphatics to the neighboring glands of the neck or axilla, establish themselves there, and set up a tubercular disease in them. It should be stated that even when the tubercle bacilli are taken into the body through the lungs or the intestine, the infectious material often passes the primary seat of infection without fastening itself there, and does not take root until it reaches the nearest lymph-glands; or perhaps, often, still more distant regions, such as the kidneys or the bones (?). Thus arises the so-called primary tuberculosis of the bronchial or mesenteric glands, leading sometimes, as we shall see later, to various severe tubercular diseases in other parts, such as the pleura or peritoneum.

It remains to mention the possibility of a primary genito-urinary tuberculosis. Primary infection of the uterus and ovaries might be referable to direct infection from without; but it is questionable whether genito-urinary tuberculosis of the male, affecting the kidneys, testicles, and prostate, arises in this manner. In such cases we may be dealing with infection due to the excretion by the kidneys of tubercle bacilli which have got into the body in some other way.

Considering the wide distribution of the tubercle bacilli, and the many chances for infection, it seems wonderful that in spite of it so many men escape the disease. One factor, which has been already mentioned by Koch, must be borne in mind, however, and that is the extremely slow growth of the tubercle bacilli. This is the reason why the bacilli do not always remain in the body, but in many cases are eliminated again before they have gained a definite foothold.

Individual predisposition, however, is another factor which is probably still more important—a factor which we can not well explain, but which we can not get on without, at the present time, in the pathology of many infectious diseases. In our conception of most of the other infectious diseases, as well as of tuberculosis, we must assume provisionally an unequal predisposition to disease in different individuals. Only a part fall ill of all who are exposed to the action of the poison. These are persons in whom it is particularly easy for the poison to establish and propagate itself.

It is very striking that the existence of the tendency to tuberculosis is often expressed in the general constitutional debility of the individual, and—still more remarkable—in certain peculiarities of his bodily frame, e. g., the shape of his thorax. This phthisical habitus (*vide infra*) is especially common in persons who come from families with a tendency to tuberculosis, and it is a peculiar and quite inexplicable expression of the predisposition to tuberculosis, due to family and hereditary influence (*vide infra*). It should be said that the hereditary tendency to tuberculosis is not invariably associated with any striking and self-evident weakness of the constitution. Persons who spring from tuberculous families may have a vigorous frame and yet fall victims to the disease; and likewise also, when there is no hereditary tendency, the most powerful build does not by any means render one invulnerable to the disease.

We now believe that many evil influences which were once thought to be causes of tuberculosis act only in increasing the predisposition to the disease. Insufficient food, bad air, severe illness, childbirth, want, and care—these alone, of course, can never produce tuberculosis; but we can easily imagine that the body which has become weakened affords less resistance to the injurious influence of the tubercular poison than does the strong and healthy body. Thus it seems to us, from our own observations, to be in the highest degree probable that chronic alcoholism increases the liability of the individual to tuberculosis. It is surprising how often drunkards, possessed of a naturally most vigorous constitution, fall victims to tuberculosis; although in this connection we ought also to consider that drunkards are so often and so long in filthy and ill-ventilated saloons that they are particularly exposed to the danger of infection.

People used often to speak of the transition of other affections of the lungs into pulmonary consumption—that is, into tuberculosis. It was imagined that an old bronchial catarrh, croupous inflammation of the lungs, or the catarrhal pneumonia accompanying measles, or whooping-cough, could readily become "tubercular." Of course the proper interpretation of such a connection is that the precedent disease prepares a favorable soil for infection with the tuberculous virus, and that consequently the tubercle bacilli fasten more readily upon the mucous membrane, which has been previuosly affected, than under normal conditions. Moreover, many of the infections which we formerly supposed to be apt to change into tuberculosis are themselves tuberculous. This is true in most of the so-called scrofulous diseases of the lymph-glands, bones, etc., and also, as we shall see later, in the overwhelming majority of cases of apparently primary pleurisy. No one will now admit the truth of the theory, which Niemeyer once vigorously defended, that a primary pulmonary hæmorrhage could cause the development of pulmonary phthisis. Certainly, in the cases which apparently support such an opinion, the pulmonary hæmorrhage is not the cause, but a symptom, of pulmonary tuberculosis.

No single factor, however, of those which favor the predisposition to tuberculosis, plays so manifest and so visible a part as does the hereditary tendency above mentioned—that is, the inborn predisposition of the individual. The fact of the heredity of phthisis meets us with such uncommon frequency that it must have forced itself upon the notice of the older physicians. In the great majority of all cases of phthisis we can make out, by close questioning, that in the family, either among the older members or among the brothers and sisters, one or more cases of tubercular disease have already occurred. The closer we investigate, and the more we search for some one of the different forms in which tuberculosis can show itself, like pleurisy, or affections of the bones and joints, the more frequently we can make out this hereditary predisposition. Some persons are indeed of the opinion that the hereditary transmission of the disease is often merely apparent, and not real, from the fact that the close relations between the children and their diseased parents, or brothers and sisters, greatly increases the danger of infection of the ordinary kind. Certainly this consideration should not be forgotten with regard to the occasional appearance of tuberculosis in families, but yet it would be impossible to explain in this way alone the extremely frequent development of tuberculosis in special families.

There can be, therefore, scarcely any doubt as to the inheritance of tuberculosis, but the explanation of this fact is far from clear. The hereditary charac-

The dangers of constitutional infection and constitutional intoxication are, on the whole, much smaller in pneumonia than in other infectious diseases; e. g., typhoid fever; but yet, this possibility deserves some consideration, particularly in certain forms of pneumonia already referred to and termed "typhoid" or "asthenic." Such particularly severe and malignant forms of pneumonia, with a high rate of mortality, sometimes appear as endemics and epidemics; but it should be added that these cases are also often marked by the extent of the local process and the development of the dangerous complications above enumerated.

The individuality of the patient plays the most important part in the prognosis of pneumonia. While a constitution that was previously healthy and uninjured usually survives the disease, one that was previously weak and diseased readily succumbs. In this fact lies the danger of pneumonia in old, weak, badly nourished persons, and in persons with a pre-existing emphysema, kyphoscoliosis, heart disease, etc. In this, too, lies the great danger of every pneumonia in drunkards. Since the nervous system is much affected by chronic alcoholism, we very often see outbreaks of delirium tremens in pneumonia. In like manner the other nerve-centers are weakened and incapable of resistance, especially the regulatory centers for the heart and respiration. Hence we can understand why even moderate drinkers, though previously strong and well to all appearances, succumb to pneumonia from failure of the heart and impairment of respiration.

If we ask upon what symptoms our prognosis in any given case should depend, we must reply that no single factor can be given especial prominence. Chief stress must always be laid upon the state of the lungs and the respiration, but attention must also be given to the general condition, the heart's action, the height of the fever, etc. The worst dangers of pneumonia have just been mentioned.

Of the abnormal terminations of pneumonia, contraction gives the best prognosis; but recovery, or at least a marked subsidence of all the symptoms, may sometimes take place after gangrene and abscess.

Treatment.—Many of the milder cases of typical pneumonia need no special active treatment when the disease takes a favorable course. Most cases get well under, or, we can almost say, in spite of any treatment. From the now obsolete method of treatment by large bleedings, and from the use of certain drugs (veratrine, tartar emetic), which are even now sometimes employed, we should expect harm rather than any benefit, yet under such treatment many patients have recovered.

We do not know of any certain means favorably to influence the pneumonic process. Whether we are destined to learn of some specific mode of treatment, perhaps by means of some serum, after the analogy of other infectious diseases, as diphtheria, we can not say. A few beginnings have already been made in this direction, but without any practical results as yet. We are at present obliged, therefore, to fall back upon a purely symptomatic and constitutional treatment of pneumonia.

The symptoms which are usually prominent in pneumonia, even in the milder cases, and of which the patient is especially desirous to be relieved, are the pain in the side, the troublesome cough, and the difficulty and distress in breathing. Since the respiratory symptoms, as we have seen, are partly due to the pain, as this improves the patient's breathing often undergoes a decided improvement. For the pain, we may first mention a number of external applications to the skin on the affected side. An ice-bag sometimes gives marked relief. Many patients can not bear this, but prefer warm poultices or cold wet compresses. The application of mustard plasters or dry cups to the skin may be of advantage. Subcutaneous injections of morphine, however, are the most effective remedy, and tious material extends over the body by ways and means which we shall learn in part later on, and attacks one organ after another, or even many at once. Finally, it is to be said that the peculiar character of the changes caused by tuberculosis explains why these are often the cause of manifold secondary processes, such as toxic effects and, particularly, secondary infections. Thus arise fever, suppuration, secondary inflammation, and other important phenomena, which will be minutely considered later.

The entire local action of the tubercle bacilli—that is, the pathological anatomy of tuberculosis—is almost wholly independent of the organ attacked. Tuberculosis belongs to the group of so-called "infectious tumors"—that is, the local action of the tubercle bacilli consists chiefly in the production of a proliferation and accumulation of cells at their place of settlement, which is termed a tubercular new growth. Without going into histological details we may say, briefly, that the whole process consists in a primary injury of the tissue, due to the invasion of the tubercle bacilli, followed by a hyperplasia of the cells peculiar to the tissue itself, and involving not only the connective-tissue cells but also those of the epithelium. In this way are developed the so-called epithelioid cells and the giant cells. The next step in the process is the migration of numerous leucocytes from the surrounding blood-vessels. The leucocytes, or round cells, collect about the above-mentioned new-formed cells, and may finally completely cover them. A network, or reticulum, is found between the individual new-formed cells and the wandering cells. This probably represents the remains of the original interstitial tissue, crowded apart by the increased number of cells. There is no new formation of vessels. The tubercle contains no blood-vessels. The tubercle bacilli lie especially inside the giant cells, but also in their vicinity.

If these changes have progressed far enough, they become visible to the naked eye as a small, circumscribed grayish point, which is called a miliary tubercle. From these minute nodules the disease itself obtained its name of tuberculosis. By approximation and coalescence of neighboring tubercles—for these keep developing because of the spread of the local infection—the tuberculous formation continually extends itself, and thus are gradually formed the large tubercular nodules, and finally the diffuse tuberculous new growth or the diffuse tuberculous infiltration.

The tuberculous new growth, as such, can scarcely ever be distinguished histologically from other infectious tumors, such as those seen in syphilis and leprosy. Tuberculosis, however, has a characteristic difference in its final stages of cheesy degeneration and eventual disintegration of the new-formed tissue, processes which are apparently connected with the absence of blood-vessels and the consequent deficiency of nutrition of the new formation. Both the tuberculous infiltration and also the portions of the original tissues inclosed by it perish, lose their nuclei, and finally become disintegrated. The manner in which they are destroyed—namely, "fatty degeneration"—belongs in the group of the so-called coagulation necroses. This process is recognizable to the naked eye, because the tuberculous infiltration when it becomes thus degenerated takes on a pronounced yellowish color. Wherever the necrotic portions of tissue are superficially situated they are cast off, giving rise to tuberculous ulcers.

Alongside the tuberculous new formation there are found in the organs affected with tuberculosis various inflammatory processes, either simple or suppurative or hæmorrhagic. We may therefore surmise that the tubercle bacilli (or the chemical toxines formed by them), besides their characteristic effects, act simultaneously in another rôle as excitants of inflammation; but it is very probable, and especially so in pulmonary tuberculosis, that many of the inflammatory changes which arise are not peculiar to the tuberculosis as such, but are to be regarded as secondary processes (*vide infra*), for the development of which the preceding tuberculous new growth, as it disintegrates, furnishes a suitable soil.

As regards the special anatomical processes and appearances in pulmonary tuberculosis, the tubercular change usually begins in the walls of the smallest bronchi, or not rarely in the alveoli themselves. The disease does not begin, however, in many different parts of the lung at once, but probably in one or two circumscribed spots only, and in a great majority of cases in one apex. We do not know why the apices are so often the starting-point of phthisis, but perhaps it is because of their relatively slight expiratory power, which renders them a favorable lodging-place for the tubercle bacilli.

The tuberculous infiltration begins in the walls of the bronchi, and thence gradually extends toward the periphery. The original tuberculous bronchitis becomes a tuberculous peribronchitis. The infectious material extends from its original focus by means of the lymph- and blood-channels out into the surrounding tissue; and also as soon as superficial ulceration takes place, the infectious matter is liable to be carried by the inspired air into other bronchi. Thus the diseased spot, small at first, keeps gradually extending. Tubercular peribronchitis is usually easily recognized with the naked eye. We notice the little lumen of the bronchus in the middle of the "cheesy" nodule, which at first is gray and later yellowish. Many of the adjacent nodules run together in part, and even entirely. The lumen of the bronchus is either wholly plugged by the infiltration, or the destruction of the necrotic cells begins in the midst of the peribronchitis. In the latter case the lumen is enlarged to a little irregular hole—the first beginning of the formation of a cavity.

The alveolar tissue of the lung can not long remain unaffected, with such a disease of the smaller bronchi. Lobular atelectasis, the necessary result of every permanent bronchial obstruction, must arise, but this soon passes (by penetration or aspiration of the germs) into a lobular pneumonia, which from its specific nature later becomes caseous. We can not go into the histological details here. The alveoli are filled with pus-corpuscles and large epithelioid cells, which are considered by many authors to be the offspring of the alveolar epithelium. The alveolar walls are also infiltrated. This finally results in the destruction of the cheesy and necrotic tissue, and consequently in the formation of cavities. At other times the neighboring nodules run together, and the tubercular infiltration thus extends, giving rise to a diffuse caseous pneumonia. These processes may all be readily recognized by the naked eye. The earlier stages of atelectasis and infiltration correspond to the jelly-like, gray coloring seen in the so-called gelatinous infiltration of Laennec, and the transition to caseation is recognized by the eye from the yellowish color.

Although all the processes thus far described are destructive in their nature, changes are also found in the lungs in tuberculosis which seem to have a tendency toward circumscribing the disease and toward healing. Prominent among these are the chronic interstitial processes. We meet with the formation of new connective tissue, partly about the tubercular infiltration, but especially where there is already destruction of tissue, and this leads to contraction and the formation of a firm cicatrix. The encapsulated cheesy masses may then be in part reabsorbed; in part they undergo calcification. The possibility of such a halt in the tuberculous process depends, however, upon certain conditions. The tuberculous new growth and its destruction must not advance too rapidly, and the new-formed tissue must not itself be destroyed before it becomes cicatrized. We see the cicatricial formation, therefore, more especially in chronic cases; we find it in places which have been affected the longest, and where the tubercular process, perhaps, has finally come to a standstill of its own accord. Macroscopically, this cicatricial connective tissue is composed of a thick, firm substance, usually pigmented—the so-called pigment induration. If the cicatricial formation follows a previous extensive destruction of the pulmonary tissue, the affected portion of the lung may thus be diminished to less than half its original bulk. Cavities and firm cicatricial tissue form the anatomical basis of such an extensive "pulmonary contraction." Either the cavities are formed in the usual way from the destruction of lung tissue, or they may be simple bronchial dilatations due to the traction of the contracted tissue—bronchiectatic cavities.

The contractile changes in pulmonary tuberculosis teach us that the tubercular process is in itself capable of healing. The incurability of most cases of phthisis is due to the fact that the infectious material from every existing tubercular nodule is carried into other bronchi, and there sets up a new tuberculosis. Thus the disease is constantly extended. The original tuberculosis, which was localized in one apex only, gradually spreads to the lower portion of the lung. The infectious material is carried by coughing into the trachea, and from this point may be carried by inspiration into the other lung. This becomes diseased, and finally there is such an extensive destruction of the lungs as to make the further continuance of life impossible.

Besides the specific tuberculous lesions in phthisical lungs, there are very often found simple inflammatory processes, such as bronchitis and lobular catarrhal pneumonia; sometimes, also, although seldom of great extent, croupous pneumonia; and finally, in exceptional instances, limited foci of gangrene. These secondary diseases not specifically tuberculous, and yet almost always associated with tuberculosis, are of the greatest clinical importance. They probably are due in most instances to the influence of secondary pathogenic germs (particularly streptococci, less often diplococci, etc.), for which the tuberculosis has merely prepared a favorable soil. Many clinical phenomena (particularly most of the febrile exacerbations of the disease) depend upon these secondary inflammatory processes which, in their turn, promote the further extension of the tuberculosis. Thus, frequently, inflammatory lesions which are due to secondary infection finally are transformed by the invasion of tubercle bacilli into foci of tuberculosis.

If we consider the list of anatomical processes which are found in tuberculosis of the lungs, and which may be combined in the most manifold ways, we can understand the great diversity in the anatomical picture in different cases. Primary tuberculosis of the bronchial wall and tubercular peribronchitis, diffuse cheesy pneumonia, and destruction of the tubercular new growths, with the formation of cavities, on the one hand; contracting interstitial pneumonia, cicatricial formation, and pigment induration on the other—these are the comparatively simple anatomical lesions from which the whole process of pulmonary tuberculosis in its most varied forms is composed. Besides this, we often find here and there one or more miliary tubercles scattered through the lungs, which are probably due very largely to an extension of the infectious material by means of the blood or lymph current; and finally the already mentioned processes of secondary inflammation, such as bronchitis and pneumonia.

The secondary tubercular diseases of the pleura, the bronchial glands, and other organs, will receive a special description.

CLINICAL HISTORY OF TUBERCULOSIS IN GENERAL AND OF PULMONARY TUBERCULOSIS IN PARTICULAR

In judging of the various appearances in the clinical picture of tuberculosis we must especially consider the following points: The place of the first infection is of the chief importance—that is, the place where a local affection, set up by the tubercular poison, first arises. One can readily understand why all those organs which are directly exposed to infection from without are most frequently affected with primary tuberculosis. Very often, as we have said, the lungs are the organs first attacked. Less often the upper respiratory passages—viz., the larynx and nose. In other cases the tubercle bacilli fasten first upon the digestive tract (the intestines, less often the pharynx and the tongue). In many other cases, however, it can not be that the tubercle bacilli directly reach the organ which seems to be first affected. This is true of the so-called tuberculosis of serous membranes, of the tuberculosis of lymph-glands, of bones and joints, of the brain and of the genito-urinary tract. Investigators have yet to determine the paths by which the tubercle bacilli reach the respective organs in these cases. The glance which we have just taken at the organs most frequently attacked by tuberculosis shows the great clinical variety of tuberculous diseases viewed from this aspect.

Another reason for the great variation in the course of tuberculosis is found in the fact that the extension of the local tubercular process may vary very greatly as regards time. Tuberculosis in one case may produce the most extensive destruction in both lungs in a few months or even weeks, and in another case it may remain almost quiescent for years, or advance only very slowly. We do not know fully on what these differences depend, but much is certainly due to the hygienic influences under which the patient lives. In the last analysis, however, we are often led to think of individual differences of constitution, which now check and now favor the rapid extension of the disease.

This predisposition is, in most cases, congenital but sometimes acquired. This is particularly true of alcoholic subjects, who often evince a feeble power of resistance to tuberculosis, although originally of vigorous constitution, so that the rapidly spreading or "galloping" forms of tuberculosis are particularly common in drunkards.

A third and final reason for the differences in the course of tubercular infection is the manner of the further extension of the tubercular poison in the body. As we shall see in the description of tuberculosis in single organs, there are different ways in which tuberculosis may pass from one organ to another. Many contingencies are involved here, and we can easily comprehend how greatly the whole clinical course of the disease must be modified by the rapidity and the degree in which individual organs are affected.

After these preliminary remarks we will pass on to the description of the clinical course of pulmonary tuberculosis.

The onset of pulmonary tuberculosis is quite slow and gradual in the majority of cases. The patient can give only an approximate idea of the time when he began to be ill. The symptoms are referred directly to the respiratory organs The cough and its attendant expectoration are the chief things which affect him. Moreover, there is often pain in the chest, either the pleuritic stitch, or pain in the sternal region, or pain between the shoulder-blades. The patient is also apt to complain of shortness of breath, especially on severe physical exertion.

Besides these symptoms, which point pretty directly to disease of the respiratory organs, there are often quite striking general symptoms. The patient's emaciation is especially noticeable. This may be partly, though not wholly, explained by his loss of appetite. Besides the emaciation there is a steadily increasing pallor of the skin. The patient also shows a general dullness, weakness, and disinclination to work. There is not infrequently a slight rise of temperature in the first stages of the disease, which causes subjective feelings of chilliness and fever. Severe night-sweats may also be noticed early.

Any such constitutional disturbance should lead the physician not to regard the mild thoracic symptoms, which are also present, as insignificant, but to think of the possibility of incipient tuberculosis. It is very important to remember that the pulmonary symptoms may be entirely subordinate to the general symptoms mentioned, and that the patient himself is apt to pay little or no attention to them. Incipient phthisis is therefore frequently diagnosticated as simple "chlorosis" or "gastric catarrh" for a long time, and is treated as such. An early and careful physical examination of the lungs and of the expectoration is the only protection against such an error.

Both the pulmonary and the general symptoms assume significance, if we have to do with a patient in whom we suspect a "tubercular predisposition." We very often meet persons in whose family, either in the parents or the brothers and sisters, several cases of phthisis have occurred. They are persons who are always pale and weak, and who have previously shown a special liability to catarrh and other diseases of the respiratory organs—e. g., pneumonia. They have perhaps had diseases which our present theories bring into direct relation with tubercular infection. We refer to those quite frequent cases of pulmonary tuberculosis in individuals who have previously suffered from "scrofulous diseases," such as chronic swelling of the lymph-glands, chronic affections of the eye or ear, or fungous diseases of the bones and joints, pleurisy, etc. (*vide supra*, page 230).

Although the first symptoms of pulmonary tuberculosis often develop in those who were not quite well before, this is true in only a part of the cases. We often see precisely the same symptoms, both the pulmonary and the general, occurring in persons who previously seemed quite well and strong. No constitution is perfectly protected against the disease. We have even seen the herculean athlete of a circus die of phthisis.

In distinction from the slow and gradual method of the development of tuberculosis which has just been described, the first symptoms in other cases may be more sudden. A definite exposure is often given as a cause, after which the first symptoms of the disease have speedily developed. It goes without saying that we must consider these harmful influences—a chilling of the body, a cold draught, over-exertion, or marked mental excitement—at most, as exciting causes. When pulmonary tuberculosis begins somewhat abruptly, either the initial symptoms are from the start directly referable to the respiratory organs (cough, thoracic pain, dyspnœa), or the chest symptoms may be obscured by the severity of the constitutional disturbance. Thus we recall a number of cases in which young persons fell sick rather suddenly with somewhat severe, febrile constitutional disturbance. At first no cause of the fever could be found, so that the diagnosis was doubtful, or was supposed to be typhoid or some other disease. Then after a time, thoracic symptoms developed and pulmonary tuberculosis could be recognized. In most cases the illness would take a rather rapid course. That form of pulmonary tuberculosis which is termed "pneumonic" (*vide infra*) also exhibits a decidedly acute onset.

Those cases of pulmonary tuberculosis which begin with hæmoptysis are of special practical importance. Of course, it must be understood that the tubercular process in the lung has existed for some time before the blood appears (*vide supra*, page 230). But in the midst of apparent health, or after some slight constitutional disturbance, comes a cough with bloody expectoration. Very often the further symptoms of pulmonary tuberculosis follow directly upon this initial hæmoptysis (*vide infra*).

In conclusion, those cases are to be mentioned in which the first signs of tuberculosis appear not in the lungs but in the larynx. The full description of these cases has already been given in the chapter on laryngeal tuberculosis.

The further course of pulmonary tuberculosis may vary so much that it is impossible to give a complete enumeration of all the possibilities.

In some cases it advances rapidly. We can make out the extension of the disease objectively, almost from week to week. At first the apex of one lung alone is attacked, soon after the lower lobe of the same lung, then the other lung, either at the apex first or in the lower part. Besides the pulmonary symptoms, there is quite a high fever, rapidly increasing emaciation, and general loss of strength. Death ensues in a few months. We term such cases florid phthisis, or "galloping consumption."

In other cases, however, the disease has a remarkably chronic course. Its onset is very gradual, or else, after rather an acute onset, there is a comparative cessation of all symptoms. The thoracic symptoms do not disappear, but they are only trifling, and do not disturb the patient. Physical examination of the lungs does not show any extension of the process for months. The fever which accompanies it is slight, if there be any. The patient remains quite well nourished, but in some cases there is a good deal of weakness. He feels better and worse by turns, his condition being greatly influenced by the care and nursing he receives.

Unilateral contracting phthisis especially has this comparatively favorable course (*vide supra*). The affection remains confined to one lung for a long time. The occurrence of contraction shows the slight tendency of the tubercular process to advance, and with satisfactory care the patient may remain quite well for years.

In cases, too, which have had severe symptoms for a long time, a temporary standstill of the affection may take place, or an actual improvement in all the symptoms. At other times, in cases which have made no advance for a long time, the symptoms suddenly grow worse.

There are all possible varieties between the extremes of florid phthisis and the very chronic cases which last for years and decades. If we recall the further modifications which the course of the disease may assume if complications arise, we can appreciate the manifold character of the clinical picture of phthisis.

Most cases with definite signs of somewhat extensive disease terminate fatally. Death ensues, either with the signs of general exhaustion, or as a result of the final failure of respiration; or it is due to the occurrence of complications, such as tubercular meningitis, miliary tuberculosis, pulmonary hæmorrhage, or pneumothorax. Yet, on the other hand, if the tuberculosis is not extensive, the disease may terminate in complete recovery. To say how frequent recovery is, is difficult, for it is probable that many insignificant cases escape diagnosis. Furthermore, a distinction should be drawn between recovery from a standpoint of pathological anatomy, meaning complete cicatrization, with disappearance of all tubercular new growth, and recovery from a clinical standpoint [arrest], meaning disappearance of all symptoms. Often apparent recovery proves deceptive.

SPECIAL SYMPTOMS AND COMPLICATIONS

1. **Symptoms on the Part of the Lungs.**—*Pain in the Chest.*—Extensive destruction of the lungs may occur without any feeling of pain. Many cases of phthisis are painless throughout their course. In other cases, however, the patient's chief complaint is of severe pains in the side or in the front of the chest. These are probably always due to co-existing affections of the pleura, such as pleurisy, or pleuritic adhesions. In patients who suffer from severe cough, pains sometimes arise in the abdominal muscles and at the insertion of the diaphragm, due to the excessive muscular contraction.

Cough.—In the majority of instances cough is one of the most distressing symptoms of phthisis, but its severity varies very much in different individuals, and at different times in the same patient. We sometimes see cases, particularly in senile, insensitive individuals, in which, in spite of advancing phthisis, cough is remarkably slight, or entirely absent. In cases with severe cough, it is often worst at night, but paroxysms of coughing of long duration are also apt to come on in the morning or evening hours, which are painful and very distressing, and exhausting for the patient. The cough is usually associated with a more or less abundant expectoration, but sometimes there is chiefly a dry cough. The cough becomes very severe if the tubercular affection attacks the larynx and trachea (see laryngeal tuberculosis).

Expectoration.—The amount of expectoration differs very much in different cases. It is most abundant when there is extensive formation of cavities in the lungs. In such cases it is often evacuated in the morning by persistent coughing. The consistency of the great part of the sputum is muco-purulent, and it differs little from that of simple bronchitis; in fact, a large part of the phthisical expectoration comes from the catarrhal inflammation of the bronchial mucous membrane. Usually, however, the amount of mucus as compared with the amount of pus is less in phthisical sputum than in that of simple bronchitis. The sputum is therefore less viscid and more fluid. The amount of mucus in the expectoration is also apt to be greater in chronic bronchitis than in tuberculosis, although mucus is seen in the latter. The expectoration which comes from cavities is almost pure pus, with only a slight amount of serum and mucus intermixed. Such sputum consists often of large separate masses described as nummular, or coin-shaped. If received in water, the uneven, rough surface of these masses is often evident, and suggests their formation in the irregular pulmonary cavities.

The admixture of blood with the sputum is of great diagnostic and practical importance. Since no other disease so often gives rise to the presence of blood in the expectoration, coughing of blood (hæmoptysis) is almost synonymous with consumption among the laity. Little streaks of blood in the expectoration are quite frequent. They have no great significance, but, of course, they may sometimes be the precursors of severe hæmorrhages. Profuse hæmoptysis takes place when the wall of a little pulmonary vessel—almost always a branch of the pulmonary artery—is infiltrated, destroyed, and finally eroded, by the tubercular new growth. The reason why hæmoptysis is not more frequent is because the contents of the vessels usually undergo thrombosis. Severe hæmorrhages very often have their origin in little aneurisms of the branches of the pulmonary artery, which penetrate into the interior of the cavities. In the cases of fatal hæmoptysis we frequently succeed in finding the little aneurism and its point of rupture.

Pulmonary hæmorrhage occurs in all stages of phthisis. The initial hæmoptysis has already been mentioned (page 236). This may be followed by other symptoms of pulmonary tuberculosis, or the hæmoptysis may cease without any immediate sequels. Pulmonary hæmorrhage may also occur at any time in the further progress of the disease. The amount of blood raised is variable. There may be one or more tablespoonfuls, or one or more pints. The blood is bright red, usually quite frothy, but little clotted, and in part mixed with other constituents of the sputum. After the first well-marked hæmoptysis, the expectoration usually is mixed with blood for several days. Again, there may be repeated hæmorrhages in a short time. Sometimes the hæmoptysis begins abruptly, not infrequently at night, without any occasion. In other cases, the hæmorrhage is referable to some distinct cause, such as bodily exertion, a violent paroxysm of coughing, straining at stool, mental excitement, and the like. Many cases of phthisis are characterized by a special tendency to hæmorrhage, while in many others hæmoptysis never occurs. Severe hæmoptyses are, of course, always an undesirable and dangerous complication, since they weaken the patient very much, and also depress his spirits. Many patients maintain their peculiar careless indifference, which is almost characteristic of the disease, despite the spitting of blood. The hæmoptysis may sometimes be the direct cause of death, but, as a rule, the patients survive it. We have no better means of determining with certainty the influence which hæmoptysis has upon the progress of tuberculosis than by the bodily temperature. If there was no fever before, and the hæmoptysis also has an afebrile course, or only a very brief rise of temperature, then we may hope in general that the patient may fully recover from the hæmorrhage and be as well as before. If, however, the hæmoptysis is followed by persistent fever, or if the fever which may have previously existed becomes higher and more persistent, we have every reason to suppose that the tuberculous process is making more rapid advances subsequently to the hæmoptysis. We here append a temperature chart (Fig. 30) illustrating the temporary influence of a hæmoptysis upon the temperature in a stationary afebrile case of pulmonary tuberculosis. The fact that the fever preceded the hæmorrhage by a few days is probably due to the tuberculous arteritis, because of which material capable of exciting fever entered the circulation before the process had caused complete destruction and bursting of the arterial wall, and consequent pulmonary hæmorrhage.

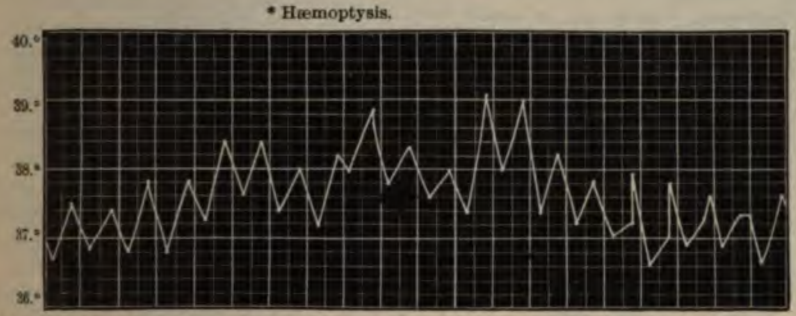


FIG. 30.—Influence of a pulmonary hæmorrhage upon the bodily temperature. (Erlangen medical clinique.)

A purulent sputum intimately mixed with blood is quite frequent and characteristic in many cases of phthisis with extensive formation of cavities. This is formed in the cavities from the mixture of the purulent secretion with little capillary hæmorrhages. In this way the sputum, which is often nummular, assumes a greasy character and a reddish-brown or chocolate color.

If fœtid or gangrenous processes develop in the lungs, the sputum becomes fœtid. In some cases we see temporarily in phthisis the characteristic sputum of croupous pneumonia, which comes from portions of the lung attacked with secondary pneumonia.

Microscopic examination of the sputum may show—besides the ordinary morphological elements, such as pus-corpuscles, red blood-corpuscles, and epithelium—two constitutents which are of decided diagnostic importance: elastic fibers and tubercle bacilli.

The demonstration of elastic fibers in the expectoration permits us to decide with certainty that there is a destructive process in the lungs, and thus it usually is direct proof of tuberculosis. Elastic fibers are also found in pulmonary gangrene, and in the very rare cases of pulmonary abscess, as well as in tuberculosis, but gangrene is easily recognized by the other peculiarities of the sputum. The search for elastic fibers in the expectoration of tubercular patients demands a certain amount of practice. We are most sure to find them if we look in the sputum, when it is spread out, for little lentiform particles, which can easily be discerned with the naked eye. These consist of necrotic shreds of tissue torn off from the walls of cavities. If we press one of these "kernels" under a cover-glass we find, in the midst of the granular detritus, beautifully twisted elastic fibers, which often have quite a definite alveolar arrangement (see Fig. 31). The elastic tissue is the only one spared in the general destruction. There is a special method of looking for elastic fibers. The sputum is boiled in sodic hydrate, diluted with water, and we look for elastic fibers in the precipitate which then forms. We are never justified, however, in deciding that pulmonary tuberculosis is absent because we do not find elastic fibers in the sputum. Their presence is the only thing that has real diagnostic significance.

The discovery of tubercle bacilli in the expectoration of phthisical patients is of much greater importance, and often this alone is decisive (see Fig. 32). They were first demonstrated by Koch, but Ehrlich devised the first simple method for their discovery, which can be easily employed by any physician.

The simplest method and the one which we now employ almost exclusively for staining the bacilli, is as follows: Some of the sputum is rubbed between two cover-glasses, which are then slowly separated, leaving upon each cover-glass a very thin layer of sputum. In order to fasten this sputum upon the cover-glass, the latter is passed three times slowly through a gas flame. The cover-glass is now held with a pair of forceps and covered with the following staining-fluid (carbol-fuchsine solution), which was first proposed by Ziehl and Neelsen: Distilled water, 100 parts; crystals of carbolic acid, 5 parts; fuchsine, 1 part; mix, filter, and add alcohol, 10 parts. This staining solution upon the cover-glass is heated to boiling for a short time, and then the staining is completed. The cover-glass is now washed, first in absolute alcohol, then in distilled water, then placed for about two minutes in a solution of 2 parts of methyl-blue in 100 parts of a twenty-five-per-cent. solution of sulphuric acid. The acid bleaches out the diffused fuchsine stain, leaving the tubercle bacilli still colored, while the pus-corpuscles are colored blue. The preparation is now rinsed in water, dried between two pieces of filter paper, and examined in Canada balsam. The whole process, when one has had a certain degree of experience, requires four or five minutes. The number of bacilli in different cases, and at different times in the same case, varies considerably. The more abundant the bacilli, the more reason have we to suppose there is a rapidly advancing process of ulceration, but of course we can never draw a broad conclusion as to the extent of the tuberculous disease from the number of bacilli in the sputum. Our only information on this point must come from the physical examination, and other clinical observations. On the other hand, the diagnostic significance of the demonstration of bacilli with regard to the recognition of a pulmonary tuberculosis can not be overrated.

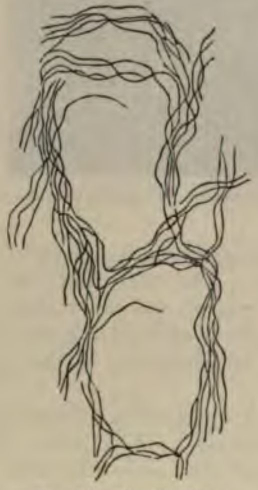


FIG. 31.—Elastic fibers.

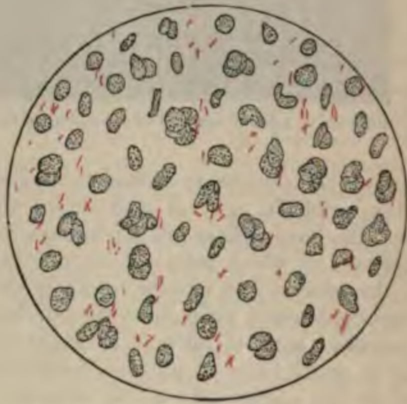


FIG. 32.—Tubercle bacilli in the sputum.

Every positive result is absolutely decisive, and the diagnosis of tuberculosis of the lungs ought certainly never to be made unless the bacilli have been demonstrated. Very frequently bacilli may be found in the sputum in early cases, at a time when no other certain sign of tuberculosis can be detected. On the other hand, we scarcely need to point out that the physician should be cautious in the interpretation of negative results. In all suspicious cases we must repeat the examination of the sputum over and over again.

Dyspnœa.—Many patients hardly ever complain of their breathing in spite of extensive destruction in the lungs. A patient who is much emaciated manifestly needs little oxygen, and the increased frequency of respiration, which is almost constant, can satisfy his needs. If there is a greater demand upon the respiration, a subjective feeling of dyspnœa may of course very readily occur, especially on a slight bodily exertion. In many cases, however, the patient complains of a difficulty in breathing even when quiet, especially if pleuritic pains or adhesions between the surfaces of the pleura prevent him from taking a deep breath; and in the final stages the dyspnœa may be extreme.

2. **Symptoms on Physical Examination.**—In many cases inspection gives us that general impression of the patient which we term the "phthisical habit." The special signs of this are as follows: A slender but often quite a tall frame, weak muscular development, a thin layer of fat, a pale and perhaps very delicate skin with a bluish translucence, sometimes a circumscribed "hectic" flush in the cheeks, a long and slender neck, a long and flat thorax, small, thin hands, etc. Of course this characteristic constitutional habit is not equally well marked in all cases. The inspection of the thorax is of special value. The phthisical or "paralytic" thorax is generally noticeable from its length, but it is narrow and flat. Unusual width of single intercostal spaces, and acuteness of the epigastric angle, are associated with a long thorax. The sternum is also long and narrow, and the sternal angle—Louis's angle—is sometimes particularly prominent. The supra-clavicular and infra-clavicular fossæ are sunken, the neck is wasted, and the shoulder-blades stand out from the thorax. On comparing the two halves of the thorax we very often observe a distinct drawing-in and flattening (contraction) on the side most affected. This change is most frequent in the upper and anterior portions of the chest, but it is not rare even in the posterior and lower.

The paralytic form of thorax is very often seen in phthisis, but it may be entirely absent.

The respiration is usually somewhat accelerated, and sometimes quite markedly so in women with disease of the apices. The feminine type of high thoracic breathing is largely changed to low thoracic or diaphragmatic breathing. A unilateral impairment of respiration is of greater importance; in such a case the apex in front or even the whole side, if there be phthisis of the lower lobe, lags in inspiration. The respiration is sometimes irregular, especially if there be pleuritic pains.

The results of percussion are, of course, entirely dependent upon the sort of anatomical changes in the lungs, and hence differ very greatly in different cases. Since the phthisical process begins in the apices in the majority of cases, our chief attention is turned to the condition of the upper portions of the lungs on percussion. Slight changes in percussion may wholly escape discovery. Only when the air contained in the lung-tissue in the part affected is replaced to a certain degree by the tubercular infiltration does the percussion-note become dull. Unilateral dullness at the apex is therefore one of the most frequent physical signs of phthisis. We can usually make it out most plainly in the upper anterior intercostal spaces first, and in incipient cases often in the supra-clavicular fossæ only, but it is also observed sometimes in the back in the supra-scapular fossæ.

As the infiltration advances the dullness becomes more extensive. For the accurate determination of the limits of dullness in the upper portions of the lungs, we recommend that percussion should be practiced in such a way as to proceed from the normal resonance of the lower portions upward toward the affected area. This method is particularly useful for the back of the chest. Often the dullness takes a tympanitic quality as a result of diminished tension in the lung, or more or less pulmonary retraction. Changes in tension may render the resonance, in beginning tuberculosis, deeply tympanitic without any accompanying dullness.

The formation of cavities in tuberculosis has a great influence on the percussion-note. The dullness on percussion may thus become decidedly less, the degree of resonance depending, of course, upon the fullness of the cavity and the character of the surrounding tissue. We often find a decided tympanitic resonance or a combination of dullness and tympany over a cavity. The different modifications of the percussion-note in cavities are given below. The "cracked-pot resonance," or buckram sound, is met with in percussing over cavities, but we also find it in many other pathological conditions, and in children not infrequently when the lung is normal.

Auscultation also gives no special pathognomonic signs of phthisis. Varying with the character and extent of the tubercular changes, abnormal respiratory sounds and adventitious sounds are heard in place of the normal vesicular murmur. With slighter changes the vesicular breathing is merely modified; it seems remarkably diminished or interrupted, or sometimes exaggerated, with prolonged expiration. When the infiltration of the lungs increases, we find bronchial respiration in place of the vesicular breathing; but, on the other hand, the formation of a cavity is a frequent cause of bronchial respiration.

Various sorts of moist râles are among the most frequent and diagnostically important of the auscultatory signs of phthisis. They are caused by the collection of secretion in the bronchi, or in pulmonary cavities. The more abundant and liquid the secretion is which is set in motion by the air current which traverses it, the more abundant and moister the râles. The larger the space is in which they develop, the coarser they are. Besides the true moist râles, there are also dry bronchitic râles (sibilant or sonorous) occasionally to be heard in portions of the tuberculous lung. In general, we may say that the extent of the tuberculous disease in the lungs can be determined by no symptom so surely as by the auscultatory signs relating to the respiration, and the adventitious sounds which may be present.

Physical Diagnosis of Incipient Phthisis.—The importance of the diagnosis of early phthisis is so great that we will briefly sketch its most important physical signs. Now that the examination of the sputum for tubercle bacilli plays by far the most prominent part in the recognition of incipient tuberculosis, and is the only decisive evidence, the physical signs of this condition have lost much of their former importance. Still, even now, the determination of the seat and extent of the early process is extremely desirable. The auscultatory signs in the beginning of the disease are in general more certain and easier to recognize than those from percussion. He who lays too much weight on the so-called "slight dullness at the apex" will often make a false diagnosis. We will mention the following symptoms: 1. Constant and evident diminution of the respiratory murmur at one apex, especially if it is associated with marked deficiency of the respiratory movement on the affected side. In some cases the respiratory murmur on the diseased side is not weaker, but it has a more indefinite and harsher character; or again it may be rude, sharp, and "puerile." 2. Markedly interrupted respiration at one apex. 3. A prolonged expiratory murmur, which has a harsh character. 4. The discovery of dry rhonchi or moist râles at one apex is most important, since we know by experience that "apex catarrhs" are, as a rule, tubercular. 5.

Definite dullness, apparent on repeated examinations, or tympanitic dullness or tympany at one apex. 6. Evident contraction at one apex, as revealed by inspection or percussion above the clavicles. 7. Some authors lay stress upon a systolic murmur in the subclavian artery, especially loud on expiration. This may occur in the beginning of phthisis, if the caliber of the vessel is narrowed by processes of contraction in the neighboring apex, but this symptom is neither frequent nor of great practical importance.

The chief rule in the diagnosis of incipient phthisis must be held to be this —not to give a definite opinion until repeated examinations have been made. The other portions of the lungs are to be carefully examined as well as the apices, since in not very rare cases tuberculosis may begin in the lower lobes. We must always consider the patient's general symptoms as well as the physical signs (*vide infra*, diagnosis).

Diagnosis of Cavities.—The positive diagnosis of pulmonary cavities by means of physical examination is often very difficult. Any one who will compare the discoveries at autopsies with the results of previous physical examination of consumptives will be in a position to confirm the truth of this statement. We may mention as the chief symptoms of a cavity: 1. Loud bronchial respiration, perhaps of an amphoric character, in places where the percussion-note is only slightly or not at all dull, but perhaps tympanitic. Such a condition means that the bronchial respiration is not due to an infiltration of lung-tissue. Bronchial respiration, however, may of course be heard over cavities which are surrounded by thickened lung-tissue, and hence give dullness on percussion. Pure amphoric respiration is very characteristic of a cavity, particularly if there is a clear, metallic quality to the sound; but this sort of respiratory murmur is heard only when the cavity is comparatively large, of regular shape, and with smooth walls. Under these circumstances the moist râles, if there are any, also have a clearly metallic sound (tinkle), and the percussion resonance may also be distinctly metallic. This last should be demonstrated by rod percussion with simultaneous auscultation. In many cases of this sort the differential diagnosis, between a large cavity and a sacculated pneumothorax (see page 298), is very difficult. 2. The so-called metamorphosing respiration, which begins as vesicular and suddenly becomes bronchial, is heard especially over cavities, and hence has a diagnostic value. 3. The different kinds of "changes in the percussion-note" over cavities are important signs. The most frequent and of greatest practical importance is Wintrich's change of note. This is when the tympanitic resonance, which is obtained over the cavity, becomes, on opening the mouth, more decidedly tympanitic, louder, and especially much higher. The respiratory change of pitch of Friedreich usually consists of a higher pitch on inspiration, but here there are numerous variations. Gerhardt's change of pitch (Weil) consists in a change of the tympanitic resonance when the patient changes his position, the pitch usually being higher when the patient sits up than when he is lying down. 4. Loud, bubbling râles are one of the most frequent signs of a cavity. They are definite indications of the occurrence of râles in a larger space than is normally present in the apices of the lungs. If we have coarse and metallic râles in the apices of the lung, there is considerable probability of the existence of a cavity, inasmuch as the normal apices can not produce such sounds.

Contraction of the Lungs [Fibroid Phthisis].—Unilateral contraction of the lungs, much more frequent on the left than on the right, is a form of tuberculosis which is made apparent both by special physical signs and also by certain clinical peculiarities. It is usually recognized at once by inspection of the thorax, one side of the thorax being remarkably retracted. The upper anterior portions of the thorax, and, in all cases of a high degree of disease, the lower lateral and posterior portions, are much less tense than the corresponding parts on the other healthy side. The fossæ and intercostal spaces on the affected side are deeper, the shoulder-blade is drawn nearer the vertebral column, and the latter is even sometimes drawn over to the contracted side (scoliosis). The resonance is diminished to a greater or less degree, over the affected side, which either lags behind or remains almost wholly at rest on respiration. The respiratory murmur is quite loud, and bronchial; and we also hear many râles, which are usually bubbling. Exceptionally the râles are few and the respiratory murmur feeble and indistinct. Anatomically, we have to do with a marked process of contraction of the interstitial connective tissue in the lungs, which is almost always associated with extensive formation of cavities, partly of an ulcerative, partly of a bronchiectatic character. The pleura is involved in the process almost without exception, but almost always secondarily; it is also thickened and contracted. If the pleuritic thickening is marked, the respiratory murmur and the vocal fremitus are decidedly weakened.

The influence of the contraction on the neighboring organs is very decided, and usually it is easy to discover. The heart especially, whose external pericardium is in most cases very adherent to the pleura, is drawn well over to the side of the contraction. The apex-beat and the cardiac dullness are correspondingly displaced. With left-sided contraction the heart may be drawn over to the line of the left axilla, and with right-sided contraction it may be drawn to the median line, or even to the right of the sternum. With contraction of the left upper lobe the anterior surface of the heart comes into immediate contact with the anterior chest-wall over a larger area than normal. We therefore see the motions of the heart over an abnormal extent, and we can often feel very plainly in the second left intercostal space the pulsation and the closure of the valves in diastole of the pulmonary artery. The upward traction of the diaphragm may be recognized by the position of the liver, or, in left-sided contraction, by the increase of the "semilunar" tympanitic space on the left. We usually find the sound lung on the other side quite emphysematous, as shown by the downward displacement of the lower boundary of the lung, and also by the drawing over of the anterior median edge of the lung to the contracted side. In a part of the cases we can make out by percussion the development of consecutive dilatation and hypertrophy of the right ventricle.

These are the chief physical signs of the so-called unilateral form of chronic pulmonary contraction. We would add here a few clinical remarks. These cases often, but of course not always, run a very chronic course, lasting for years. The general condition and the nutrition of the patient may thus remain comparatively undisturbed for a long time. The patient looks somewhat pale and cyanotic, yet he is so well nourished as to present a very marked contrast to the appearance of the ordinary cases of phthisis. The appetite remains good, the fever is entirely absent, or else a slight degree of fever may be at times discovered by careful examination. The cough and expectoration, too, though often quite troublesome, are at other times very slight, especially when the patient has good care and nourishment. We need not wonder, then, that formerly many physicians did not consider that these cases had anything to do with phthisis—"consumption"; and yet we are convinced by many clinical and anatomical observations that, ætiologically, they are in by far the greatest part tubercular. They represent a very slow form of tuberculosis, which has time to develop interstitial processes which lead to contraction—that is, to local healing. If such cases come to autopsy, their tubercular character is usually definitely confirmed. We find undoubted tubercular lesions in the other lung and also in the remaining organs—e. g., the intestines. Sudden changes for the worse may occur in every "pulmonary contraction," even those cases which seem favorable; the other lung may become highly tubercular, a miliary tuberculosis or a tubercular meningitis may develop, etc. On the whole, however, the slow course of this form of chronic tuberculosis is characteristic and of great practical significance, and its prognosis is therefore comparatively favorable.

We can not deny that a non-tubercular unilateral contraction of the lung may occur. As a result of bronchitis (particularly in cases due to the inhalation of dust), and also after pleurisy, processes of contraction develop, which are associated with the formation of bronchiectasis, and certainly have nothing to do with tuberculosis. In rare cases also croupous pneumonia is followed by unilateral contraction of the lung; and finally there is a rare and by no means satisfactorily investigated form of unilateral chronic interstitial pneumonia, with contraction, often associated with the formation of bronchiectasis. The differential diagnosis of these conditions from tuberculous contraction rests in part upon the clinical history, which should be carefully taken, but mainly upon the absence of tubercle bacilli in the expectoration.

In conclusion, we must mention that there are very many transitional forms between pulmonary contraction and the other varieties of pulmonary tuberculosis. We find more or less extensive processes of contraction in one apex in most cases of phthisis.

Disseminated Pulmonary Tuberculosis.—There is a form of pulmonary tuberculosis which it is very hard to make out on physical examination. In this we have to do with numerous peribronchial nodules disseminated over the whole lung. As there is still a good deal of normal tissue, containing air, between these nodules, percussion affords no dullness, and auscultation gives at most diffuse rhonchi; hence this form is often confused with chronic bronchitis or pulmonary emphysema. The diagnosis can seldom be made from the physical signs, but only from the other symptoms, such as fever, emaciation, striking pallor of the skin, and the sputum.

This form of phthisis sometimes runs a chronic course, but usually it is quite rapid. It occurs in elderly people, and also in children. Many forms of "disseminated, coarse granular" tuberculosis are transitional forms between this and genuine acute miliary tuberculosis.

Pneumonic Form of Pulmonary Tuberculosis.—A special clinical interest attaches to that form of pulmonary tuberculosis termed pneumonic, which we have already briefly mentioned. The disease begins in a decidedly acute manner with dyspnœa, frequent cough, and pain in the side. Sometimes we observe even a distinct initial chill. The expectoration is scanty, viscid, mucous, and often reddish or hæmorrhagic. It sometimes also has a peculiar greenish color. Even after a few days of illness we find, on examination, the well-marked signs of lobar infiltration. There is first pneumonic resonance, then a dull tympany, with fine moist râles and bronchial breathing. In most cases, but not all, a lower lobe is involved. The disease is almost invariably regarded at first as a croupous pneumonia, but the expected crisis does not appear. The fever remains high, the infiltration does not undergo resolution, the râles become coarser, the patient looks pale and wretched. Now, suspicion of tuberculosis being aroused, the sputum is carefully examined, and soon, although perhaps not at the first examination, tubercle bacilli are found. All cases of this sort take a rapid and unfavorable course. They may be called galloping consumption. Upon autopsy we find diffuse tubercular infiltration, and usually incipient cavity formation in numerous places. Other portions of the lungs than that first and most violently attacked ordinarily present considerable tuberculous change. In these instances it is evident that we have to do with an acute infection of a large portion of the lung with tubercle bacilli, which of themselves exert a powerful inflammatory action, with unusual virulence. In rare cases there may also be a mixed infection, due to the pneumococcus.

3. General Symptoms in Pulmonary Tuberculosis.—In the description of the general course of pulmonary tuberculosis we have already mentioned the value of the constitutional symptoms in diagnosis and prognosis.

Fever.—Only a few cases of phthisis run their course entirely without fever, but it may be absent for a time, even for weeks and months; and indeed, in cases with a very slow and favorable course (e. g., where there is unilateral contraction), there may be no fever at all for years. The more carefully we measure the temperature the more often shall we find a slight evening rise up to 100° or 101° (38° to 38.5° C.), or at least between 99° and 100° (37.5° to 38° C.), even at times when the patient is doing well. Many cases that pursue a comparatively favorable course have for a long while a so-called sub-febrile condition. That is, the bodily temperature is normal in the morning, but in the evening invariably rises to about 100° or 100.7° (37.8° to 38.2° C.), or even higher. (See Fig. 33).

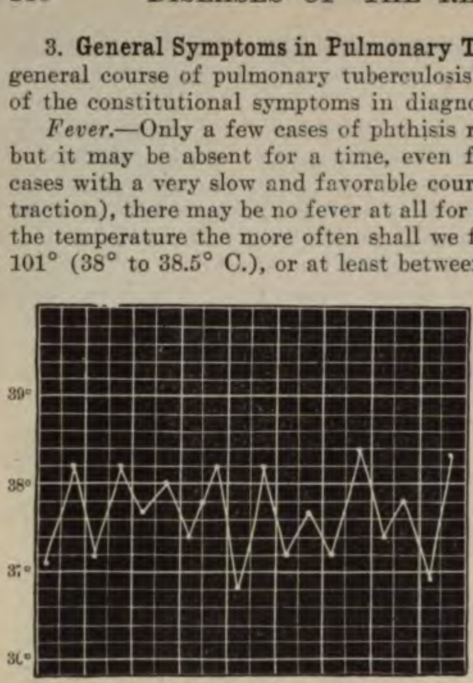


FIG. 33.—Sub-febrile state in chronic pulmonary tuberculosis.

Very often, particularly in the more rapid cases, there is considerable elevation of temperature, and the varieties and course of the fever are more or less characteristic. We should first mention the "hectic fever" (see Fig. 34) which is often observed. For months the temperature chart may present a uniform appearance with a morning temperature approaching or reaching the normal, while there is a regular elevation every evening to 102° or 104° (39° to 40° C.). In general, the higher the evening exacerbations the more unfavorable the case. In other cases the temperature chart is quite irregular; longer or shorter persistent periods of elevation alternate with periods when there is no fever. Particularly toward the end of the disease, as the bodily weakness increases, the previously regular, intermitting temperature grows irregular. At this time the intermissions often become more marked, and not infrequently general collapse temperatures—95° to 93° (35° to 34° C.)—are observed. Again, the fever may for limited periods take on a more continuous character, probably in connection with exacerbations of the tuberculous process. In some few cases with acute onset (*vide supra*) we have also seen, in the beginning of the disease, a tolerably high and approximately continuous fever, passing gradually into the ordinary *febris hectica.* The cause of the fever in pulmonary tuberculosis is not yet settled. The special question is whether the tubercular process causes the fever of itself (by production of toxines), or whether the fever is due to the secondary inflammatory processes, such as the absorption of septic and toxic material from the decomposing contents of the bronchi and the pulmonary cav-

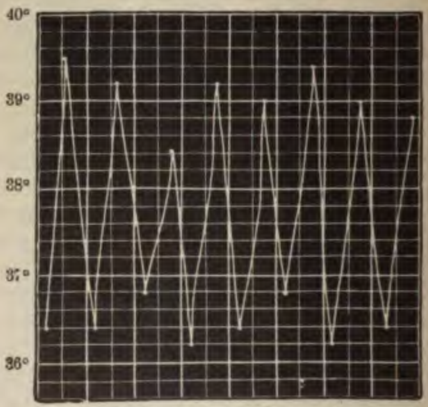


FIG. 34.—Hectic fever in chronic pulmonary tuberculosis.

ities. The practical importance of the fever in pulmonary tuberculosis is very great. The emaciation and weakness of the patient are caused mainly by fever (*vide infra*), as are many subjective symptoms, including headache, chilliness, and perspiration. It is important to remember that in observations of the fever we possess one of the most positive means of forming an opinion as to the course of pulmonary tuberculosis. In cases which are either completely stationary or very chronic there is no fever at all. The sub-febrile condition shows a slow, but still continuous, advance of the disease. Continuous hectic fever is an unfavorable sign, and indicates a comparatively rapid march of the disease—the more rapid, the higher the evening temperature. In the florid or galloping forms we find tolerably high fever, at times continuous and at times remittent. Subnormal temperatures are almost always of bad omen.

All changes in the general course of the disease for better or worse stamp themselves clearly on the temperature chart. The onset of complications and secondary disease is often first indicated by the thermometer. Whether an attack of hæmoptysis passes over without permanent damage, or whether it is followed by an aggravation of the disease, can usually be told by the temperature sooner than in any other way (*vide supra*, page 238). Thus we see that the persistent and careful observation of the temperature in consumption deserves to rank as one of the most important means of estimating the status and course of the illness.

Emaciation.—The great emaciation of the patient is very striking in most cases of phthisis. The muscular system and the fatty tissue are affected in equal degree. The soft parts of the thorax are often especially involved. The emaciation is due in part to the patient's loss of appetite, and to the small amount of food which he takes in consequence thereof, but the chief cause lies in the persistent fever and the increased metamorphosis of tissue. Quite a high degree of emaciation, however, may appear in the beginning of the disease with no fever. This we are wont to ascribe to the "general illness," but the special cause of it is unknown. Under favorable external conditions phthisical patients may make quite a decided gain in weight, especially at the times when they are free from fever. In very chronic cases, which run their course from the first without fever, the nutrition of the patient may remain good for a long time. Toward the end of the disease emaciation sometimes reaches its highest degree, and many phthisical patients die "wasted to a skeleton" in the true sense of the word.

Anæmia—Color of the Skin.—In most cases anæmia appears in the course of the disease, to be recognized by the pale and sallow color of the skin and of the visible mucous membranes. The anæmia only rarely reaches that degree of peculiar waxy pallor that is found in idiopathic pernicious anæmia. If the pallor is extreme, however, there is usually some special reason, such as profuse hæmoptysis or the complication of amyloid degeneration. The existence of the anæmia is also the reason why the phthisical patient does not look cyanotic in spite of the respiratory disturbance. In the more chronic forms, where the general nutrition suffers less, we often see a cyanotic coloring of the lips and cheeks. Sometimes the skin of phthisical patients assumes a dirty, dusky hue. We have already spoken of the circumscribed "hectic flush of the cheeks" seen with the fever.

General Weakness—Night-Sweats—Nervous Disturbances.—We need not say that the general emaciation and anæmia are accompanied by a marked decline in the patient's power of endurance. He finally becomes so helpless that he can scarcely move alone in the bed.

The tendency which very many patients have to severe night-sweats is universally acknowledged but not wholly explained. It may have some connection with the fall from the evening febrile temperature to the morning remission, and perhaps it is due to the greater accumulation of carbonic acid in the blood from the disturbance of respiration.

The disease has remarkably little influence upon the higher nervous functions, especially those of the mind. Most patients have a perfectly clear intellect to their latest breath. We all know the contented, hopeful, sanguine disposition of many patients, who do not recognize their own danger until the last stages of the disease. Occasionally the anæmia and the general disturbance of the nutrition of the brain lead to mental alterations, such as confusion, distraction, or melancholic conditions.

We find, more frequently, disturbances in the peripheral nerves and muscles. Among these are neuralgic pains, and pains of an indefinite character, which have their seat in the legs, or sometimes in the arms, especially in the ulnar region and the sciatic nerve. These may be very distressing. Marked hyperæsthesia of the skin and deeper parts is also not uncommon. The cause of such disturbances is probably often to be looked for in the degenerative changes in the peripheral nerves (Vierordt and others). Well-marked multiple neuritis has been repeatedly observed in tuberculous patients (see section on nervous diseases).

We very often see an increased reaction upon direct mechanical irritation in the emaciated muscles, and great liveliness of the so-called idiomuscular contractions, which is shown, for example, on percussing the pectoral muscles on the anterior wall of the chest. The phenomena grouped under the name of tendon reflexes are also much increased in phthisis.

4. **Symptoms and Complications on the Part of Other Organs.**—1. *Pleura.*—In pulmonary tuberculosis the pleura is also involved as a rule. The affection is almost always the result of a direct extension of the process from the lung to the pleura. At the autopsy, we find in the pleura a few or many miliary tubercles, besides the simple inflammatory process—tubercular pleurisy.

In many cases, in which we have to do only with an adhesive pleurisy and with pleuritic contraction, we can merely suspect the disease of the pleura, but it can not be directly made out and differentiated clinically from the pulmonary affection. In other cases we can diagnosticate a dry pleurisy in phthisis from the occurrence of the pleuritic friction-rub. The symptoms of pleurisy become more marked if there is a pleuritic effusion, which is usually readily discovered by a physical examination. The patient's pain and dyspnœa are usually much increased by such a complication. Besides a simple sero-fibrinous effusion we quite frequently find purulent and even hæmorrhagic effusions in tuberculosis of the pleura.

The formation of pneumothorax is an important complication in the pleura in phthisis. This is due to the rupture of a superficial pulmonary cavity into the pleural cavity, and the entrance of air into the latter. The different forms of pneumothorax and its symptoms will be described under diseases of the pleura.

2. *Larynx, Pharynx, and Trachea.*—The symptoms of laryngeal tuberculosis and their relation to pulmonary tuberculosis have already been given under diseases of the larynx (see page 148). We saw there that, although there is a primary laryngeal tuberculosis, most cases are secondary in their development to a pulmonary tuberculosis.

The same holds true in regard to the much rarer tuberculosis of the pharynx. In some cases this may be of primary origin, but it is usually a result of re-inoculation with tuberculosis by means of the sputum, or of a direct extension of the tubercular process from the larynx to the pharynx. Tubercular ulcers of the pharynx are found most frequently on the soft palate, on the tonsils, on the root of the tongue, and on the boundary between the pharynx and the larynx; they are rare in other parts of the pharynx. In exceptional cases tubercular affections are seen in the mouth—on the tongue. The local discomforts which all these ulcers cause is usually very considerable. Disseminated miliary tubercles, too, have been repeatedly seen in the mucous membrane of the pharynx.

3. *Stomach and Intestinal Canal—Peritoneum.*—Tubercular ulcers in the mucous membrane of the stomach are exceedingly rare, but we very often notice some symptoms on the part of the stomach. Loss of appetite is a particularly common symptom in phthisis. Vomiting occurs often in phthisical patients, especially when the larynx is involved. It is usually brought on by paroxysms of coughing. Less frequently the cause of the vomiting is gastric catarrh, occasioned by the irritation of the sputa which have been swallowed; but in some cases the gastric symptoms depend upon the general condition—e. g., the anæmia.

Although the tubercle bacilli swallowed with the sputum hardly ever infect the stomach, probably from the acid reaction of its contents, they very often attack the intestinal canal. In the majority of the cases of phthisis we find tubercular ulcers, either singly or in considerable numbers, in the vicinity of Bauhin's valves [the ileo-cæcal valve], in the lower part of the ileum, and the upper part of the large intestine.

Intestinal tuberculosis does not always cause very marked clinical symptoms, but as a rule we find diarrhœa in patients with tubercular ulcers of the intestine. They may have three or four stools in the twenty-four hours, and even more, but the stools have nothing characteristic. We rarely see a slight admixture of pus or blood in them. Tubercle bacilli have been repeatedly discovered in the dejections, but the search for them is rather difficult. We must call attention, however, to the fact that many patients have diarrhœa during life in whom we find at the autopsy no intestinal tuberculosis, but only a simple intestinal catarrh. Severe diarrhœa of a persistent and refractory character may also occur in association with amyloid degeneration of the intestine, which is not infrequently seen in connection with other amyloid changes. Sometimes tuberculous ulcerations of the intestine are found at autopsy which, during life, had caused no diarrhœa.

In cases of severe intestinal tuberculosis we sometimes meet with meteorism. With deep ulcers, extending to the peritoneum, we often see marked tenderness of the abdomen.

The peritoneum may be affected by the tubercular ulcers of the intestine in a twofold manner. Genuine peritonitis from perforation, with a purulent or even a sanious exudation, is quite rare, and is excited by the rupture of an ulcer and the entrance of the contents of the intestine into the abdominal cavity. An infection of the peritoneum with the tubercular poison is more frequent. This may arise from deep-seated ulcers, which do not reach actual perforation, so that we have a peritoneal tuberculosis, or a tubercular peritonitis. During life peritonitis from perforation and that from tuberculosis are not always to be distinguished. We must also mention that simple ascitic fluid is sometimes found in the abdominal cavity in phthisis, which may lead to a false diagnosis of peritoneal tuberculosis.

Another way in which we may have a peritoneal tuberculosis in the course of phthisis is from the extension of the process in a tubercular pleurisy, through the diaphragm to the peritoneum.

4. *Liver and Spleen.*—We very often find a few or even many tubercles in the liver in phthisis, but they have no clinical significance. The liver is almost always infected with the tubercular poison from tubercular ulcers in the intestines, from which the poison passes to the branches of the portal vein and then to the liver. Fatty liver and amyloid or lardaceous liver are more important clinical changes. We can sometimes recognize the former by making out on physical examination the increase in the size of the organ, and by feeling its characteristic blunt lower edge. Moreover, we must emphasize the fact that, in our experience, fatty infiltration of the liver is found much more rarely at the autopsy of consumptive subjects than the statements of many earlier authors would lead one to expect.

Amyloid degeneration of the liver appears almost always in association with amyloid change in other organs (kidneys, spleen, intestine). If the amyloid degeneration is advanced, the liver is considerably enlarged, and it is usually possible to feel distinctly its sharp and resistant edge; and, not infrequently also, its firm upper surface.

Miliary tubercles or single large tubercular nodules in the spleen have a pathological interest only. Great splenic enlargement is found in amyloid degeneration.

5. *Kidneys, Urinary Passages, and Sexual Organs.*—The presence of miliary tubercles in the kidneys is the first change in them to be mentioned, but it has no clinical significance. Extensive tuberculosis of the genito-urinary apparatus, however, may produce marked symptoms, such as pyuria. Genito-urinary tuberculosis will be discussed in a later part of this work. In regard to the symptoms of amyloid kidney, which may develop in the course of phthisis in connection with amyloid disease in other organs, we will refer to the section on renal diseases.

Genuine cases of nephritis, both acute and chronic, are also found quite frequently in phthisis, often combined with amyloid disease. These can not escape notice if the urine is carefully examined. Their development is probably always referable to the excretion of toxic material, arising from the disease in the lung.

6. *Circulatory Organs.*—The rate of the pulse is often increased in consumption. This increase in frequency may be merely proportional to the fever, if any exists. It is also usually seen and it may be considerable when there is no fever. A persistently rapid pulse, when the temperature is little, if at all, elevated, may therefore be an important diagnostic sign of pulmonary (and all other) tuberculous diseases. The increase of the pulse, which readily comes on from comparatively trifling external causes, is especially noteworthy. It may be seen after slight physical exertion, or upon mental excitement, as during the physician's visit.

Anatomical changes in the heart are rare, except that it is often remarkably small and flaccid. Moderate fatty degeneration of the heart, slight endocarditis of the valves, or occasional tubercles in the heart, causes no symptoms. The occurrence of tubercular pericarditis, however, is important. This almost always arises from the extension of the tubercular process from the adjacent pleura, but in exceptional cases pericarditis has been seen as a result of rupture of a tuberculous lymph-gland or a pulmonary cavity into the pericardium.

7. *Lymph-glands.*—The lymph-glands are a favorite seat for tubercular changes. We have stated above that the so-called scrofulous, cheesy lymph-glands, which are seen chiefly in the neck and the axillæ, are affected with tubercle in the majority of cases. The tubercular infection probably develops here from slight injuries and excoriations of the skin, by which the bacilli enter the body and reach the neighboring glands by means of the lymph-current. In other cases the infection comes perhaps from the mucous membrane of the pharynx. In tuberculosis of internal organs, too, we very often find the corresponding lymph-glands enlarged and more or less cheesy. The bronchial lymph-glands are swollen as a result of pulmonary tuberculosis, the mesenteric and retroperitoneal glands as a result of intestinal tuberculosis. The tuberculosis of the bronchial lymph-glands is of especial importance in children. Indeed, the tubercular virus which has gained access to the lungs may apparently reach the bronchial glands by means of the lymph-channels even without affecting the lungs themselves, and occasion a tuberculous disease of the glands. Glands thus diseased break down and discharge into the lungs, and in this way generate a secondary pulmonary tuberculosis. This is one reason why the pulmonary tuberculosis of

children so often begins not in the apex but in the middle or lower portion of the lung.

Pressure from the enlarged glands may affect the air-passages, the branches of the pulmonary artery, the veins, the recurrent nerve (paralysis of the vocal cords), and even the aorta. Perforation of the cheesy bronchial glands into the œsophagus, the blood-vessels, etc., has also been observed. Tuberculosis of the bronchial glands does not present any definite type of disease, however, and, although we may sometimes suspect it when there is pulmonary tuberculosis, we can only rarely diagnosticate it with certainty. The author himself observed a noteworthy case of tuberculosis of the bronchial glands, with compression of one vagus nerve in a patient who, during life, for weeks coughed up large amounts of a purely sero-mucous expectoration containing no bacilli.

8. *Nervous System.*—We have already mentioned various nervous symptoms in the description of the general symptoms. We must also add that tubercular meningitis is seen in the course of phthisis (see page 1059), and that large solitary tubercles may occasionally develop in the central nervous system.

9. *Skin.*—We have spoken of the great tendency which many patients have to severe sweats, especially at night. The frequent appearance of pityriasis versicolor, especially on the skin over the thorax, is also worthy of note. We often see moderate œdema of the legs and ankles, which is due to weakness of the heart. More marked œdema of one leg sometimes arises from thrombosis of the femoral vein. We must also mention here, in conclusion, the specific tubercular disease of the skin—lupus. This occurs alone, as a rule, without a co-existing pulmonary tuberculosis; but, on the other hand, the old term "scrofulous lupus" had reference to the fact that we often find other tubercular affections in lupus besides the disease of the skin. Thus it does not seem strange that lupus and phthisis have been repeatedly observed to co-exist. Cutaneous tuberculosis may develop not only in the ordinary form of lupus, but also in nodules of considerable size, or in rather extensive ulcers. Certainly some of the cases of so-called corpse-tubercle belong to true tuberculosis. We have seen a similar tuberculous cutaneous disease in a woman who for a long time had washed the soiled handkerchiefs of a consumptive patient. We have also seen a tuberculous affection of the skin upon the chin of a patient very ill with consumption. In this case, no doubt, the skin became infected by the sputum.

Diagnosis.—The diagnosis of pulmonary tuberculosis has become absolutely certain, since the discovery of the tubercle bacilli, by the demonstration of their presence in the sputum (*vide supra*). In all incipient cases, in which the other symptoms of the disease have not yet made themselves manifest, but the suspicion of incipient phthisis has been aroused by a persistent cough, by marked pallor and emaciation, by slight hoarseness, by an evening rise of temperature, by the appearance of night-sweats, by the presence of a hereditary predisposition, and similar symptoms, the finding of tubercle bacilli in the sputum is often the sole deciding factor. Still, it must not be forgotten that the examination for bacilli is decisive only when its result is positive, and that the greatest attention should be given also to all the other symptoms. To form an opinion as to the severity of a particular case, and as to its exact distribution and the variety of the tubercular process, is at present possible only by means of a consideration of the other symptoms, and in particular of the results of physical examination. The latter, therefore, has lost none of its importance by the discovery of the tubercle bacilli.

Confusion between phthisis and other diseases is twofold. Where the constitutional symptoms are predominant, and there are no marked pulmonary symptoms, an existing tuberculosis may be overlooked. In the beginning, especially, many cases of phthisis are considered to be merely anæmia, chronic gastric catarrh, or simple bronchitis. If a continuous or intermitting fever appears in an early stage of phthisis, before any marked pulmonary symptoms have developed, the disease may be mistaken for malaria or the like. On the other hand, it is by no means rare to consider patients phthisical who are suffering from some entirely different affection. He who lays too great stress on the uncertain results of percussion will often make a false diagnosis. Severe latent diseases of the stomach, or certain general diseases, such as anæmia, diabetes, or chronic nephritis, may be mistaken for phthisis. Other pulmonary affections, too, may be confounded with tuberculosis, especially chronic bronchitis, emphysema, bronchiectasis, fœtid and gangrenous processes, and carcinoma of the lungs. A careful, unprejudiced, and complete examination of the patient is the only possible protection against such errors. It is very important for the physician to know that there is such a thing as hypochondriacal phthiseophobia. Some nervous individuals are tormented by a constant dread of becoming consumptive. Such persons complain of thoracic pain, dry cough, weakness, and other imaginary symptoms, which might readily mislead the physician into supposing that they actually have incipient pulmonary tuberculosis. Of course, the correct interpretation of such conditions is usually no difficult matter to the experienced physician, who is guided by the general impression of "nervousness," and by the complete absence of any objective physical signs.

In this connection we ought, furthermore, to express our opinion of the diagnostic value of Koch's tuberculine (*vide infra*). Koch made the important discovery that tuberculous patients, particularly those suffering with pulmonary tuberculosis, exhibit a particular "reaction" after the injection of small amounts of tuberculine (gramme 0.001 to 0.002 of Koch's preparation). When this reaction occurs there develops, some four or five hours after the injection, a fever of 102° to 104° (39° to 40° C.), associated with chilliness, headache, pain in the limbs, nausea, and languor. This constitutional reaction passes off after some twelve or fifteen hours. If the tuberculous process is located in the skin, trachea, or some other part where it is accessible to direct observation, a very decided local reaction may usually be observed besides the constitutional disturbance. The tuberculous tissue becomes swollen, red, and finally in part necrotic. In patients with tuberculosis of the internal organs, also, this local reaction probably takes place, but of course it can not be actually seen, although it may perhaps be inferred because of the secondary phenomena. Thus, in pulmonary tuberculosis there may be an increase in the cough and expectoration. On the other hand, if an injection of tuberculine is made in a person who is healthy, or who is suffering from some disease other than tuberculosis, then, according to Koch, there will be no reaction at all to small doses. To obtain any reaction in persons who are healthy, or, at any rate, not tuberculous, it is said that the dose of Koch's preparation must be increased to the amount of gramme 0.01. If these statements were true, the appearance or the absence of a reaction after the injection of a small amount of tuberculine would be an important and, indeed, a decisive factor in determining whether tuberculosis did or did not exist in any given case.

These statements of Koch have been shown, however, by extensive investigations, to be true only in general, and not in every case. Other observers as well as the present author have occasionally found that healthy persons, or persons free from tuberculosis, have reacted to doses of one or two milligrammes of tuberculine; while, on the other hand, patients with indubitable pulmonary tuberculosis have shown no distinct reaction even to considerable doses of tuberculine. Consequently the result of an injection of tuberculine can never be completely decisive from a diagnostic point of view, and in practice, therefore, the employment of tuberculine for diagnosis has been adopted by only a few; but still, it would be a mistake to discard this means of diagnosis completely. In doubtful cases when we can not be sure of the diagnosis, the injection of about two milligrammes of tuberculine is of real importance, and the result of the test is certainly a valuable factor in summing up such a case; yet the verdict is never so unimpeachable as in cases in which the tubercle bacilli have been discovered.

Prognosis.—It is very difficult to make a general statement as to the prognosis of pulmonary tuberculosis. There is no doubt that tuberculous foci in the lungs, if of limited extent, may become completely healed, at least from a clinical point of view. Indeed, such recovery probably takes place oftener than many suppose; at least, it is not very exceptional to find at the autopsy of elderly persons cicatricial contractions in the apices of the lungs which may, without doubt, be regarded as healed tuberculosis. In many of these cases the previous tuberculosis had never come to the knowledge of any physician. At the present day the discovery of tubercle bacilli in the sputum has rendered the diagnosis of even very limited tuberculous processes an easy matter, and, consequently, cases of recovery from pulmonary tuberculosis are now recognized much more often than formerly.

Nevertheless, the prognosis of pulmonary tuberculosis must always be regarded even now as very serious, and when the disease has made any great progress the prognosis is decidedly unfavorable. Many cases of apparent recovery exhibit merely a temporary improvement, to grow worse again, and the assertion that treatment is absolutely hopeless in almost all cases in which the disease has reached an advanced stage is, unfortunately, too well established to require elaborate argument.

There is, however, one fact of extreme importance which should never be disregarded in the prognosis of pulmonary tuberculosis. There is a great difference in the duration of the disease. It is possible for the process to go on for years, while the patient feels tolerably well, and yet the disease smoulders. From this point of view cases may be distinguished as "benign," compared with those of rapid progress, and this prognostic distinction has the greatest importance in practice, although it is often very difficult to recognize. Many a consumptive, when first examined, gives one the impression that he can not live a fortnight longer, and yet the disease lasts many months, with improvement in most of the symptoms and general alleviation of the patient's condition. Again, one believes the disease to be incipient, encourages the family, and the patient dies in a few weeks of galloping consumption.

Certain complications may occur which it is impossible to foresee, such as pulmonary hæmorrhage, pneumothorax, tubercular meningitis, and miliary tuberculosis. Apart from these, however, there are certain conditions which lead us to expect a comparatively favorable course for the disease: such are a vigorous constitution unimpaired by bad habits, of which drinking is particularly unfavorable; a good weight; freedom from hereditary taint; limited extent of the local process; later life; the absence of complications; and, finally, the persistent absence of fever. This last factor is so important that we must lay special stress upon it. If there is no fever whatever in a case of pulmonary tuberculosis, the patient is at that time in a condition in which the disease is quiescent; and a decided improvement, perhaps even cure, may be expected if appropriate means are employed. On the other hand, whenever fever appears we know that the disease is not quiescent, but is actively advancing with more or less rapidity. The important points about the temperature in this regard have been already emphasized (see page 246). It is self-evident, also, that the worldly circumstances of the patient, as well as the factors which we have just alluded to, are of great prognostic importance. On these, for instance, depends the possibility of proper care, of abundant nourishment, and, it may be, of a change of climate.

Treatment.—1. *Prophylaxis.*—The question of what prophylactic measures may effectually prevent the extension of the disease has entered upon a new stage since our definite knowledge as to the infectious nature of tuberculosis. We can no longer doubt the contagious character of phthisis, in support of which isolated examples were previously brought forward. Even if, according to all experience, the danger of contagion is not very great, still it is foolish to ignore it entirely. We must therefore call the attention of the relatives of phthisical patients to the possibility of this danger, and we should not permit the children of such patients to be uselessly exposed to it. We should take satisfactory precautions for isolation, and also for disinfection of the sputum. Suitable sputum cups should be employed, and care should also be taken to prevent the dissemination of the expectoration, either in its fresh state or after it has become dry. The future will teach us whether much evil may not be averted by such measures.

The "prophylaxis" till now employed was almost exclusively confined to hardening and strengthening the threatened individual as much as possible. We should try to strengthen the bodies of children of a weak habit, with "scrofulous" symptoms, and children from families in which cases of tuberculosis have already occurred, and thus to arm them against the enemy that threatens them. Good food, fresh air, and a diminution of the sensitiveness of the body by cold sponging and cold baths—these are the factors whose favorable influence is generally recognized.

The removal of certain foci of tubercular disease, already existing, from the body may prove of great prophylactic importance. We refer to the timely treatment or extirpation of scrofulous—that is, tubercular—swellings of the lymph-glands, healing or resection of tubercular bones and joints, etc. Although in individual cases we can of course never know whether the part removed is the sole focus of disease in the body, still we are undoubtedly justified in trying to remove at least one possible source of some later general infection. A fuller discussion of this important point must be left to the works on surgery.

2. *Therapeusis.*—Physicians have often thought that they had discovered a specific remedy for tuberculosis, but apparently they have thus far been mistaken. Formerly, the inhalation of antiseptic substances, such as carbolic acid, benzoate of sodium, and iodoform, was recommended, but this practice is now almost entirely given up. Arsenic was for a time much used, best given in pills containing gr. $\frac{1}{20}$ (grm. 0.003) repeated several times a day; but this practice, again, has not held its own. Arsenic may be tried in incipient cases, particularly those associated with marked anæmia, but great curative influence is not to be expected from it. Creasote has won far more advocates. Continued for a considerable time in large doses (fifteen to thirty grains, grammes 1 to 2, or more, in the course of twenty-four hours), it is regarded by many physicians as an excellent remedy in incipient and even in advanced pulmonary consumption. It is best prescribed in gelatine capsules, or in a mixture of one part of creasote to two parts of the tincture of gentian, of which twenty to eighty drops may be given three times a day in a considerable amount of milk, or in wine. This remedy is usually fairly well borne, and the patients are pleased with the improvement it causes in their appetite and general condition. Of late, physicians have employed guaiacol, the active ingredient of creasote, instead of the drug itself, especially since carbonate of guaiacol has been brought forward, a preparation which has very little of the disagreeable odor and taste of creasote itself. Carbonate of guaiacol is administered in powders containing about eight grains (gramme 0.5), in daily doses at first of twenty-five grains (gramme 1.5), gradually increasing to thirty or forty-five grains (grammes 2 to 3). Dyspeptic disturbance sometimes follows the use even of guaiacol, but still this remedy is, in general, better borne and more read-

ily taken than creasote. As to the specific and therapeutic use of guaiacol in pulmonary tuberculosis, it is not easy to form a decided opinion. It is certain that many patients praise these remedies, and improve considerably while using them; but, on the other hand, the indiscriminate laudation of many authors is decidedly unjustifiable. When the disease is slowly progressing with persistent sub-febrile temperatures, the author has scarcely ever felt sure of any distinct influence exerted by guaiacol or creasote upon the temperature and the disease. Nevertheless, it is often advisable in practice to make trial of these remedies, particularly of the carbonate of guaiacol. The remedy must be employed for months and, if possible, in increasing doses. Among the remedies which are recommended as having a specific influence, we will also mention cinnamic acid, which is contained in Peruvian balsam. This has been employed extensively by Landerer in the treatment of various tuberculous diseases, and particularly in pulmonary tuberculosis. He gives it in the form of small subcutaneous injections of a five-per-cent. emulsion, made up of the sweet oil of almonds and the yolk of egg. The results obtained by Landerer, in the institute of Krähenbad, in the Black Forest, appear very favorable, but as yet they lack confirmation. The method has not yet been generally adopted.

At the end of 1890 great interest was excited, as is well known, by the statement of R. Koch that he had extracted from pure cultures of tubercle bacilli, by means of glycerine, a substance called tuberculine, by which he was able to heal tuberculous processes both in animals and men. This assertion aroused the thoughtless enthusiasm which is unfortunately so common with regard to therapeutic questions, and which indeed in this case was rendered excusable by the high authority of the discoverer. In fact, within a few weeks after the remedy was made known, numerous reports were published of cures due to tuberculine. But the longer the experiments were pursued the more evident it became that these extravagant opinions were not substantiated, and there set in a revulsion of sentiment which has led to many adverse criticisms, perhaps equally unjustifiable with the early praise. It is absolutely impossible to speak of tuberculine as an established remedy for tuberculosis. In many patients there does indeed appear a decided improvement under treatment, but these cases are such as were favorable any way, and therefore they may owe their improvement to the general symptomatic treatment and regimen which they have also enjoyed. At any rate, tuberculine has done no harm in such cases. Then, again, we have seen numerous cases of rather severe disease, in which it was not possible to notice any distinct influence of the tuberculine upon the general course of the illness. Perhaps the condition of the patient improved, or remained unchanged; or, again, it grew worse. Yet, whatever the change, it was such as might have occurred independently. Finally, there are, in the third place, cases which have been seen by the author as well as other observers, in which there was such a decided change for the worse shortly after the commencement of treatment, that one might really suppose that the remedy had exerted an actually harmful influence. We refer especially to cases in which the patient, previously without fever, exhibited after the injection a persistent elevation of temperature, with a more rapid advance of the local process in the lungs.

Consequently, we may say that, in all advanced cases, we can expect nothing from tuberculine. In incipient cases we may make trial of it, but we should be extremely careful in our dosage, so as to avoid any chance of doing injury. Koch's rule at first was that the physician should begin with injections of gramme 0.001, and gradually increase. In this way was discovered the interesting fact that the necessary dose for the production of the "reaction" (*vide supra*, page 252) became greater and greater. Many patients would finally bear the injection of one hundred milligrammes of tuberculine without any reaction at all. At first it was regarded as the object of treatment to reach this point of tolerance, but the practice is now abandoned. One should begin with very small doses, about one fourth to one half a milligramme, and increase so slowly and by such minute gradations as to avoid any marked reaction. By such a method we can almost entirely exclude any harmful effect, and it may not be impossible that some of the patients who have had a favorable result actually owe their improvement to the tuberculine, but it is extremely difficult to make sure of this. It would require the continuous observation of many patients for years, to enable one to form a reliable opinion with regard to this question.

It may be seen that at present we can only speak with great reserve as to the possible therapeutic value of tuberculine, but nevertheless, its discovery was an event of the greatest value and interest, regarded from a therapeutical standpoint. There is no doubt of the specific influence of this substance upon tuberculous processes. There is scarcely any sight more astonishing to the physician than the reaction of a patch of lupus upon the skin to a tiny dose of tuberculine introduced into the circulation! Taken into consideration with other bacteriological facts, this affords us a clear glance into a future when we shall succeed in making the wonderful influence of the material produced by the growth of bacteria available for the cure of infectious disease, and, in particular, of tuberculosis. Perhaps there are the same possibilities for tuberculosis as in the case of diphtheria, the treatment of which, by means of the antitoxic serum prepared by Behring, is apparently so very successful; but with regard to tuberculosis our methods are as yet incomplete, and a final opinion as to their value would be premature. Meanwhile, therefore, we physicians must continue to lay the greatest stress in the treatment of tuberculosis upon those methods which are termed in the widest sense hygienic and constitutional. It is undeniable that, by the correct and faithful employment of such methods, many very satisfactory successes may be obtained. For the present, we must be discreet, and seek to influence the disease in the old accustomed manner by those therapeutic measures which have come down to us from ordinary medical observation and experience.

The hygienic and constitutional method of treating consumption has for its object the greatest possible promotion of the natural powers of healing. We aim at this, in the first place, by avoiding as much as possible all influences which might cause a further extension of the disease, and by re-enforcing, so far as we can, all influences which increase the resisting powers of the individual, and the processes of spontaneous cure. The factors which are of most importance in this connection are, first, diet; second, rest; and third, the uninterrupted enjoyment of good air. To obtain these three therapeutic factors all at once requires the renunciation by the patient of his calling, and his usual mode of life. The treatment of tuberculosis, therefore, should begin with the demand that the patient, for as long a time as possible, should devote himself exclusively to the care of his health. The next point is the choice of the place in which the "cure" shall be carried on. In many cases the proper management of the patient may be pursued under the ordinary surroundings of home. Often, however, this is not the case because the last two, or even all three, of the essentials named can not be provided at home. It, therefore, is the duty of the physician in every individual case to determine how the desiderata are best to be obtained. According to the patient's means we consider respectively a residence in the country (if possible, in a picturesque and wooded region); a special health-resort; or, finally, a suitable sanitarium. From a therapeutic standpoint, there is no doubt that treatment in a specially adapted sanitarium is chiefly to be recommended. In this all the requirements for recovery can be best carried out, and the patient constantly remain under medical observation. In most cases it is only external considerations, for instance, pecuniary, which keep the patient from a sanitarium. It is a true philanthropy, therefore, which has of late led everywhere to efforts to render the benefits of institutional treatment accessible to the less wealthy classes. The requirements for proper treatment are also partially fulfilled in the so-called public health-resorts for pulmonary consumptives. These resorts, however, have the disadvantage that the patient is left far more to his own resources, and is consequently apt to be incautious, and hence jeopardize his recovery. A public health-resort would, therefore, be especially chosen for patients who have already been in an institution, and have learned what mode of life is proper for them; or for those who have already so far recovered that they may be allowed a certain degree of freedom.

With regard to the minutiæ in carrying out the above-enumerated essentials of treatment, we would add as follows: 1. *Diet.* This should be as nourishing and abundant as possible. Meat, milk, eggs, farinaceous foods, and butter are chiefly to be recommended, care being taken that the body should receive a sufficient amount of carbohydrates and fats, as well as an abundance of albumen. Many special "cures" for pulmonary consumption have a value, in so far as they lead to the ingestion of an abundant amount of easily assimilated nourishment. Such are cures with milk, koumyss, or kefir. It has even been proposed to introduce large amounts of such nourishment as milk and pulverized meat into the stomach by means of a stomach-tube, and thus accomplish "overfeeding." This method has not become very popular, although it may be indicated in some cases. It is very important to see that the patient has a diet that is not only abundant, but also palatable and varied. If pure milk is not readily taken, we may try the addition of coffee, tea, common salt, or brandy. With regard to the prescription of alcohol we recommend, unhesitatingly, moderate amounts of beer, and particularly such beer as is rich in extractive matter; and perhaps also extract of malt, and porter. Small amounts of good wine may contribute to the improvement of the appetite and the general condition. On the other hand, we think it useless and, in some circumstances, harmful, to order large amounts of the stronger alcoholic beverages, such as port wine and brandy, as prescribed in many sanitariums. Artificial foods, such as various preparations of meat, somatose, neutrose, and the like, should be used merely as makeshifts. The employment of cod-liver oil to the amount of two to four tablespoonfuls a day is sometimes not inappropriate, if it is well borne.

2. The two other factors of cure—*rest, and the continuous enjoyment of good air*—are best fulfilled by the fresh-air treatment, which is of late gaining in importance and popularity. The patients spend the greatest part of the day lying in the open air on comfortable reclining chairs. At the same time they avoid any unnecessary bodily exertion, any great demands upon respiration, and any irritation of the respiratory passages. Gain in bodily weight is promoted. The limitation of the diseased processes in the lungs is favored. Of course, in such matters also the individual should be considered, for moderate exercise in the open air is certainly for many patients not harmful, but rather beneficial. Often the methodical carrying out of the open-air treatment under suitable conditions demands the resources of a sanitarium, but we may find in a garden or on a veranda a sunny spot, screened from the wind, where the patient may lie comfortably and pass the entire day till near sunset in the open air. In case of necessity the patient must content himself with a place by an open window. The advantages of the climatic health-resorts (*vide infra*) consist principally in the fact that they render possible a continuous enjoyment of the open air, even during the colder part of the year. It has been maintained that climatic factors, particularly elevation, exercise a specific influence upon the healing of pulmonary tuberculosis; but this has not yet been proved. The best-known sanitariums for

pulmonary disease are found in Falkenstein in the Taunus, Görbersdorf, St. Blasien, Reiboldsgrün, Hohenhonnef, and Andreasberg in the Hartz Mountains, and Davos and Arosa in Switzerland. Of the public health-resorts which are particularly suitable for consumptives, we would name for summer use the acidulated, alkaline, and chloride-of-sodium waters of Ems, Obersalzbrun, and Reinerz; the chloride-of-sodium waters in Reichenhall, Salzungen, and Soden; the mud springs in Lippspringe, Inselbad, and Weissenburg in Switzerland. For summer climate, the following are to be recommended: Beatenberg, Heiden, Engelberg in Switzerland; Badenweiler, St. Blasien, Rippoldsau in the Black Forest, and many others. For the colder portion of the year we have, in their lofty situation, Davos, Arosa, and others. The more vigorous the constitution of the patient, the more proper it is to recommend him to go to a great elevation in the winter; while delicate subjects are usually better suited in the southern resorts.

Of course only the very distant health-resorts in Algiers, Egypt, Malta, and the much-praised Madeira, can furnish a certain guarantee of constant mild weather. The Sicilian health-resorts (Catania and Palermo), and also Ajaccio, afford favorable climatic conditions; while the health-resorts of the Riviera, Meran, Gries, Arco, Gardone, Lugano, Pallanza, and Montreux, are much more uncertain in this respect, and therefore are to be used merely as stopping-places by the way during the spring and autumn months.

We must also state that, in incipient cases, a residence by the sea, or a long sea-voyage, may sometimes be of great help. We have ourselves known several young physicians who have become ship surgeons on account of incipient phthisis, and who have returned from the voyage much stronger, and some of them apparently entirely well.

We can not go into a full description here of all the health-resorts mentioned. We can not omit, however, calling special attention to the fact that we should always ask ourselves, in choosing a health-resort, whether the expense and inconvenience thus imposed upon the patient can be balanced by the possible benefit. The earlier the stage of the disease and the better the general condition of the patient, the more will his physician be justified in urging him to make every sacrifice in order to regain his health.

We must particularly insist with the patient that no cure of tuberculosis can be accomplished by a single visit to any health-resort, but that recovery must be achieved by living continuously for years in accordance with all the demands of hygiene. On the other hand, it is wrong, both from a medical and from a humane standpoint, to send away consumptives in the last stages of their disease, to perish far from home and friends. Consumptives with fever should never be sent from home, unless they find refuge in a real sanitarium where they may enjoy the constant observation and treatment of a physician.

[Our own health-resorts for consumptives are too well known to demand extensive consideration here. The prime object is to secure for the patient a pure air, with such climatic conditions that he can pass the largest amount of time out of doors, at the same time that within doors his comfort is provided for, and a sufficiency of suitable and well-cooked foods is attainable. In Colorado, New Mexico, and California, large numbers of former consumptives are leading active lives. Florida, Aiken, Thomasville, Asheville, and some other southern resorts, are good winter asylums for many cases, but patients should not return to New England before June 1st. An out-door life in the Maine or Adirondack woods during the warmer months is highly to be recommended for early and otherwise suitable cases. Saranac, N. Y., affords every comfort during the colder months, combined with the very best medical attendance, and many people do well there. A relative disadvantage under which nearly all American health-resorts labor, as compared with those of Europe, consists in the greater difficulty in providing occupation and thus securing a mental attitude most favorable to recovery. In general, the northern sea-board is much less favorable than the interior, and early cases often do well during the winter removed from the dampness of the coast, with its alternations of freezing and thawing. A change of climate is a very important step, and should receive the most careful consideration of the physician—the circumstances of the patient, the stage and character of his disease, his tastes, etc., being carefully weighed before a decision is reached.]

Finally, the employment of hydrotherapy should be mentioned in the hygienic treatment of tuberculosis. Although this can not exert any specific influence on the disease, and therefore must not be prized too highly, yet it is often beneficial in the form of cold sponging, or brief cool douching and the like; and the stimulation of the skin has a favorable influence upon the general health. In severe febrile cases we may order sponging and rubbing in bed with cold water, brandy, vinegar, and the like. Also for the relief of symptoms (*vide infra*), such as pain, fever, and perspiration, we may often employ sponging, cold or warm compresses, and the wet pack.

The symptomatic treatment of phthisis is directed in the first place against the pulmonary symptoms. We use much the same remedies to help the cough as in chronic bronchitis. We try inhalations with a solution of common salt, or of the alkaline carbonates, or, if there is much secretion, with solutions of tannin and the balsams, such as turpentine, or balsam of Peru. When there is severe, spasmodic cough, inhalations with narcotic solutions sometimes give some relief, such as cherry-laurel water, opium, or bromide of potassium.

Morphine stands first among the drugs employed to check the cough. We should be cautious and sparing in its use at first, but it is an indispensable remedy in severe and hopeless cases. It relieves the irritation of coughing, the pain and the oppression in the chest, and at least gives the patient for a time the desired sleep. In chronic cases, with moderately severe symptoms, we may use for a long time the milder narcotics with advantage, particularly codeine (powders of gr. 0.5–1.0, gramme 0.03–0.05) or phosphate of codeine; also the extracts of hyoscyamus and of belladonna (extracti hyoscyami 1, aquæ lauro-cerasi 20, fifteen to twenty drops every two hours). [Heroine is sometimes useful in doses of $\frac{1}{12}$ grain.—V.] It is important that the patient should learn to suppress his inclination to cough, at least up to a certain point. Sips of cold water often quiet the cough, as may also a "cough drop" held in the mouth, or a pastille containing the salts found in Ems mineral water.

If the patient complains of difficulty in loosening the expectoration, we prescribe expectorants, the action of which often fails to meet our desires, but which can not be dispensed with in practice. The expectorants most frequently used are carbonate of ammonia, ipecacuanha, apomorphine, and senega. We very often combine expectorants with narcotics, as in Dover's powder.

If severe pain in the chest comes on, we often use local applications: mustard plasters, warm poultices and cold compresses, painting with iodine, or embrocations of chloroform. Narcotics, such as morphine, are indispensable in severe dyspnœa, which usually occurs only in the last stages of the disease or as a result of pneumothorax.

The treatment of hæmoptysis is important. As a slight admixture of blood in the expectoration often precedes a severe hæmoptysis, such an appearance always suggests caution. When there is any hæmoptysis, absolute rest in bed is necessary. We should avoid any careful examination of the lungs, especially any severe percussion. We should lay a flat and not too heavy ice-bag over the lung on the side from which we suspect the hæmorrhage; the cold is usually well borne, but sometimes it aggravates the cough, and must then be omitted. We would also recommend swallowing bits of cracked ice. Narcotics, such as morphine, are the most suitable internal remedies, since they aid the cessation of the hæmorrhage by suppressing the attacks of coughing. The more troublesome the cough the more necessary is it, therefore, to administer morphine in solution, by the mouth or even subcutaneously. Of remedies to check the blood we should name, first, extract of ergot or ergotine, which may be given by the mouth, or, still better, subcutaneously, in doses of 2 to 8 grains (grammes 0.1–0.5) several times a day. As to sphacelotoxine, and other special preparations of ergotine, there has been as yet no extended experience. We may also try the fluid extract of hydrastis, with equal parts of an elixir, giving thirty to forty drops of the mixture several times a day; also, acetate of lead and atropine. The influence of these remedies, particularly those last named, is, however, quite uncertain. The inhalation of a one- to two-per-cent. solution of perchloride of iron usually excites cough, and is therefore more harmful than beneficial. A very popular remedy among the laity, and one almost always at hand, is common salt, of which several teaspoonfuls may be given in water. When the hæmorrhage recurs frequently, it is also advisable to "tie off" the limbs—that is, to apply bandages rather firmly around the middle of the upper arms and the thighs. This causes venous congestion, and hinders the return of blood to the lungs. When pulmonary hæmorrhage has occurred, the diet should consist at first of nothing but cold milk, eggs, and similar food. Hot food, alcohol, and large amounts of meat are to be forbidden. It is a good thing to give acids, such as lemonade, or aromatic sulphuric acid, well diluted.

Even when the bleeding has ceased we must keep the patient several days in bed, and for a longer time be extremely cautious, since the hæmorrhage is apt to be repeated.

The hectic fever of consumption is remarkably little influenced by antipyretic drugs. It often is entirely useless, or even, on account of its evil effect upon the stomach, actually harmful to give to feverish consumptives, for long periods of time, large doses of such drugs as quinine, antipyrine, and antifebrine, particularly as the fever often of itself has deep morning remissions. The prescription of antipyretics is only justified when they make the patient feel better. On the other hand, it is very appropriate to give a cold sponging and rubbing to the whole body or chest, with water or alcohol, especially in the evening when the fever is high. The sponging is almost always well borne, and makes the patient feel brighter and easier. The cold pack may also be tried occasionally.

Cold sponging often diminishes the troublesome sweats in phthisis, but if this does not check them, we may often prescribe atropine to advantage, grain $\frac{1}{120}$ to $\frac{1}{60}$ (gramme 0.0005–0.001) at night, but its action does not usually last very long. Lately agaricine in $\frac{1}{12}$- to $\frac{1}{6}$-grain pills (gramme 0.005–0.01) has been recommended for the night-sweats in phthisis; also picrotoxine, of which grain $\frac{1}{8}$ to $\frac{1}{6}$ (gramme 0.008–0.01) is given in pill or solution at bed-time, and lately camphoric acid, grains 20–30 (grammes 1.5–2.0) in wafers. Dusting the body with a powder of five parts of salicylic acid to ninety-five of French chalk is also good. Sage tea is a favorite remedy for night-sweats—two or three cups of it cold at night—and so are milk and cognac.

If there is loss of appetite, small doses of quinine, compound tincture of cinchona, wine of cinchona, and other bitter remedies, such as tinctura amara (P. G.), are sometimes of service. It is also frequently a good thing to prescribe a little muriatic acid, five to ten drops of the dilute acid, with the meals. It is often very hard to check diarrhœa in phthisis. Opium, combined with tannin or acetate of lead, is most effective. This subject will be discussed more fully in the chapter on intestinal tuberculosis.

We often prescribe preparations of iron, combined sometimes with quinine or arsenic (*vide supra*), in the beginning of the disease to improve the general condition and the anæmia, but, as experience shows, iron is contra-indicated in patients who are feverish or who have a tendency to hæmoptysis.

The treatment of the diseases complicating phthisis is to be found in the appropriate chapters.

CHAPTER VII

ACUTE GENERAL MILIARY TUBERCULOSIS

Ætiology.—Acute miliary tuberculosis is a form of tuberculosis which we are justified in describing particularly because of its anatomical relations and its peculiar clinical history. The disease is characterized anatomically by the extremely abundant development of miliary tubercles in a comparatively short time in many organs of the body. We can not liken this process to anything but an overwhelming of the body with tubercle bacilli, which in some way reach the different organs simultaneously, and in them give rise to the eruption of tubercles. Buhl advanced the hypothesis a long time ago, that a cheesy focus could be found somewhere in the body in every case of acute miliary tuberculosis, and that the general infection of the body resulted from the absorption of these cheesy masses by the blood. Later investigations, however, have given us a much more definite explanation of the nature and manner of this general infection. Ponfick found, in some cases of acute miliary tuberculosis, an extensive tuberculosis of the thoracic duct with breaking down of the tubercular new growth. It is easy to see how, in this way, a large amount of tubercular material could be brought directly into the circulation, from the free communication of the lymph-duct with the subclavian vein, and thus be "disseminated" through the different organs in a short time. Still more frequently, however, the tuberculosis of the large venous trunks, discovered by Weigert, especially the pulmonary veins, seems to be the starting-point for an acute general miliary tuberculosis. Usually there are tubercular lymph-glands, or sometimes other foci of tubercular disease, which involve the wall of a neighboring vein, gradually break through it, and project into its lumen. If caseation and ulceration result in this spot, the infectious material is of course constantly washed off by the blood-current and carried away, and thus it reaches the other organs.

Since such a tubercular focus—e. g., a tubercular bronchial gland—may remain for a long time entirely without symptoms, we can understand how miliary tuberculosis may break out in an acute form in persons who previously seemed perfectly well. In other cases the patient has already suffered from some tubercular affection, and suddenly the conditions occur somewhere in the body which lead to the development of miliary tuberculosis. Thus we sometimes see it break out in a patient who has ordinary phthisis, but acute miliary tuberculosis is one of the rarities in advanced phthisis. If we find, at the autopsy of a case of acute general miliary tuberculosis, old phthisical changes in the lungs, which is by no means very common, they consist of old, partly cicatrized foci, pigment indurations, etc. We see miliary tuberculosis rather frequently as a sequel to pleuritic effusion. We have already previously called attention to the fact that in such cases the pleurisy itself is a tubercular disease. Miliary tuberculosis is also seen in persons with old tubercular affections of the bones and joints, such as coxitis and vertebral caries, with tubercular swellings of the lymph-glands, as in the neck and the axillæ, or with tuberculosis of the genito-urinary organs. In such cases, of course, the tubercular affection which is discovered during life, is not always the source of the general miliary tuberculosis, but the discovery of the existence of such an affection is of the greatest significance in diagnosis, as in this way our attention is strongly directed to the possibility of a general tubercular affection.

In some cases an outbreak of miliary tuberculosis has been seen to follow other acute diseases, such as typhoid, or measles.

Pathological Anatomy.—Except for the presence of an old tubercular affection in some organ, and except for the tuberculosis of a vein or of the thoracic duct, which are as a rule apparent, and which have been described above, the anatomical lesion in acute miliary tuberculosis consists in the dissemination of miliary tubercles through a large number of the organs of the body. The lungs, the liver, and the spleen are constantly affected; almost as constantly the kidneys, the thyroid gland, the marrow of the bones, the heart, and the choroid; less constantly, but still quite frequently, the serous membranes and the meninges. The miliary nodules may be found in large numbers in all the organs mentioned. They may in part be easily recognized by the naked eye, and in the lungs they may be very plainly perceived by the touch. In many organs, however, especially in the liver and often in the spleen, they are hard to recognize with the naked eye, but they are easily discovered by the microscope. In regard to the histological structure of miliary tubercles, and the discovery of tubercle bacilli in them, we must refer to what has been said in the chapter on pulmonary tuberculosis, but we must also mention that, in some of the more chronic cases, some of the nodules may grow to be large tubercular foci; from the size of a lentil to that of a pea. Less developed cases of miliary tuberculosis are also found, in which only a limited number of organs are attacked, and these with less severity.

Clinical History.—The clinical symptoms of miliary tuberculosis depend upon two factors, the first being the general infection of the body, and the second the local tubercular affection of certain organs. Although in many organs miliary tuberculosis is entirely without symptoms, as in the liver, the kidneys, the heart, and the marrow of the bones, in two organs—the lungs, and more especially the brain—it leads to the most marked local symptoms. The miliary tuberculosis of the choroid, discovered by Cohnheim and Manz, is also without symptoms, but it can be made out with the ophthalmoscope, and it is therefore of great diagnostic value.

Miliary tuberculosis affords quite different pictures, according to the predominance of one or the other of the groups of symptoms mentioned. We distinguish the four following forms:

1. *Miliary Tuberculosis, with Predominant Symptoms of General Infection: the so-called Typhoidal Form.*—This form may in part greatly resemble typhoid fever. The patient, who previously seemed quite well, or in whom some local manifestation of tuberculosis was suspected, falls ill with gradually increasing general symptoms, dullness, loss of appetite, headache, and fever. Since there is no local affection to be discovered to explain the symptoms, the disease at first may well be taken for typhoid. The general condition grows worse constantly, the fever is high and continually rises, and cerebral symptoms appear. In some cases an exanthematous eruption, like roseola, may increase the resemblance to typhoid. With careful observation, however, symptoms are almost always detected later in the disease which are, to a certain degree, characteristic of miliary tuberculosis, and are due to the existence of that disease either in the lungs or in the brain. The patient's complexion assumes a peculiar pallor, and with it a definite cyanotic hue. The respiration becomes remarkably deep, and there is dyspnœa; or signs of a tubercular meningitis arise, such as rigidity of the neck, loss of consciousness, disturbances in the innervation of the ocular muscles, etc., and death follows with these symptoms. These cases last from ten days to three weeks, reckoning from the beginning of the severe symptoms.

2. *Miliary Tuberculosis, with Predominant Pulmonary Symptoms.*—These cases, too, may begin quite suddenly, almost like an acute croupous pneumonia, or they may develop gradually with quite a long prodromal stage. From the onset the symptoms point especially to disease of the lungs or the pleura. The patient complains of a stitch in the side, cough, and dyspnœa. The expectoration may bear a decided resemblance to that of pneumonia. Such cases, especially if they begin abruptly, are at first often erroneously regarded as croupous pneumonia, particularly if we find fine râles almost like crepitant râles over certain portions of the lungs. But the expected crisis does not occur: the fever continues; the dyspnœa, general weakness, and anæmia of the patient increase; the physical signs of pulmonary disease (diffuse bronchitic râles) become more and more extensive. The patient's face is pale, cyanotic, and anxious. Death ensues with all the signs of impaired respiration. The course is usually somewhat more protracted than in the typhoidal form, lasting for three or four weeks and more.

3. *Miliary Tuberculosis, with Predominant Cerebral Symptoms, due to Tubercular Meningitis.*—Tuberculosis of the meninges does not belong among the regular lesions of general miliary tuberculosis. It develops in about half the cases, according to our estimation; but where it occurs it almost always gives the whole case the characteristic imprint of tubercular meningitis, by which the other symptoms are entirely concealed. The predominant symptoms are headache, fever, stupor increasing to deep coma, rigidity of the back and neck, and disturbances in the innervation of the ocular muscles. In such cases the tuberculous meningitis may alone be diagnosticated, and not the universal miliary tuberculosis. In fact, all the other signs of general miliary tuberculosis are not infrequently obscured by meningeal symptoms, and yet we have repeatedly observed, even in the deepest coma of the patient, a peculiarly deep and hurried respiration, which was the only noticeable sign referable to the miliary tuberculosis in the lungs.

The symptoms of tubercular meningitis in many cases predominate in this type of the disease from the onset, but in other cases they come on during the attack and form its final period. The duration of the disease varies accordingly.

4. *Miliary Tuberculosis with a Protracted Course and Indefinite Symptoms for a Long Time—Intermitting Form.*—Besides the forms already mentioned, cases occur which usually take quite a protracted course, lasting for eight or ten weeks, and having such indefinite symptoms that an absolute diagnosis is for a long time, or even throughout the disease, quite impossible. The patient complains of a number of general symptoms, such as headache and dullness, and also of thoracic symptoms, for which, however, we can find on examination no sufficient basis. There is almost always fever, usually not very high, and with a very irregular course; but we have seen a regular daily rise of temperature for a time in some cases, and attacks of fever with quite a severe chill, so that at first we thought of an irregular malarial intermittent fever—the intermitting form. Later on the symptoms gradually increase. The apparently inexplicable loss of strength, and the patient's anæmia and emaciation, are marked, and they are important in diagnosis. Finally, either severe pulmonary symptoms or the signs of tubercular meningitis set in, and to these the patient succumbs.

We must mention particularly that the four forms of miliary tuberculosis just described are only the types of the disease. In individual cases we often meet with variations and transitional forms between these types.

Single Symptoms.—1. *General Symptoms.*—In all cases of acute miliary tuberculosis the general condition of the patient is very serious. Most patients have a subjective feeling of severe illness, although they make little special complaint of it from the painless character of the disease. As the disease increases, there is often a marked feeling of anxiety and oppression besides the dyspnœa. There is, especially in the face, quite a peculiar pallor, characteristic of the disease, and associated with a marked cyanosis of the lips and cheeks.

2. *Fever.*—Acute miliary tuberculosis almost always runs its course with a more or less high fever, a course without fever having been observed in only a few instances. It often happens, in more protracted cases, that the temperature may be nearly normal for a time, or only slightly elevated. There is nothing characteristic or typical in the course of the fever. In the cases with typhoidal symptoms the fever is usually quite high, between 103° and 105° (39.5°–40.5° C.), so that the temperature-curve may be exactly like that of typhoid. In other cases the fever is irregular and is broken by many remissions, remitting or intermitting quite regularly for some time. Death ensues with a moderately high temperature or in collapse. In cases with tubercular meningitis there is also a marked rise of temperature at the close, up to 108° (42° C.) and over.

3. *Respiratory Apparatus.*—It goes without saying that physical examination of the lungs may give no definite results. Almost all positive evidence is often wanting, and the contrast between the labored breathing and dyspnœa and the insignificance of the physical signs in the lungs is an important feature in diagnosis. Auscultation, as a rule, gives the signs of an intense bronchial catarrh; we hear rhonchi or numerous fine and medium moist râles all over both lungs. The respiratory murmur itself is usually higher in pitch than normal, and in many cases it is obscure, rough, or harsh. In one of our cases there was heard, over a circumscribed area of the lung, a wholly peculiar, sharp, "lapping" sound on inspiration, which we have never heard under any other circumstances. Jürgensen describes a soft friction sound, due to miliary tuberculosis of the pleura. Percussion usually gives no objective changes. At times the resonance is rather tympanitic, or slightly dull in some places.

In some cases circumscribed pneumonic infiltration has been observed in the lungs in acute miliary tuberculosis, which, as we have said, may give rise to a confusion between miliary tuberculosis and croupous pneumonia, from the presence of marked dullness, crepitant râles, and bronchial respiration.

We must mention, finally, that in some of the cases physical examination of the lungs shows old changes in them, a phthisical affection of the apex, a former pleurisy, and the like. Positive evidence of such old tubercular affections may be of great diagnostic value in doubtful cases.

Dyspnœa has been repeatedly mentioned among the pulmonary symptoms. The respiration is usually very much accelerated, especially during the more advanced stage of the disease, so that we see in adults forty, sixty, and even seventy respirations a minute. The respiration is also very deep, and is sometimes noisy. As a rule there is cough, but it is usually troublesome only in the cases with severe bronchitis. It is often very slight. The expectoration is usually scanty, and it is not characteristic. Special mention must be made of the fact that tubercle bacilli are absent in it, unless old ulcerated tubercular foci are present at the same time in the lungs.

4. *Circulatory Apparatus.*—The pulse is frequent, about 100 to 120 a minute, often weak and small, and sometimes irregular, especially if tubercular meningitis co-exists. The miliary tubercles, which post mortem are almost always to be found in the heart, especially in the endocardium, cause no symptoms. In uncomplicated, acute, miliary tuberculosis there is little if any increase in the number of white corpuscles in the blood. The presence of tubercle bacilli in the blood will be mentioned below.

5. *Digestive Apparatus.*—Vomiting is frequent at the onset of the disease. The bowels are usually constipated, but in many cases there is a moderate diarrhœa. The loss of appetite, the thirst, and the dry tongue are due to the general

disease and the fever. The spleen is usually somewhat, but not very much, enlarged.

6. *Nervous System.*—In many cases in which the pulmonary symptoms predominate the intellect remains quite clear until the last, but in other cases cerebral symptoms, such as headache, dizziness, stupor, and delirium, come on quite early, and are a part of the general infection. As has already been said, the nervous symptoms in the cases combined with tubercular meningitis become quite prominent, but in individual instances it may be hard to decide whether they are due to such a complication, or are merely severe general symptoms.

7. *Eyes.*—The ophthalmoscopic examination of the retina is of special diagnostic importance, since the diagnosis may be made absolutely certain by finding miliary tubercles in the choroid. A negative result, however, is never decisive against the diagnosis, since the tubercles are sometimes absent, or at least are very few in number. Their discovery is almost always difficult, and demands much practice in the method of examination. In cases with tubercular meningitis we sometimes find an optic neuritis.

Diagnosis.—The diagnosis of acute general miliary tuberculosis is ordinarily and justly considered very difficult. It quite often happens that at the autopsy a miliary tuberculosis is found which was not even suspected during life. It must be confessed that frequently, in such cases, we might very well have thought of acute tuberculosis. If, therefore, the possibility of acute miliary tuberculosis is brought to our attention during the patient's life, we can occasionally make an absolute diagnosis.

The severe general condition, usually associated with fever, is most important, and for this no local cause can be found. Then come the pulmonary symptoms, especially the peculiar dyspnœa, for which there is also no adequate corresponding physical change to be discovered. It gives decided support to our suspicion if we can make out a distinct predisposition to tuberculosis, either hereditary or constitutional, or the history of a previous tubercular affection, especially pleurisy, and also chronic affections of the bones. The peculiar cyanotic pallor of the patient is very characteristic.

On these factors rests the differential diagnosis between the "typhoidal" form of miliary tuberculosis and typhoid fever. Marked roseola and considerable enlargement of the spleen are distinct arguments for typhoid, although they sometimes occur in miliary tuberculosis, and so are the intestinal symptoms of typhoid, such as meteorism, the characteristic, loose yellow stools, and possibly intestinal hæmorrhage; but we must not forget that both the roseola and the intestinal symptoms may be absent in typhoid. The course of the fever must always be considered in the differential diagnosis. It is much more frequently irregular and atypical in tuberculosis than in typhoid. Of course, the temperature-curve is not an absolutely decisive factor. The blood does not afford any indubitable signs of distinction between the two, since in neither disease is there a decided leucocytosis. If the number of leucocytes is conspicuously small, 5,000 or less, typhoid is strongly suggested. The Widal serum-reaction with typhoid bacilli (see page 22) is very important, and, when it turns out positively, decisive. On the other hand, of course, the absolute demonstration of miliary tuberculosis in the choroid is unequivocal evidence in favor of miliary tuberculosis.

In many cases the onset of meningeal symptoms may aid the diagnosis. Of course, if the patient is not seen until the last stages of meningitis, especially when there is an incomplete history, the diagnosis is next to impossible. In this case, if the fluid obtained by lumbar puncture is found to contain tubercle bacilli, the diagnosis may be settled (compare the chapter on tubercular meningitis).

Acute tuberculosis is often confounded with severe bronchitis, especially in old persons who are considered emphysematous. The very severe general condition, the pallor, the rapid loss of strength, and the fever, are the only things here which call our attention to acute tuberculosis, and render the diagnosis possible. We have already indicated the possibility of confusion at the onset between miliary tuberculosis and croupous pneumonia.

Of decisive diagnostic value in all cases is the demonstration of tubercle bacilli in the blood, which is indeed a difficult matter, but it has already been accomplished more than once (Weichselbaum and others).

Prognosis.—The cases described in literature as "cured miliary tuberculosis" are so uncertain in their diagnosis that they can not be regarded as convincing. We must therefore consider the prognosis as absolutely fatal. The differences in the course of the disease have been already mentioned.

Treatment.—Although drugs are absolutely powerless, still the case in hand must always receive treatment, especially if the diagnosis can not be made with absolute certainty. Our prescriptions are purely symptomatic. The cases with a typhoidal course are to be treated just like typhoid, with baths, stimulants, etc. Tepid baths, and also local applications to the chest, expectorants, and narcotics, are indicated when the thoracic symptoms predominate. If meningeal symptoms set in, we may try ice, perhaps local blood-letting, iodoform salve, or mercurial ointment externally, and iodide of potassium internally.

CHAPTER VIII

GANGRENE OF THE LUNGS

Ætiology.—The sole cause of pulmonary gangrene—that is, the death and putrid decomposition of the lung-tissue—is the entrance of the bacteria of putrefaction into the lungs. The opportunity for inhaling them is certainly very great, but the normal organism apparently possesses the property of nullifying them and making them powerless. Under certain conditions, however, they take root in the lung and cause the death of the pulmonary parenchyma, which then, as a result of the presence of these specific bacteria of putrefaction, succumbs to that peculiar form of putrid decomposition known as "moist gangrene."

The factor which most frequently gives rise to the development of pulmonary gangrene is the entrance of organic foreign substances, especially bits of food, into the lungs. The bacteria of putrefaction either enter the lungs with the foreign substance, or they settle there later and set up a putrid decomposition, first in that portion of the lungs, and then in the neighboring lung-tissue. The entrance of organic foreign substances into the lungs occurs in different ways. It often happens from swallowing the wrong way, or from an accidental inhalation. In this way pulmonary gangrene may arise in previously healthy people, but it occurs especially in patients who are very low, very stupid, and soporose, and also in the insane, in patients who can not swallow or cough well, and in patients with paralysis of deglutition, as in bulbar paralysis. Bits of food may also reach the lungs from eructations and vomiting. Thus are explained the cases of pulmonary gangrene which occur in patients with cancer of the stomach, and, still more frequently, with cancer of the œsophagus. Putrid organic material may also reach the lungs from ulcerative and ichorous processes in the mouth, the pharynx, and the larynx. In cancer of the tongue, the pharynx, and the larynx, in other ulcerative processes, and in injuries or wounds from operations in the mouth and pharynx that have become septic, pulmonary gangrene may develop quite readily.

Finally, septic foci in the vicinity may extend to the lungs or perforate into a bronchus. In this way pulmonary gangrene may arise from the perforation through the pleura into the lungs of an ulcerated cancer of the stomach or a gastric ulcer, or in rare cases from vertebral caries, or from sanious lymph-glands.

In some cases the cause of the pulmonary gangrene can not be made out, since the entrance of a foreign substance has perhaps been wholly unnoticed, as may happen in children, or during sleep. We had a grown-up young woman under observation for a long time with pulmonary gangrene, and one day she coughed up several fragments of chicken-bones, but she could give no account of how they entered the lungs.

Experience teaches us that pulmonary gangrene is more apt to develop in persons with impaired nutrition, in old, marantic individuals and drunkards, than in those who are healthy. The tendency of patients with diabetes mellitus to pulmonary gangrene is remarkable.

Pulmonary gangrene often develops secondarily to some other pulmonary affection. We have already spoken of the relations between it and fœtid bronchitis. Fœtid bronchitis, on the one hand, often leads to pulmonary gangrene through an extension of the process to the alveoli; and, on the other hand, when there is a gangrenous focus in the lungs, the bronchi are often infected to a wide extent by the putrid secretion coming from it, and then there arises fœtid bronchitis. Both diseases often run into each other without any sharp boundary; but gangrene may develop secondarily in other affections of the lungs. A new infection with putrid material, however, is always requisite, and the affection of the lungs that already exists furnishes merely a favorable soil. This is the only explanation of the process when croupous pneumonia "runs into gangrene," or when gangrene develops in catarrhal pneumonia, in bronchiectasis, or in phthisis.

Although the agents of putrefaction enter the lungs through the bronchi in most of the modes of origin of pulmonary gangrene that have been mentioned, they may also be transported into the lungs by the blood-current. We call these forms embolic gangrene. We find such gangrenous nodules in the lungs in connection with extensive gangrenous bed-sores, puerperal processes, suppurative caries of the bones, etc. In these cases the putrid material enters a vein from the seat of the primary process and is brought to the lungs, and here we find, as a result of the putrid character of the embolus, not a simple infarction, but an embolic gangrene.

Pathological Anatomy.—We more frequently find pulmonary gangrene in the lower lobes than in the upper, corresponding to its mode of origin. Either both lungs are affected or only one, and the right somewhat more frequently than the left. We distinguish a diffuse and a circumscribed form, according to the extent of the gangrene. Embolic gangrene belongs to the latter form, and its nodules, by preference, lie near the pleural surface.

We can easily recognize the anatomical changes in gangrene. The lung-tissue is changed to a discolored, dirty, greenish-gray mass, which gradually and progressively becomes dissolved, forming a most foul-smelling ichor. We find, left in it, necrotic fragments of tissue and vessels. Gangrene cavities, with irregular, ragged walls, are formed from the partial expectoration of the softened gangrenous nodule. The lung-tissue in the vicinity of the gangrenous spot is to a greater or less extent inflamed, partly in the form of catarrhal pneumonia, partly in the form of circumscribed croupous pneumonia. The inflamed parts in the vicinity are gradually involved in the gangrene, so long as the process extends, but finally a suppurating line of demarkation may be formed about the gangrene, the whole gangrenous fragment is in a measure sequestrated, encapsuled, and gradually expelled, and so healing becomes possible. We have already stated that fœtid bronchitis may arise from a gangrenous nodule.

Whenever a gangrenous nodule reaches the pleura, a purulent and usually a sanious pleurisy follows from direct infection. Pneumothorax may arise from perforation of a gangrenous cavity.

Clinical History.—The symptoms of gangrene depend for the most part upon the local affection in the lung. The condition of the expectoration is characteristic, and it alone may decide the diagnosis.

In many ways the expectoration greatly resembles that of fœtid bronchitis, and indeed a great part of it does not come directly from the gangrenous nodule, but is the secretion of the diseased bronchi. The penetrating stench of the sputum, a most repulsive, putrid odor, is very striking. The patient's breath and cough also have this stench, which infects the whole vicinity. The amount of the sputum is usually large; it may reach ten or twenty ounces (200–500 cubic centimetres) in twenty-four hours. If the sputum is collected in a glass it forms three layers, like the sputum of fœtid bronchitis—an upper layer, muco-purulent, greasy, consisting in part of nummular sputa, and covered with much froth; a middle serous layer, in which some firm masses from the upper layer float; and a lower layer, almost wholly of pus, but greasy and greenish-yellow, which usually contains many large and small plugs and shreds of tissue. We find in these plugs, on microscopic examination, beautifully twisted needles of the fat acids (see Fig. 24, page 168) imbedded in countless bacteria, fat-drops, and detritus, and often collected in large bundles; but besides these we find in the sputum the constituents of the parenchyma of the lungs, and this alone is the decisive factor in distinguishing between pulmonary gangrene and simple fœtid bronchitis.

Traube's statement that in pulmonary gangrene the expectoration contains few, if any, elastic fibers, because the elastic tissue itself is destroyed by gangrene, is not correct; or, at any rate, it is too sweeping. We have almost invariably found in the expectoration an abundance of elastic tissue, as well as other fragments of the parenchyma, pigment granules, and the like. Yet there is a probability, no doubt, that in pulmonary gangrene the elastic tissue is for the most part destroyed. Filehne succeeded in extracting from the sputum of pulmonary gangrene, by means of glycerine, a ferment which in alkaline solution completely dissolved elastic tissue in a few days. The sputum always contains in enormous numbers bacteria of various kinds, both cocci and bacilli. Which of these are the special cause of the gangrene has not yet been definitely settled. The chemical examination of the sputum shows the presence of those substances which may always be found in the putrefaction of organic matter—tyrosine, leucine, ammonia, sulphuretted hydrogen, butyric acid, valerianic acid, caprylic acid, etc. The fresh sputum usually has an alkaline reaction, but on standing it becomes acid.

Many cases of gangrene lead to erosion of the vessels and severe hæmoptysis. Slight admixtures of blood in the sputum are not infrequent.

The other symptoms on the part of the lungs are not especially characteristic of gangrene. Most patients complain of cough, pain in the side, and more or less severe dyspnœa. Physical examination, as a rule, permits us to make out the seat of the nodule, but not always, since the physical signs, of course, depend upon the situation and extent of the gangrene. Small nodules, situated centrally, often give no objective evidence of their presence. Every extensive infiltration, however, must cause dullness on percussion. Over the area of dullness we hear bronchial respiration, and usually quite numerous moist râles. If a gangrenous cavity is formed, the physical examination may show plain symptoms of a cavity—tympanitic resonance on percussion, amphoric respiration, coarse moist râles, etc.

The physical signs are sometimes due to the accompanying pleurisy; the dullness is more complete, the respiratory murmur and the vocal fremitus are diminished, and the adjacent organs are displaced by the abundant effusion; but an absolute diagnosis of an accompanying pleurisy is often to be made only by an exploratory puncture. We have already spoken of the occasional development of pneumothorax.

In many cases there is fever, of quite an irregular character and of very varying intensity. In the cases in which the gangrenous nodule is sequestrated, and the secretion can be freely emptied through the bronchi, so that there is no absorption of septic material into the blood, fever may be entirely absent.

We often see gastric and intestinal symptoms in pulmonary gangrene, the disturbance being without doubt due to swallowing some of the fœtid sputum. Many patients complain of loss of appetite, and sometimes of vomiting or diarrhœa. In severe acute cases there is sometimes a well-marked typhoidal state, with such symptoms as stupor, delirium, and great cardiac weakness. This condition is probably caused by the absorption of septic material into the blood. Rheumatic pains in the muscles and joints are seen in this disease, just as in fœtid bronchitis. Finally, it should be remarked that the appearance of secondary abscesses in the brain (see page 1107) has been repeatedly observed in pulmonary gangrene. This fact must be borne in mind when, in the course of pulmonary gangrene, marked brain symptoms are developed, especially if we have not only such general symptoms as coma, but also such local symptoms as hemplegia and other forms of paralysis, and convulsions.

The general course of the disease shows very great variations. In all cases in which the pulmonary gangrene is secondary to some other affection, the course and the general type of the disease depend very largely upon the primary attack, but the cases of idiopathic gangrene also present great variations. The onset is either quite gradual and slow, or quite acute, and associated with fever and thoracic symptoms. The stinking expectoration and the bad odor from the patient's mouth first direct the attention to the existence of putrid processes in the lungs. The disease is usually very chronic, lasting for months or even years. Many remissions and intermissions occur. With proper care and treatment we may see a decided improvement, and often apparently a complete cessation of the disease. The bad odor ceases, the expectoration diminishes or disappears entirely, and the patient's strength and nutrition become almost normal; but relapses may occur after long intervals. When the affection is of slight extent we may even see a complete recovery.

Pulmonary gangrene always takes a worse course in previously weak and marantic persons, and an unfavorable termination may follow in a comparatively short time. Death ensues either from a general loss of strength, as a result of the disease, or from complications, such as pulmonary hæmorrhage, ichorous pleurisy, pneumothorax, or abscess of the brain. Rupture of an ichorous empyema outwardly, or into the peritoneum, or into other cavities, is rare.

Special mention must be made of the fact that the symptoms of pulmonary gangrene are not always so very pronounced. In persons who are weak and run down we often see pulmonary gangrene at the autopsy, although during life there have been no marked symptoms, not even offensive sputum or fœtor from the mouth.

Diagnosis.—The diagnosis can not be made with certainty unless the characteristic sputum is present. We can decide whether the sputum comes from a fœtid bronchitis or from the fœtid contents of a bronchiectasis, or from actual gangrene, only by finding under the microscope the remains of lung-tissue in the expectoration. Physical examination in gangrene, at least in part of the cases, also gives the signs of infiltration or of cavity-formation in the lungs.

Prognosis.—The prognosis depends first upon the nature of the underlying disease, and then upon the extent of the affection, the strength of the patient, and the possibility of sufficient care and proper treatment. If the process in the lung becomes sequestrated, marked improvement may follow, even in the severest conditions; but we must always remember that a relapse is possible. Complete recovery from pulmonary gangrene is certainly extremely rare, if it ever occurs. We have already mentioned the dangers which may cause a fatal termination in pulmonary gangrene.

Treatment.—Prophylaxis plays an important part in those cases in which there is danger of the entrance of bits of food into the air-passages from defective deglutition. We must think of the possibility of this with all patients who show great stupor, and also with patients who have pharyngeal paralysis, in order to watch them while taking food, and eventually to try artificial feeding with the œsophageal tube.

The treatment of already existing pulmonary gangrene has, as its chief aim, to check the putrid processes of decomposition in the lungs. Unfortunately, the remedies at our command are not in all cases sufficient for this. The different disinfecting inhalations are the most effective. They are used in the same way as in fœtid bronchitis (*vide supra*). Turpentine deserves the most confidence, and it may also be given internally with good results. According to Lépine, however, terpine acts even better than turpentine. We may also call attention to inhalations with carbolic acid, Curschmann's carbolic mask, inhalations with salicylic and boracic acids (4 parts of salicylic and 20 of boracic acid to 1,200 of distilled water), bromine (bromine and bromide of potassium, 2 parts of each to 1,000 of water), and similar substances.

Besides oil of turpentine, other internal remedies are recommended: half a grain to a grain (gramme 0.03–0.06) of acetate of lead every two hours, creasote, carbolic acid, etc. Their action is uncertain. Of late, myrtol has been greatly extolled. It is given in capsules containing gr. ijss. (grm. 0.15), of which two or three are to be taken every two hours.

The general treatment of the patient is very important—he should have good food, and live in as good air as possible. We must treat the pain in the chest and the cough symptomatically, local applications and morphine being most useful. The fever seldom gives occasion for direct interference. We may try to relieve the gastric and intestinal symptoms by giving antiseptics internally, especially by small doses of muriatic acid, salicylic acid, or creasote, as well as by the ordinary remedies, such as bitters and opium.

If a secondary ichorous pleurisy develops, with or without pneumothorax, removal of the fluid by operation is necessary, if the patient has sufficient strength to bear it.

In some few cases trial has been made of opening up the foci of gangrene by surgical means. As yet, it must be confessed, the results have not been very encouraging.

CHAPTER IX

DISEASES FROM THE INHALATION OF DUST

(*Pneumonoconiosis*)

Although there are a number of important contrivances in the respiratory apparatus to prevent the entrance of foreign substances into the lungs, still, if a person remains in a dusty atmosphere, so many particles of dust may be inhaled that they are not without effect on the lung-tissue. The diseases arising from the inhalation of dust are usually purely professional diseases, which occur especially in workmen whose occupation involves the continual inhalation of some kind of dust. In earlier chapters of this book, particularly while considering chronic bronchitis, we have already emphasized the harmful influence of the inhalation of dust. We have seen how the habitual respiration of organic dust in particular (from grain, wool, wood, and tobacco) is very apt to lead to severe forms of bronchitis and bronchiolitis. We must now draw attention to certain specific diseases similarly caused.

We must first mention, however, a condition of the lungs which can scarcely be regarded as pathological, although it has its origin in the constant inhalation of dust, especially of coal-dust—the ordinary black pigmentation of the lungs. There can now no longer be any doubt, although there was once a long dispute about it, that the black pigment in the lung comes, in large part at least, from the inhalation of carbon. The particles of carbon pass into the lungs themselves, and thence into the bronchial glands by means of the lymphatics. A certain part of the coal-dust inhaled is removed with the expectoration, and it may easily be found in it microscopically, and often by the naked eye, as we see it in the well-known black expectoration which we often have in the morning, if we have spent the previous evening in a room filled with smoke. In Germany, Traube was the first to discover the particles of carbon in the expectoration of a charcoal-burner. In the man's lungs, after death, the vegetable origin of these particles could be recognized, and Traube gave the correct explanation of them. In workmen who inhale large amounts of charcoal-dust, anthracite coal-dust, soot, or graphite, the "normal" pigmentation of the lung passes into a pathological condition, *anthracosis pulmonum.* With this is usually associated an extensive chronic bronchitis. In the expectoration of such patients are found many cells, filled with black particles of coal, even long after they have left the dusty atmosphere. The pigmented cells are leucocytes, or perhaps some of them epithelial cells (see Fig. 35).

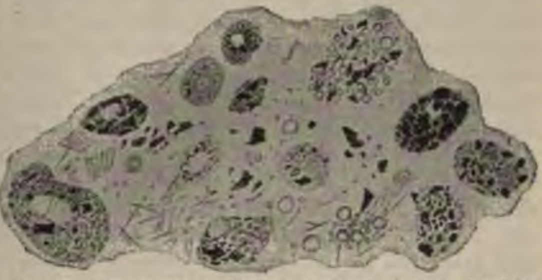


FIG. 35.—Expectoration of a man who worked on graphite. Numerous cells filled with particles of carbon. (Erlangen medical clinique.)

Zenker first discussed comprehensively the fact of the entrance of the different sorts of dust into the lungs and the consequent results. Besides the anthracosis already mentioned, the pulmonary disease from inhaling the dust of flint and other stones is of especial importance, the so-called stone-cutter's lung—*chalicosis pulmonum*—and also that from inhaling metallic dust, especially oxide of iron—*siderosis pulmonum.* The "stone-lungs" have been observed in workmen in the stamping-rooms of glass-factories, in mill-stone cutters, stone-polishers, stone-hammerers, plasterers, workers in porcelain, masons, slate-quarrymen, potters, etc. "Metal-dust lungs" occur in file-cutters, iron-workers, mirror-polishers, and especially in grinders, who inhale a mixture of stone- and iron-dust. The first case of a "red iron lung" was observed by Zenker, in a girl who had inhaled a thick dust of iron for ten or twelve hours a day. Her work was to color blotting-paper with a powder of red oxide of iron. During the inhalation of all these and similar kinds of dust, a portion of the inspired particles of dust are taken up by the leucocytes, and perhaps also by the epithelial cells, finally reaching the lymph-channels of the lungs. Some of the particles of dust remain in the interstitial connective tissue of the lungs. Others find their way to the bronchial and retro-bronchial lymph-glands.

If the inhalation of dust is long continued, it causes not only this abnormal pigmentation of the lungs, but also macroscopic anatomical lesions. These consist, partly, in a more or less severe and extensive chronic bronchitis, and partly in an interstitial inflammation which is due to the chronic irritation of the foreign matter (e. g., flinty particles), and leads to the formation of connective tissue. The lungs are studded with nodules, which feel hard to the touch, and grate on section with a knife. All of these nodules consist of firm connective tissue, in which the particles of stone or iron are encapsuled. By the union of single nodules we may get more extensive induration and cicatricial formation. Chemical examination of such lungs gives, as might be supposed, a large amount of silicic acid, iron, etc.

In most of the cases which come to autopsy we find further changes in the lungs, which are not the immediate result of the inhalation of dust, but consist of sequelæ and complications. Chronic diffuse bronchitis in the worker in dust, like any other chronic bronchitis, may give rise to pulmonary emphysema, and later to cardiac hypertrophy, etc. We very often find in the lungs co-existing and pronounced tubercular changes. It need hardly be emphasized that these changes are not the direct result of the inhalation of dust, but that the changes in the lungs set up by such an inhalation furnish merely a favorable soil for infection with tuberculosis. In most cases, the " dust-lungs " acquire a marked clinical significance chiefly from the sequelæ mentioned—namely, emphysema and tuberculosis. The circumscribed nodules of interstitial pneumonia have no very marked symptoms following them. In all cases in which there is a fatal termination, with pulmonary symptoms, we should regard the immediate action of the dust as much less the cause of death than are the secondary diseases.

The actual points to be considered in judging of the clinical symptoms of the diseases from inhaling dust are contained in what has been said. The symptoms are those of chronic bronchitis, pulmonary emphysema, or chronic phthisis, and attention to the injurious influences associated with the patient's calling is the only possible way of making a diagnosis, but in individual cases it may always be a matter of doubt how far other accidental causes of disease may come into play.

The prognosis depends, in the first place, upon whether the patient can be removed from the action of these injurious influences or not, but we must also mention the fact, which has been often observed, that many individuals get somewhat used to the dust. After they have once recovered from the initial bronchitis, such persons can live in an atmosphere of dust for a long time without any noticeable injury.

The prophylaxis of diseases from inhaling dust forms an extended chapter in the hygiene of occupations, which we can not dwell upon here. The workman must be taught the danger to which he is exposed, and the danger itself must be diminished as much as possible by sufficient ventilation of the work-rooms, by cleanliness, and, under some circumstances, by a change in the technicalities of the business.

We need not give any special directions regarding the treatment of diseases from inhaling dust. It is founded on the same principles as the treatment of chronic bronchitis, emphysema, and chronic pulmonary tuberculosis.

CHAPTER X

EMBOLIC PROCESSES IN THE LUNGS

(Hæmorrhagic Infarction of the Lungs)

Ætiology.—The sources from which the material for an embolic plugging of branches of the pulmonary artery comes lie either in the right side of the heart or in the veins of the body. Pathological anatomy teaches us how often thrombi are formed in the veins, especially in the veins of the lower extremities and in the pelvic veins, and in the right side of the heart, in the recesses between the trabeculæ, in the auricles, on the valves and the chordæ tendinæ, and at the apex of the ventricle. The particles, torn loose from thrombi so situated, are carried on by the blood-current, reach the lungs, plug a larger or a smaller branch of the pulmonary artery, according to the size of the particles, and thus cause further changes in the lung-tissue. Since the branches of the pulmonary artery are " terminal arteries," and since thus the vascular territory belonging to each branch can not be supplied, or can be supplied only to a small amount, with blood by collateral circulation from other vessels, the closure of a branch of the artery shuts the territory supplied by it out of the circulation. The pressure, in the part of the vessel lying peripherally to the point of plugging, becomes almost nil, and, as a result, there is a collateral or backward current into the region shut off, flowing from the capillaries in the vicinity, and probably even from the veins belonging to it. Yet the blood which flows in is under so slight a pressure that it does not flow through, but stagnates in the affected area. The walls of the capillaries and veins, in which the normal blood-current has ceased, lose their natural consistency as a result of this. The vascular walls become abnormally pervious. The fluid of the blood, the white blood-corpuscles, and also very many red blood-corpuscles, penetrate through the walls of the vessel into the surrounding tissue, and change it into the so-called hæmorrhagic infarction.

Every embolic closure of a branch of the pulmonary artery does not necessarily result in the formation of an infarction. Upon the sudden plugging of a main trunk, or of several large branches of the pulmonary artery, death may ensue at once, so that naturally all further changes in the lung-tissue cease. We also find quite frequently, especially in central portions of the lung, embolism of single branches of the pulmonary artery without the formation of an infarction. In such cases there must necessarily be a little circulation in the vascular territory which has been shut off, either by anastomoses between the territory of the pulmonary artery and that of the bronchial or mediastinal artery, or by the neighboring capillaries, whose arteries of supply remain open.

The changes thus far described are the result of a purely mechanical closure of a pulmonary artery. We have noticed this especially when simple fibrinous plugs have given the occasion for the embolic process. Pulmonary infarctions are most frequent in chronic heart disease, in all forms of primary and secondary dilatation of the heart, but especially in disease of the mitral orifice, most often mitral stenosis. Thrombus formation is frequent in the dilated right side of the heart, and furnishes material for pulmonary emboli; but these emboli are seen in all other possible conditions of disease, in which thrombosis in the right side of the heart or in the veins may occur.

The changes in the lungs assume quite a different appearance if the embolic material is not simple fibrine, but if it contains at the same time some specific infectious matter. If emboli reach the lungs from an acute malignant endocarditis in the right side of the heart, or, as is most frequently the case, from a purulent (septic) phlebitis anywhere in the body, giving rise to a puriform, liquefying thrombus, the specific factors in inflammation—that is, bacteria—get into the lungs. Thus arise embolic abscesses and embolic gangrenous nodules in the lungs. We have already spoken of the latter, and the former are among the most constant lesions in every typical case of pyæmia.

The fundamental facts as to the occurrence and significance of embolic processes in general, and those located in the lungs in particular, were discovered by Virchow. For a fuller understanding of the results of embolic closure of the vessels we must thank chiefly Cohnheim.

Pathological Anatomy.—Hæmorrhagic infarctions may involve one or more lobules, or almost a whole lobe of the lung, according to the situation of the embolus. Most infarctions are situated at the periphery of the lung, and have approximately a conical shape, corresponding to the extent of the region of the vessel. The base of the cone lies against the surface of the pleura. It generally projects a little above that surface, and its dark color can usually be plainly recognized through it. The pleura itself is the seat of a fibrinous inflammation at the point to which the infarction reaches, and sometimes for a large space around it. The conical shape of the infarction is plainly recognized on section. The lung-tissue is changed to a firm, fragile, uniformly black-red tissue, devoid of air. The embolus can usually be readily found in the branch of the pulmonary artery leading to the infarction. Under the microscope we see a diffuse infiltration of tissue, with red blood-corpuscles in the infarcted portion. The alveoli and finer bronchi are also filled with coagulated blood. Under favorable circumstances, in cases of longer standing, the blood may be reabsorbed in part; so that the lung again contains air, but it remains much pigmented in that place, and more or less indurated from the development of interstitial connective tissue. In exceptional cases there is complete destruction of the pulmonary tissue where the infarction is situated. This broken-down material is either discharged or absorbed; then cicatrization with contraction takes place.

Hæmorrhagic infarctions are usually situated in the lower lobes, and more frequently on the right side than on the left.

The smaller embolic abscesses are sometimes very numerous, and are disseminated over the whole lung. In larger abscesses the conical shape may often be plainly recognized. When an abscess extends to the pleura, a purulent pleurisy arises from direct infection. Combinations and transitional forms between the ordinary hæmorrhagic infarction and embolic abscesses are occasionally found in the lungs.

Symptoms.—We often find at the autopsy embolism of single branches of the pulmonary artery, with or without infarction, which has caused no symptoms at all during life.

Embolism of the main trunk, or of a large branch of the pulmonary artery, may cause sudden death, as has been repeatedly observed in patients with heart disease, or with venous thrombosis. If death be not immediate, sudden severe dyspnœa and oppression arise. The diagnosis may at least be suspected in such a case if we know of a possible source for an embolus. In some instances, when an embolus is situated in a large branch of the pulmonary artery, but has not completely filled it, we can hear a systolic vascular murmur over the affected spot, as has been observed by Litten. The diagnosis, however, becomes certain if the further signs of infarction appear later.

The most characteristic symptom of infarction in the lungs is the bloody expectoration. If we see quite suddenly bloody sputum in a patient with mitral stenosis, we are usually right in assuming a hæmorrhagic infarction of the lung. Either the sputum consists almost entirely of dark blood, or the blood is mixed with more or less mucus; but there is never much air in it. The bloody expectoration often lasts for several days.

We try to learn more of the size and situation of the lesion by a physical examination of the lungs. Of course this often gives a negative, or at least a doubtful, result. It goes without saying that small infarctions, and also those which are central, can not be made out by physical examination. Large peripheral infarctions may give rise in many cases to dullness on percussion, crepitant râles, and harsh or bronchial respiration, but it is often hard to decide in an individual case whether the physical signs which we meet with are not due to other pathological changes in the lungs, such as bronchitis or hydrothorax. We sometimes hear a pleuritic friction-sound in some part of the chest a few days after we suspect that an infarction has occurred, by which the diagnosis gains additional certainty. We have already mentioned the subjective symptoms in embolism of a large pulmonary vessel—sudden dyspnœa and oppression. Small nodules often cause no special symptoms, but in other cases the patient feels a severe pleuritic pain, due to irritation of the pleura.

Fever may be wholly absent, though we sometimes see a moderate rise of temperature at the onset.

The embolic abscesses in the lungs hardly ever give rise directly to clinical symptoms. They form a part of the general picture of pyæmia and similar general infectious processes. Marked symptoms on the part of the respiratory apparatus are seen only when the abscesses are present in very large number. If an empyema develops from a focus which extends to the pleura, it sometimes occasions definite physical signs.

It follows from all that has been said, that in the diagnosis of embolic processes the chief stress must always be laid on the presence of an ætiological factor. We must regard the bloody sputum as the main direct symptom in hæmorrhagic infarction. Embolic abscesses in the lungs may often be suspected in pyæmic diseases, but they can hardly ever be diagnosticated with certainty.

The prognosis is entirely dependent upon the underlying disease. In heart disease the occurrence of a hæmorrhagic infarction is usually on the whole an unfavorable sign, since it points to weakness of the right ventricle, and hence to the formation of a thrombus in it; yet it often happens that the symptoms of a pulmonary infarction may pass away entirely.

We need not give special directions for treatment. It is in part purely symptomatic, and in part coincident with treatment of the underlying affection. As regards prophylaxis, we must bear in mind the absolute necessity of as perfect rest as possible for those patients in whom the presence of venous thrombi—e. g., in the femoral veins, suggests the possibility of pulmonary embolism.

CHAPTER XI

BROWN INDURATION OF THE LUNGS

(Lungs of Heart Disease)

In heart disease, especially in mitral stenosis, we often find a peculiar change in the lungs, whose origin must be sought in the long-persisting engorgement of the pulmonary circulation. The lungs are hard and dense, and show on a fresh section an abnormal brownish-yellow color. In the larger pulmonary vessels, both arteries and veins, there is a thickening and cloudiness of the intima as a result of the stasis. We see here and there on the surface of the section, and beneath the pleura, little dark spots of pigment and fresh hæmorrhages. We term this condition brown induration of the lungs.

Microscopic examination shows that the capillaries are evidently dilated and twisted as a result of the persistent stasis. They even extend a good way into the alveoli, whose lumen is thus actually diminished. The interstitial connective tissue seems somewhat thickened, and we find in it many brown pigment-granules, the remains of the extravasated and decomposed red blood-corpuscles. The pigment-granules are some of them free, and some contained in cells (escaped leucocytes). In the intima of the larger vessels we often find fatty degeneration of the endothelium.

With regard to the clinical importance of the pulmonary changes due to heart disease, we should say that it is very probable that the dyspnœa of such patients is aggravated by the diminution of the alveolar spaces throughout the lungs, as a result of their being crowded with desquamated pulmonary epithelium. Clinically, however, we can not well distinguish this factor from the other causes of dyspnœa.

We have no positive factors by which to diagnosticate brown induration of the lungs during life. The anatomical lesions, too, show a certain variation, not always to be explained, in that, under apparently the same conditions, the brown induration is often very marked, and often extremely slight. In cases in which we find this induration in the cadaver we have repeatedly heard, during the patient's life, a very sharp, puerile respiratory murmur, which seems to be characteristic of many cases of the "heart-disease lung." We might lay still more stress upon the presence of large, characteristic cells in the expectoration, which are thickly filled with large and small yellow or brown pigment-granules (see Fig. 36). These large pigmented cells ("cells of heart disease") are especially abundant in the expectoration of patients with extreme mitral stenosis. They are identical with the above-mentioned pigment-cells which are found in the lymph of the alveoli after death. Many investigators regard them as alveolar epithelium, while we are more inclined to regard them as mainly leucocytes which have taken up the pigment of the disintegrated red blood-corpuscles. Besides these pigment-cells we also frequently see the still intact red blood-corpuscles in the expectoration of patients with heart disease.

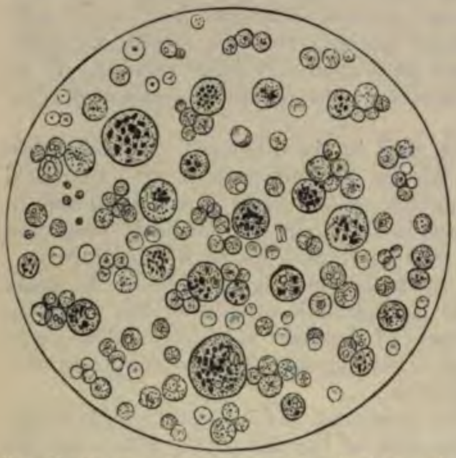


FIG. 36.—Sputum of a patient with mitral stenosis, containing the so-called "cells of heart disease." (Personal observation.)

The prognosis and treatment coincide with those of the underlying cardiac disease.

CHAPTER XII

TUMORS OF THE LUNGS. CANCER OF THE LUNGS. ECHINOCOCCUS OF THE LUNGS. PULMONARY SYPHILIS

1. New Growths in the Lungs. Cancer of the Lungs.—Most of the new growths which are met with in the lungs are of a secondary nature. Secondary cancers are sometimes found in the lungs, with carcinoma of other organs, whose

origin may always be explained by supposing a growth of the primary tumor into a vein, and the consequent carriage of the germs of the growth to the lungs. These secondary nodules in the lungs usually cause no special clinical symptoms, unless they are very numerous and extensive, when they give rise to dyspnœa, and physical signs. There once came to the clinique in Leipsic a case of secondary, and very extensive, miliary carcinosis of the lungs, which ran a brief and fatal course, simulating acute miliary tuberculosis with predominant pulmonary symptoms.

Other secondary new growths deserving mention are enchondroma and sarcoma. We have seen an extensive development of secondary pulmonary sarcoma, following primary sarcoma of the bronchial lymph-glands, and also following lympho-sarcoma of the lymph-glands of the neck, which grew into the jugular vein. We have also seen secondary deposits of sarcoma repeatedly in connection with primary congenital sarcoma of the kidney (see page 644).

Enchondroma, sarcoma, and endothelioma appear, also, as primary new growths in the lung. Pulmonary cancer is, however, the only primary tumor which has any great clinical importance. In its clinical relations we can also rank with it certain malignant, metastatic forms of alveolar sarcoma. The typical cancer of the lungs is always a cylindrical-celled carcinoma, which undoubtedly arises from the bronchial epithelium. It is especially common in elderly persons, over forty, and seems to be found somewhat more frequently in the right lung than in the left, and in the upper lobes than in the lower. By its diffusion the lung-tissue in the parts affected by cancer is changed to a yellowish-gray and quite soft and crumbling mass, devoid of air. We can usually scrape away from the section the characteristic cancer-juice, in which the microscope shows the typical cancerous elements. The pleura is very often involved. The new growth has either extended directly into it, or single, and more circumscribed, secondary nodules have formed in it. The lymph-glands are almost invariably affected, especially the bronchial glands, and also the axillary and cervical glands. Secondary carcinoma of other organs is rare, but it is found in some cases in the other lung, the liver, the brain, and elsewhere.

It is almost always difficult to interpret correctly the clinical symptoms of cancer of the lungs at the beginning of the disease. They are referred to some other, more common chronic pulmonary disease, such as chronic bronchitis, phthisis, or pleurisy, but in the further course of the disease we succeed, at least in a number of cases, in making a correct diagnosis. In other cases, especially in old people, the growth may remain latent.

The general pulmonary symptoms have little that is characteristic. The patient complains of gradually increasing difficulty in respiration, and of pressure and distress in the chest, which may finally increase to the most intense dyspnœa. Most patients suffer very much from the labored, frequent, and spasmodic cough. The expectoration in some cases has no peculiarity, but it often assumes, at least for a time, a characteristic consistency which is extremely important for diagnosis. It contains blood, and also has a peculiar "currant-jelly-like" appearance. Under the microscope we can sometimes make out the characteristic elements of the tumor in it. Severe hæmoptysis is also seen in cancer of the lungs.

Physical examination of the lungs gives positive results in many cases, such as dullness, bronchial respiration, diminished respiration, râles, and sometimes pleuritic friction-sounds, none of which has anything characteristic in itself, although these signs may be of distinct significance in making out the seat and the extent of the new growth. Yet sometimes the area and peculiar limitations of the dullness—for example, if confined to the sternum—are so different from usual that from these factors alone suspicion of a new growth is excited. The sense of resistance upon percussion is quite considerable; the respiratory murmur over the tumor is often inaudible or concealed by the stridor due to encroachment on the lumen of the larger bronchi. Finally should be noted the not infrequent appearance of a diffuse swelling, and sometimes of a slight œdema of the skin over the affected spot.

The occurrence of certain sequelæ is of great diagnostic significance. The chief one is swelling of the lymph-glands in the neck or axilla, and also certain symptoms of compression, which either are produced directly by the new growth, or are due to the secondary enlargement of the lymph-glands. Pressure on the superior vena cava, or a large branch of it, causes œdema in the face, neck, over the wall of the chest, or in one arm. The subcutaneous veins in the regions named appear dilated and tortuous. Pressure on the œsophagus causes difficulty in deglutition; pressure on the brachial plexus, intense neuralgic pains and paresis of one arm; pressure on the recurrent nerve, paralysis of the vocal cords and hoarseness; pressure on the trachea or a primary bronchus, the symptoms of tracheal or bronchial stenosis. The pleura is often involved so that the signs of a pleuritic effusion associate themselves with the other symptoms. It is of diagnostic importance that in such cases the exudation is not infrequently hæmorrhagic (*vide infra*, new growths of the pleura). Besides the symptoms already mentioned we must consider the general symptoms. As in carcinoma in general, so in pulmonary carcinoma, the well-known cancerous cachexia gradually develops. The patient grows dull, loses his appetite more and more, disturbances of digestion and sometimes moderate elevations of temperature develop, until he finally succumbs to general marasmus.

The whole duration of the disease is from six months to two years. The prognosis is fatal. The treatment can be only symptomatic, and we employ the same remedies as in other pulmonary affections.

We must still briefly consider a new growth in the lungs which is extremely interesting from a theoretical point of view. In workmen in the cobalt mines of Schneeberg, in the Saxon Voigtland, the development of malignant lymphosarcomata in the lungs, with the occasional formation of metastases in the glands, the liver, the spleen, etc., is of frequent occurrence. The disease runs its course under the type of a chronic pulmonary affection, and almost always ends fatally. The endemic occurrence seems to point to an infectious origin for the tumor.

2. ECHINOCOCCUS OF THE LUNGS.—Primary echinococcus of the lungs is very rare. In most cases the echinococci are brought to the lungs secondarily from other organs, either by way of the blood-current, or, as is far oftener the case, by perforation of an echinococcus of the liver through the diaphragm.

The symptoms of echinococcus of the lungs are manifold. The parasite sometimes remains entirely concealed. In other cases a more or less severe, and often febrile, affection of the lungs, is developed, with pain in the chest, cough, and sometimes bloody expectoration, and dyspnœa. Physical examination of the lungs gives in some cases dullness, absence of respiratory murmur, and diminished vocal fremitus, while after the expectoration of the echinococcus (*vide infra*) symptoms of a cavity may ensue. A correct interpretation of all these symptoms is possible only when, as has often been observed, the echinococcus cysts are coughed up, or at least when parts of them, like the membranes or the hooklets, are found in the expectoration.

The termination of the disease may be favorable if the echinococci are expectorated, or if we succeed in removing them by operative means. We can hardly hope to be able to kill the parasite by inhalations of turpentine or benzine. Sometimes the echinococcus cyst becomes gangrenous, or suppurates. Rupture into the pleura, into the peritoneum, into the pericardium, and externally, has also been observed. This last termination is the most favorable, since otherwise, if the affection progresses, a fatal result may be caused by the sequelæ, or rarely by the occurrence of suffocation. The details of the natural history of the echinococcus are given in the chapter on echinococcus of the liver.

3. Pulmonary Syphilis.—This would also be the place to speak of syphilitic new growths in the lungs, but, in our opinion, in spite of the quite abundant literature of this subject in recent times, no definite clinical description of pulmonary syphilis can be made. Those physicians who are disposed to consider every pulmonary disease in a previously syphilitic subject to be of a syphilitic nature, certainly regard many things as pulmonary syphilis which have nothing at all to do with syphilis. At least, we have found that all those cases which at first suggested a diagnosis of pulmonary syphilis, finally, upon more accurate examination and after longer observation, have turned out to be something else, usually tuberculosis. There are a few indubitable cases of chronic indurative contraction of the lungs following primary syphilitic infiltration. The clinical picture here does not differ in any characteristic manner from that of ordinary chronic interstitial pneumonia. A probable diagnosis may be made from the knowledge of the syphilitic taint, the simultaneous existence of other syphilitic lesions—e. g., of the liver—the physical signs of chronic pulmonary disease, and the failure to find tubercle bacilli in the sputum on repeated examinations. It is also well established that there may be syphilis of the larger and medium-sized bronchi, which is recognized at the autopsy by extensive radiating cicatrices in the bronchial mucous membrane, which sometimes lead to stenosis. Single gummatous nodules in the lungs are of the greatest rarity. We sometimes find in the pleura peculiar radiating cicatrices, which probably are of syphilitic origin. The pulmonary syphilis of the new-born, which occurs in the form of single nodules or as a diffuse syphilitic infiltration, the so-called *pneumonia alba*, has only a pathological interest.

If the existence of a syphilitic disease of the lungs is suspected, of course specific treatment should be employed. Iodide of potassium must be administered and perhaps also a course of mercurial inunctions. The results are not apt to be brilliant, however, because it is of course impossible to restore the portions which have been cicatrized.

SECTION V

Diseases of the Pleura

CHAPTER I

PLEURISY

(*Pleuritis*)

Ætiology.—Pleurisy is divided into primary and secondary. In a strict sense, the only cases of pleurisy which can be termed primary are those in which the pathogenic organisms penetrate into a healthy body, and become localized in the pleura without any previous organic lesion elsewhere. No doubt many a case of pleurisy seems primary from a clinical standpoint, which is really not so. The disease antecedent to the pleurisy may be so slight as to present no symptoms and attract no attention. The pleurisy appears as an apparently independent disease, and is the first thing which attracts attention to the previously existing affection.

Exclusive of traumatic pleurisy, resulting from such injuries as a penetrating thoracic wound, we know positively of only one form of pleurisy—viz., the rheumatic—which is primary. This is, from an ætiological point of view, most closely allied with acute articular rheumatism (Fiedler). In polyarthritis (see page 680) it is not very exceptional to have pleurisy develop secondarily. In many cases the rheumatic affection attacks the pleura at first, without any great involvement of the joints. Then often follow articular trouble, endocarditis, and the like, confirming, or for the first time disclosing, the ætiology of the attack. Whether still other pathogenic influences may produce primary pleurisy is not settled. Some cases of acute pleurisy, setting in with high fever, we have felt justified in referring to diplococcus infection, as suggested by the association of herpes and other symptoms. In such instances it is scarcely possible to exclude with certainty the presence of a small focus of pneumonia. In genuine primary pleurisy the pathogenic germs must first enter the circulation, and by that means reach the pleura. It is frequently impossible to find out any special exciting cause for the attack. Often it is ascribed to catching cold.

Among those varieties of pleurisy which appear from a clinical standpoint as primary, while their existence is really referable to a previous lesion of the body, by far the most frequent and important is the tubercular. On this point it was only gradually and by means of long experience that physicians obtained a correct conception of the truth. We believe it right to say that the great majority of all cases of apparently primary pleurisy are tubercular. The infection of the pleura is due to the invasion of it by germs from some tuberculous focus, situated near by. Either there are small tuberculous patches in the lungs which extend to the pleura, or, probably yet more frequently, bronchial or retro-bronchial lymph-glands, being tubercular, break into the pleural cavity and promptly excite pleurisy. In many of these cases the further course of this disease clearly shows that the pleurisy was tuberculous at the start, and not infrequently the physican is justified, even at the very beginning of the illness, in expressing his suspicion of tuberculosis (*vide infra*, diagnosis). In many other cases of pleurisy its secondary character is clear from the start. Most of these occur from the direct extension of the inflammatory process, from some neighboring organ, to the pleura. When considering pulmonary diseases, we had occasion to point out that the various pathological changes in the lungs, when they extend to the pleura, involve it in the disease. Thus we see pleurisy associated with croupous pneumonia, lobular catarrhal pneumonia, pulmonary gangrene, hæmorrhagic infarction, embolic abscesses, and, most important of all, pulmonary tuberculosis. Many of the diseases just enumerated are frequent complications of the most diverse diseases. Hence, it is easy to understand that pleurisy is a not infrequent phenomenon in all sorts of severe illnesses.

Inflammation from other neighboring organs beside the lungs may extend to the pleura. Inflammation of the contiguous serous membranes may spread to the pleura by continuity; thus pleurisy complicates pericarditis and peritonitis. Inasmuch as the pleura and the outer surface of the pericardium lie in direct apposition and, furthermore, the pleural cavity and the peritoneal cavity are directly connected by the lymph-channels of the diaphragm, we can easily understand that not only serous and purulent, but also diplococcus, inflammation of the pericardium and peritoneum may have pleurisy for a sequel.

Another variety of secondary pleurisy is due to the conveyance of the inflammatory organisms not from the immediate neighborhood, but from other parts, by way of the circulation. Here should be mentioned pleurisy due to general sepsis, to polyarthritis, to nephritis (see page 632), and to genuine gout (which see). The factors which give rise to the inflammation are either organic or, in the case of gout and nephritis, chemical in their nature. The inflammation they excite in the pleura is most varied in its kind and degree.

Of late years bacteriological investigations have been made by E. Levy, Prince Ludwig Ferdinand, and others, to obtain a more accurate knowledge as to the special variety of germs in the various forms of pleurisy, due to infection with organized material. It must not be forgotten, however, that these bacteriological investigations have been, for the most part, directed not to the diseased tissue, but to the inflammatory pleuritic exudation. This, however, in many cases contains no bacteria whatever. In particular, the exudation in most cases of tuberculous pleurisy, whether serous or purulent, is absolutely sterile, and in the secondary pleuritic exudations, in acute polyarthritis and nephritis, the search for bacteria has failed again and again. In other cases of pleurisy, however, staphylococci have been found in the exudate, and in empyema streptococci also. Metapneumonic pleurisy, whether serous or purulent, is often, though not always, characterized by the presence of genuine pneumococci. In general, the question with regard to the special cause of the disease will have to be whether the pleurisy, in each case, is a part of the original primary disease—that is, whether we find tubercle bacilli in tuberculosis and diplococci in pneumonia, or whether the pleurisy is a secondary complication, so that, for example, we find streptococci in typhoid fever. Further investigations are needed for complete enlightenment with regard to all these matters.

Pathological Anatomy.—The inflamed pleura is markedly injected, it has lost its normal luster, and instead has a dull surface. This dullness is due to the coagulated fibrinous exudation upon the pleura, the exudation, in mild cases, forming only a thin layer. In more advanced cases, however, the surface of the pleura is covered with thick, rough, and shaggy masses of fibrine. As long as the fluid in the pleura is little or not at all increased, we speak of a simple fibrinous or dry pleurisy (*pleuritis fibrinosa vel sicca*).

In other cases, however, besides the layer of fibrine there is an abundant exudation of fluid from the capillaries of the pleura, forming a pleuritic effusion. This is ordinarily of a simple serous character—serous and sero-fibrinous effusions. The fluid collects between the surfaces of the pleura, or, if there is at the same time an abundant coating of fibrine, between the gaps and in the meshes of the fibrinous exudation. In such cases there are often many flakes of fibrine floating in the fluid.

Upon microscopic examination there are invariably found a few leucocytes even in serous exudations, and also sometimes a very few blood-corpuscles and endothelial cells (often swollen or fatty-degenerated), and plates of cholesterin.

If the number of pus-corpuscles in the exudation becomes much increased, we have a sero-purulent, or a purulent exudation. This is always due to the presence of a specific organized poison which excites the suppuration. The pleurisies which come from embolic abscesses, from gangrenous foci in the lungs, and from carious ribs, and those which arise from the rupture of tubercular cavities into the pleura, are almost always of a suppurative character. We call the purulent pleuritic effusion empyema. If putrefactive agencies enter the pleural cavity at the same time with the pus poison, as in the pleurisies which develop in pulmonary gangrene, the purulent exudation assumes an ichorous, putrid character—ichorous effusion.

Under certain circumstances the effusion assumes a hæmorrhagic character—hæmorrhagic effusion—especially if hæmorrhages occur from the old or newly formed capillaries dilated by the inflammation. They arise partly by diapedesis and partly from rupture of the walls of the vessels. The exact cause of the hæmorrhages is usually unknown. We know by experience that hæmorrhagic effusions are most frequent in tubercular pleurisy, a fact which is of diagnostic importance. The exudation may also be hæmorrhagic in connection with new growths of the pleura, after severe croupous pneumonia, in septic disease (e. g., puerperal fever), and finally when there is a general hæmorrhagic diathesis, as seen in scurvy, purpura hæmorrhagica, and leukæmia. In all these last-mentioned cases it should, of course, be considered that we are not always dealing with a true inflammatory exudation, but merely with a hæmorrhage into the pleural cavity.

The amount of fluid collected in one pleural cavity is, in the majority of cases, somewhere between a pint and a quart (500–1,000 cubic centimetres), but it may reach three or four quarts. Every large effusion must influence the position of the yielding walls of the pleural cavity, the chest-wall, the lungs, the mediastinum, and the diaphragm, through the consequent increase of pressure in the affected pleural cavity; and the resultant symptoms of pressure on the neighboring organs are of the greatest clinical significance. Attention is first called to the lungs themselves. Since the normal lung is expanded in the thorax beyond its elastic equilibrium, it will retract as soon as a part of the pleural cavity is occupied with fluid. Until it has reached its position of elastic equilibrium there can be no question of a positive pressure on the lung. The lung floats on the effusion, in a certain way, if there be no adhesions, but, as the amount of the fluid further increases, compression of the lung follows. With a very large effusion the lung is pushed wholly up and back against the vertebral column, and is changed to an almost bloodless, airless, flat mass. It is, however, possible that the atelectasis of the lung is not caused exclusively by compression from without, but that, after the normal respiratory movements have ceased, a part of the air in the lung may be absorbed by the vessels, or even by the effusion.

We also see the results of the pressure exerted by the pleuritic effusion on the mediastinum and diaphragm, as well as on the lungs. Displacements of the heart arise from the lateral pressure on the mediastinum, which must take place if the pressure in the diseased pleural cavity is equal to that of the atmosphere, for a greater and positive pressure is unnecessary, since a negative pressure prevails on the healthy side.

The downward pressure of the diaphragm, which usually affects both halves of it, although in unequal degree, makes itself manifest on the right by the low position of the liver, and on the left by the downward displacement of the stomach and large intestine. It must be particularly noticed, however, that adhesions may prevent all the pressure-displacements which we have mentioned, both of the lungs and of the neighboring organs.

As regards the further changes and terminations of the pleuritic processes, they depend upon the amount and character of the effusion. Favorable cases may result in complete recovery and absorption of the effusion. The fluid contents are taken up directly by the lymphatics of the pleura, and the solid constituents, the fibrine and the white blood-corpuscles, are decomposed, dissolved, and absorbed. Old fibrinous patches sometimes become impregnated with lime salts, so-called "pleuritic ossification."

Whenever a case of extensive pleurisy, with an abundant fibrinous or fluid exudation, gets well, there is a marked cicatricial contraction of the pleura, in which the whole thoracic wall is involved. It requires months for the lungs and the thorax to regain their normal expansion, if they can ever do so.

That recovery from large pleuritic exudation is so often incomplete, is explained for the most part by the nature of the original disease. We often find that temporary improvement is followed by a fresh relapse of pleurisy, or by the appearance of extensive and usually tuberculous disease of the lungs or other organs.

In the case of purulent exudations, also, final absorption of the fluid is possible. This is especially likely when the empyema is meta-pneumonic and benign, but this demands much time, and thick, cheesy masses of pus are often left in the pleural sac. In most cases of empyema, if there is not timely operative interference, the pus seeks an outlet for itself. It may break through the visceral pleura into a bronchus, and be emptied externally, thus giving rise to a pyo-pneumothorax; but in many cases the pleura seems to be destroyed only superficially, and the pus is pressed into the alveoli as into a sponge, especially by the movements of coughing, and thence reaches the bronchi, without letting the air enter the pleural cavity (Traube). In other cases the empyema breaks externally through the chest-wall—*empyema necessitatis*. The point of rupture is usually found in the vicinity of the sternum, where the chest-wall is thinnest. In very rare cases the empyema breaks into the deeper parts of the trunk, or into the abdominal cavity.

Course of the Disease.—We will speak in what follows especially of the course and symptoms of ordinary, apparently primary (*vide supra*), fibrinous or serofibrinous pleurisy, the so-called simple pleuritic effusion. What is said of it obtains in large measure in the other form of pleurisy also. The physical signs, of course, are almost wholly independent of the character of the effusion. As far as the different forms of pleurisy differ clinically, we will mention their peculiarities below.

Only rarely is the onset of pleurisy quite acute and sudden, beginning with a rigor. In such cases we must guard against confusing it with croupous pneumonia. Pleurisy usually begins slowly and gradually. The symptoms, which the patient himself feels, are in many cases to be referred directly to the disease of the pleura. One of the most constant is the pleuritic pain, the stitch in the side. A more or less severe pain comes on in the side at every deep breath, and hence upon any physical exertion; also upon movements of the body, in stooping, coughing, or gaping. Shortness of breath soon appears, and constantly increases. There is often a frequent, dry cough. Sometimes there are scarcely any cough and expectoration. Such expectoration as there may be is usually simply mucous. Besides that, severe general symptoms almost always develop; the patient feels dull, looks pale, and has no appetite. Patients who can endure a good deal often keep at work for a long time, until, after feeling miserable for three or four weeks, they are forced to stay at home and summon a physician. It is very important to know that in not a few cases the general symptoms are much more prominent, at the beginning of pleurisy, than the local ones. The patient comes to the physician complaining only of weakness, loss of appetite, or headache, and the physical examination is the first thing that shows the presence of perhaps a large pleuritic effusion.

In most of the severe cases the further course is slow like the beginning, but sometimes the severest symptoms, most intense dyspnœa, marked cyanosis, etc., may come on in a short time, owing to a sudden increase of the effusion. On the other hand, in mild cases the symptoms may disappear again in a few weeks, but the objective signs in such cases are generally to be made out for a longer time. The disease ordinarily lasts for at least five or six weeks, and often much longer. Gradual recovery follows, or the onset of some new disease, usually tubercular (*vide infra*).

Single Symptoms.—The *pleuritic pain*, the stitch in the side, is one of the most frequent subjective symptoms. We have previously mentioned that in primary diseases of the lungs, too, as in croupous pneumonia, the stitch in the side is due to the accompanying pleurisy. It is remarkable that the intensity of the pain in no way corresponds to the apparent intensity of the disease. There is often the severest pain in the side when the physical examination shows almost nothing. On the other hand, we often hear a decided pleuritic rub without the patient's complaining of any special pain. Pressure on the chest-wall on the affected side is often very painful. With severe pain we may consider the possibility of an invasion of the intercostal nerves by the inflammation. We have never observed cases, like those described by some authors, of "crossed pleuritic pain"—that is, cases in which the pain is localized on the side not affected.

Cough and Expectoration.—The cough is probably excited directly by the disease of the pleura. We often find that the pain in the side and also the cough are brought on by a deep inspiration. Expectoration is entirely absent in uncomplicated pleurisy, or it is scanty, and consists simply of mucus. Much expectoration always means a pulmonary complication. A large amount of purulent sputum is evacuated if a purulent effusion breaks into the lungs (*vide supra*).

Dyspnœa.—The respiration is usually shallow, and consequently frequent, because of the pleuritic pain. In every large effusion which prevents respiration in one lung the dyspnœa becomes more severe, and may, with a very extensive effusion, reach the highest degree of orthopnœa. The stronger the patient was before the disease, and the more rapidly the effusion develops, the more severe, as a rule, is the dyspnœa.

Fever.—Most severe pleurisies are associated with fever, but its height is not very great, so that it quite rarely reaches 104° (40° C.). The fever has no typical course. In cases with an acute beginning it is sometimes quite continuous, or slightly remitting at first. If improvement takes place, the fever goes down in about two or three weeks by lysis, so that this part of the temperature-curve may be precisely like the period of defervescence in typhoid.

In the more protracted cases the fever gradually becomes more remitting, varying between 100° and 101° (38°–38.5° C.), and it assumes more and more the form of hectic fever. The longer the evening rise of temperature lasts, the more we are justified in suspecting tuberculosis. In empyema we see a higher, irregular fever, sometimes associated with severe chills.

The pulse is constantly rapid, up to 100 and over. In all severe cases the strength and tension of the pulse are much diminished. Irregularity of the pulse is not infrequent. All these changes are probably due in great part to the pressure of the effusion on the heart and large vessels. Lichtheim has discovered experimentally that it is not the compression of the vessels in the compressed lung which lowers the arterial tension.

General Symptoms.—Pleurisy is almost always associated with a pronounced general malaise, muscular weakness, and dullness. The patient is pale, and often markedly cyanotic in cases with much disturbance of respiration. There is great emaciation if the disease is of long duration.

The appetite declines from the outset. There is often occasional vomiting, especially in the first period of the disease. The bowels are usually constipated. Many patients complain of headache.

The condition of the urinary secretion is very important. In every pleuritic effusion the amount of urine is decidedly diminished so long as the effusion increases or remains at the same height. The daily amount is sometimes only eight or ten ounces (200–400 cubic centimetres). The urine is also concentrated, its specific gravity being about 1020–1028. Sediments of urates often form. This diminution of the excretion of water by the kidneys is largely the result of the diminished arterial pressure. An increase of the amount of urine is always a favorable symptom, often the first sign of beginning absorption of the effusion. If a large effusion is rapidly absorbed, the amount of urine may increase to eighty or a hundred ounces (2,500–3,000 cubic centimetres) daily. The urine, then, of course, is abnormally clear and of low specific gravity.

Physical Signs

1. **Fibrinous Pleurisy—Pleuritis Sicca.**—Simple fibrinous pleurisy sometimes gives rise to no physical signs at all. If it develops as a result of some pulmonary affection, the physical signs present are often dependent upon the latter alone.

In many cases, however, dry pleurisy may cause marked objective signs. On inspection, we are struck by the impaired mobility of the affected side on respiration, which is due to the pain caused by breathing. Because of this same tenderness the patient at first often lies on the sound side. Percussion gives no qualitative change of resonance as yet. With the beginning of exudation slight dullness appears, at first almost always in the posterior portion of the lungs. Sometimes the resonance becomes tympanitic as a result of retraction of the lung. We can almost constantly make out, especially in the back, that the lower edge of the lung moves less than usual on respiration. Auscultation gives a respiratory murmur that is either qualitatively unchanged or indefinite, but it is always diminished. The peculiar pathognomonic sign of dry pleurisy, however, is the pleuritic friction-rub, that characteristic rubbing, grating, creaking sound, which arises from the sliding of the rough pleural surfaces over each other, and is detected especially in the lateral portions of the thorax. We can hear it both on inspiration and on expiration. It is often jerky, one rub following another after a considerable interval. If we are sure we hear a pleuritic rub, it is direct evidence of the existence of a dry pleurisy, but its absence will not let us exclude pleurisy. The friction-sound must be absent if there are pleuritic adhesions. We can often feel a marked rub by laying the hand on the chest. Sometimes the patient feels it himself, but in other cases he has no sensation of it. We may confound a slight rub with fine crepitant râles. Repeated examinations before and after the patient has coughed usually confirm the diagnosis, since the râles are often changed by coughing.

To be distinguished from the mild form of dry pleurisy just described is that variety in which there is an extensive and abundant fibrinous exudation, with scarcely any liquid. We have repeatedly seen severe cases following pneumonia, or even apparently primary cases, which displayed marked dullness and sense of resistance on percussion over the whole of one side of the chest, with diminished or absent respiration, and yet upon exploratory puncture only a few drops of serous exudation could be obtained. Evidently we had to do with the formation of large amounts of coagulated fibrine. Attacks of this sort usually run a severe and tedious course, but they may, nevertheless, get well at last.

2. **Pleuritic Effusion.**—Small amounts of fluid in a pleural cavity escape discovery. Physical signs first appear when the amount of effusion reaches eight or ten ounces (200–300 cubic centimetres).

Inspection shows first the more or less marked impairment of motion on the affected side on respiration. If the amount of the effusion is large, there is an evident distention of the affected side in the lower posterior and lateral portions of the thorax. The intercostal spaces are flattened or even a little convex. The nipples and shoulder-blades are farther removed from the median line on the affected side than on the healthy side. The hypochondrium on the affected side is more prominent. In an extraordinarily large effusion on the left side we have seen and felt, in the left hypochondrium, the lower surface of the diaphragm, which was actually arched downward. By direct measurement in severe cases we can make out that the affected side is expanded several centimetres.

With every large effusion there is marked dyspnœa and accelerated respiration. The slight excursions of the affected side on respiration are usually very striking, while the sound side moves so much the more. In this stage of pleurisy the patient often lies upon the affected side, in order to breathe with the healthy lung with as little restraint as possible. With large effusions complete orthopnœa may develop.

The signs due to displacement of the neighboring organs, which are noticeable on inspection, will be mentioned below in the appropriate connection.

Everywhere that a layer of fluid comes between the lung and the chest-wall there is a loss of clearness in the percussion-note. If the thickness of the layer of effusion is five or six centimetres, the resonance seems completely dull or flat. The pleuritic dullness is almost always made out first in the lower posterior portions of the thorax, more rarely in the lower lateral portions. With a slight effusion the height of the dullness is only a few centimetres, but, with much effusion, it rises higher in the back and the lateral portions of the thorax; and on the right, resonance gradually grows dull anteriorly and inferiorly, above the liver. With very large effusions the dullness may begin in front at the second or third rib, or in rare cases even the whole half of the chest, front and back, may give a totally flat percussion-note. Pleuritic dullness is always attended with a marked feeling of resistance on percussion.

With medium-sized effusions, when the dullness does not extend over the whole back, the upper boundary of the dullness usually forms an oblique line, highest at the vertebral column and thence running obliquely downward to the side of the thorax. Exceptionally the line runs the other way. On the right it is not possible to distinguish by percussion the lower limit of the exudation from the liver. On the left we can often distinguish its lower limit from the tympanitic resonance of the stomach, and are thus aided in diagnosis (*vide infra*, displacement of organs).

[In moderate effusion without adhesions or pneumonic complications, the line of flatness in the back, the patient being in the vertical position, is lowest near the spinal column, and rises in a curve like the letter S as it passes outward toward the axillary region. The experiments by Dr. Garland, of Boston, with reference to this point are well known. Before attempting to mark out this line the patient should be told to take several deep inspirations, in order to inflate the triangular portion of lung which dips down near the vertebræ.]

The percussion-note above a pleuritic effusion deserves attention. The beginning of pleuritic dullness is almost always relative, gradually passing to an absolute flatness. The pulmonary resonance above the beginning of dullness is usually tympanitic, from retraction of the lung-tissue. We find the tympanitic resonance beautifully distinct in large effusions in the first and second intercostal spaces in front. It is loud and deep, and remains unchanged with the mouth open—Skoda's resonance. With very large effusions, which cause an actual compression of the lung, we sometimes find, in the second intercostal space, a dull tympanitic resonance, which becomes higher on opening the mouth. This resonance arises from the vibrations of air in a large bronchus surrounded by compressed lung—"Williams's tracheal tone." With large effusions we sometimes hear over the retracted lung, in the upper anterior intercostal spaces, a distinct buckram sound—the "cracked-pot sound."

Displacement of the neighboring organs, which is made out chiefly by percussion, forms one of the most important physical signs in pleurisy with effusion.

In right-sided effusions the liver, especially the right lobe, is displaced downward. We find the lower border of the liver dullness extending several centimetres below the ribs. In very large effusions the liver may be pushed down to the level of the umbilicus. The pushing of the mediastinum to the left in large effusions may be recognized upon percussing from the right toward the left, by dullness over the upper part of the sternum, reaching to or beyond the left border of the sternum. The displacement of the heart to the left in the majority of well-marked cases is associated with a displacement of the apex of the heart upward.

This is easily explained by considering the position of the heart and the direction of the pressure, which first acts from below. We recognize the displacement of the heart chiefly by the position of the apex-beat, which is seen and felt at or outside the left mammillary line in the fifth space, or often higher, as we have said—in the fourth. Percussion gives a corresponding displacement of the left boundary of the cardiac dullness to the left.

In left-sided effusions the displacement of the heart to the right, which can usually be made out even in moderate effusions, is especially noticeable. Resonance over the lower part of the sternum is diminished, the heart's dullness extends to the right border of the sternum or several centimetres beyond it. In the most marked cases the heart is pushed to the right mammillary line. The displacement of the mediastinum is also to be made out over the upper part of the sternum, the dullness reaching to the right border of the sternum or beyond. The low position of the diaphragm is made out by a depression of the left, and in marked cases of the right, lobe of the liver. It is an especially important sign, however, that dullness occurs in the zone, about a hand-breadth wide, of normal tympanitic resonance above the left border of the ribs—the "semilunar space" of Traube. The normal tympanitic resonance here comes from the stomach or large intestine. As the diaphragm is pressed downward the pleuritic effusion presses on these organs. The semilunar space is therefore diminished, and finally, with large effusions, there is absolute dullness down to the edge of the ribs.

Changes in dullness in pleuritic effusions may occur with a change of the patient's position, but they may often be absent on account of adhesions. As a rule the normal change in the position of the lower border of the lung corresponding with respiration is abolished.

Auscultation always gives a diminished respiratory murmur over the pleuritic effusion. With a beginning effusion it may sound approximately vesicular, but later it becomes indefinite, harsh, and finally bronchial, if the larger bronchi remain open for the respiratory current of air. The bronchial respiration sounds distant and low, and has the character of the sharp German *ch*, but in rare cases it also assumes a distinct amphoric tone, so that it sounds almost like a cavernous respiration. The respiratory murmur may finally disappear entirely over very large effusions. Above the upper boundary of the effusion the respiration almost always sounds harsh. Among the adventitious sounds we must mention the pleuritic friction sound, which of course can be heard only at the upper boundary of the effusion, where the two pleural surfaces meet. Moist râles and rhonchi signify a co-existing disease in the lungs. With slight effusions we often hear, on deep breathing, pure crepitant râles on inspiration, as the walls of the alveoli and bronchioles in the atelectatic lung, which were stuck together, are torn apart.

On auscultation of the voice we sometimes hear bronchophony, and sometimes that bleating, nasal sound known as ægophony. Baccelli advanced the theory that auscultation of the whispered voice might be of service in diagnosticating the character of the effusion. With a serous effusion we can understand a whisper distinctly through the thorax, but not with a purulent effusion, since theoretically the cell-elements destroy the waves of resonance. This theory holds true in many cases, but by no means in all.

On auscultation of the heart we notice, as a result of its displacement, an abnormal extension of the region over which the heart-sounds are audible. If the inflammation spreads from the pleura to the outer surface of the pericardium, we can sometimes hear an extra-pericardial friction-rub, accompanying both the respiration and the action of the heart.

The vocal fremitus is always diminished over the pleuritic effusion, and in marked cases is entirely absent.

3. **Absorption of the Effusion—Pleuritic Contraction.**—The beginning absorption of the effusion is usually first made evident by the percussion-note in the upper part of the dullness becoming clearer and sometimes tympanitic. The respiratory murmur is also plainer. Where it was bronchial it becomes indefinite and gradually vesicular again. The vocal fremitus is again to be felt. All these improvements take place gradually but slowly. It is usually a very long time before the percussion-note resumes its normal clearness.

The changes in the form of the thorax are especially striking. Only in pleurisies with slight effusion does the somewhat expanded thorax resume its old form without further change. After every severe pleurisy with large effusion there is, during its absorption, a marked contraction of the affected half of the chest. In cases of moderate intensity the contraction affects only the lower lateral portions of the thorax, in severe cases the upper and anterior portions as well. We find the most marked contractions in children and young persons with a yielding thorax. The circumference of the affected side is much less than that of the sound side. The ribs are pressed together and the intercostal spaces become very narrow. The fossæ are deepened and the nipples and shoulder-blades are drawn nearer the vertebral column, which takes on an abnormal lateral curvature, in which its convexity is directed usually toward the affected side, but sometimes to the sound side. Dullness and diminution of the respiratory murmur and vocal fremitus continue with the contraction of the pleura, but they no longer depend upon the presence of a fluid effusion, but are due to the pleuritic thickening.

The process of marked contraction always lasts for months, or even longer. In favorable cases the contraction of the thorax may be readjusted very much later, often after years. The thickening is absorbed, and the lungs and thorax gradually expand, but in other cases there are extensive adhesions between the pleural surfaces, especially over the lower lobe, which result in a permanent disturbance of respiration. In almost all cases of pleurisy with contractions there arises a vicarious emphysema in the lung on the sound side.

Complications.—Peculiar complications of pleurisy are rare. Where such occur they are due either to the primary disease which has led to the pleurisy, or to the simultaneous action of the same cause of disease, such as tuberculosis. Hence it happens that we speak of the frequent "complication" of pleurisy with chronic bronchitis or with tuberculosis of the lungs or other organs. It is important to bear in mind that, by a direct advance of the inflammation, the pleurisy may also invade the pericardium, and rarely the peritoneum, through the diaphragm; but we see this extension of the process almost solely in tubercular and purulent pleurisies. We must mention, finally, that we have seen several cases with a large serous effusion, in which an acute hæmorrhagic nephritis occurred. For the paralysis of the arm on the corresponding side observed in some cases of empyema, compare what is said in regard to reflex paralyses on page 833.

VARIOUS FORMS OF PLEURISY

1. **Primary Rheumatic Pleurisy.**—As we have already pointed out, when considering the ætiology of pleurisy, clinical observations have lately led to the belief that some of the cases of acute primary pleurisy are due to the same cause as acute articular rheumatism (which see). Although there are as yet no sufficient number of decisive bacteriological investigations, we are, nevertheless, strongly inclined, from our present experience, to acknowledge the correctness of this view, even if we do not estimate the frequency of rheumatic pleurisy nearly so high, as, e. g., does Fiedler, on the ground of his observations in Dresden.

Rheumatic pleurisy usually develops rather suddenly in persons previously healthy. Sometimes it is preceded by mild rheumatic pains in the muscles or some of the joints. The local discomforts, such as pain in the side, are often very severe, the fever is moderately high, though rarely over 104° (40° C.); and as the case progresses such rheumatic symptoms as swelling of the joints and endocarditis may associate themselves with the pleurisy, and confirm the diagnosis. In general, however, the course of the disease is favorable. The fever lasts, as a rule, only a week or two, and even when there is a large exudation it finally undergoes complete absorption with recovery.

2. **Tubercular Pleurisy.**—In an ætiological sense we must declare the larger part of the ordinary "pleuritic effusions," which clinically seem primary, to be tubercular. The further course of the cases, if we can watch them long enough, almost always permits us finally to recognize the tubercular nature of the disease; yet we can not say that some other tubercular disease, particularly phthisis, is always the immediate sequel of the pleurisy.

In a comparatively small number of cases do the symptoms of acute tuberculosis, or more frequently of chronic phthisis, appear as an immediate result of the pleurisy, which at that time is usually still present or in the contracting stage. The objective changes of phthisis are evident either in the apex or in the lower lobe of the affected side. The fever continues, the pulmonary affection advances, the other lung is also attacked, and the disease takes a fatal course under the type of an ordinary phthisis, now more acute and now more chronic.

In other cases acute tubercular affections arise sooner or later as a result of the pleurisy—tubercular meningitis, or general miliary tuberculosis. In other cases still the disease develops under the form of tuberculosis of the serous membranes, to which we will return again in the description of tubercular pericarditis and tubercular peritonitis. We often have to do with a double pleurisy with no evident complication in the lungs. In varying succession are added the symptoms of chronic tubercular peritonitis, with pain, swelling, and effusion of fluid into the abdomen, or the symptoms of tubercular pericarditis. Death finally ensues with persistent hectic fever and increasing general emaciation and weakness. The whole affection usually runs a chronic course, lasts for months, and often shows marked remissions and temporary improvements.

In very many instances, on the other hand, the pleuritic effusion has throughout an apparently favorable course. After some weeks the fever ceases, the effusion is absorbed, the patient gets up, and is finally discharged as nearly well. Of course, some dullness and retarded motion often remain in the affected side, but even these may gradually disappear. These cases, too, very often turn out in the end to be tubercular. After a longer or shorter period of apparent health, sometimes after the lapse of years, a "new" disease appears—that is, either a return of the pleurisy, a pleurisy on the other side, or some other acute or chronic tubercular affection. In such cases, too, we must look upon the former pleurisy, in an ætiological sense, as tubercular. It is not impossible, however, for even a tubercular pleurisy to recover, and for the recovery to be permanent, if no other organ is at the same time affected by tuberculosis, especially if the lungs remain intact.

Finally we must mention the cases in which a pleuritic effusion develops secondarily to an already pronounced phthisis. Here too we almost always have to do with a tubercular pleurisy.

The anatomical changes in tubercular pleurisy consist in the ordinary signs of inflammation, and also the presence of the specific nodules of tubercle. The number of tubercles differs very much in different cases. The pleura is in some cases completely studded with miliary nodules, and in others we find the tubercles, at least with the naked eye, only in single spots. The effusion is usually of a sero-fibrinous character. Sometimes it is hæmorrhagic, as the majority of cases of apparently primary "hæmorrhagic pleurisy" are generally of a tubercular nature. Empyema also (*vide infra*) occurs not infrequently in association with tuberculosis; and finally, in a few cases the exudation has been of a peculiar milky character, and has contained numerous oil-globules, probably originating from fatty-degenerated and disintegrated leucocytes and endothelium.

3. **Purulent Pleurisy—Empyema.**—A purulent exudation is developed in the pleura when the inflammation of that organ is due to a specific cause which excites suppuration. So far as is yet known, the streptococcus pyogenes seems to be the most frequent factor in producing empyema. It is found in the pus of empyema, due to such causes as external injury, caries of the ribs, pulmonary tuberculosis, pneumonia, and pyæmia. Less often empyema is excited by staphylococci. This is almost always a rather favorable form of the disease. Likewise comparatively benign is meta-pneumonic empyema, which in most instances is caused by the pneumococcus. The empyema of tuberculosis is, as has been already pointed out, in most cases devoid of bacteria (A. Fränkel and others), and here we may have to do with the formation of chemical poisons capable of exciting suppuration.

Empyema usually causes severe symptoms. The fever is higher than in the other forms of pleurisy, but it is irregularly intermittent, and is often associated with chills. There are severe general symptoms besides the fever, such as great languor, headache, a dry tongue, and a rapid pulse. We sometimes notice a slight œdema of the chest-wall on the affected side. Otherwise the local symptoms and disturbances are, of course, the same as in the other forms of pleurisy. If the pus is not evacuated artificially, the empyema may finally break externally or into the lungs (*vide supra*). In the latter case a very large expectoration of pus suddenly occurs, and is usually followed by pneumothorax.

Diagnosis.—Our chief attention in regard to diagnosis is directed to the distinction between pleurisy and acute or chronic pneumonia, which is not very easy in all cases. We will briefly contrast the distinctive features as made out on physical examination.

Inspection.—A marked distention of the affected side points to effusion; it does not occur in pneumonia.

Percussion.—The dullness in pleurisy is complete, and the feeling of resistance on percussion is very marked; in pneumonia, however, the dullness is rarely so marked, and there is often a tympanitic sound. The discovery by percussion of signs of displacement of the neighboring organs is of especial weight, as these signs are always absent in uncomplicated pneumonia, while with few exceptions they can be easily demonstrated in every case of pleurisy when the exudation is at all considerable.

Auscultation.—Diminished or suppressed respiratory murmur points to pleurisy, loud bronchial breathing and râles to pneumonia; but we must not forget that in pneumonia auscultation may give the same signs as in pleurisy, if a bronchus is plugged.

Vocal Fremitus.—Marked vocal fremitus over dullness is direct evidence of pneumonia, diminished or absent vocal fremitus of pleurisy; but the vocal fremitus may also be diminished in pneumonia if a bronchus is plugged.

Besides the physical signs, other phenomena to be observed are the mode of commencement, the course of the disease, the fever, the expectoration, and the appearance of herpes. The most reliable means of deciding in all doubtful cases is exploratory puncture, although even then we may of course be left in doubt whether an infiltration of the corresponding portion of the lung may not exist, in addition to pleurisy. If the exudation is largely fibrinous, or if there is an abundant new growth of inflammatory connective tissue, the results of aspiration may be negative. Hence, in doubtful cases we should always make repeated trials.

If we have diagnosticated a pleuritic effusion, the next question is always as to the character of the effusion, because the prognosis and treatment are to a large degree dependent upon this. Although certain well-known ætiological circumstances, and the severity of the fever and the general symptoms, often permit us to suspect the nature of the effusion, whether serous or purulent, the only certain information comes from an exploratory puncture with a hypodermic syringe. If the syringe is carefully disinfected, and the operation is cautiously conducted, this procedure is entirely devoid of danger, and we must urgently advise its employment in all doubtful cases in order to settle the diagnosis. It is indeed of practical importance to know that there is a likelihood in empyema of obtaining no fluid by means of exploratory puncture (*vide supra*), even when, upon incision, pus is found. We have had this experience repeatedly. In doubtful and severe cases in which there is suspicion of empyema, it is therefore decidedly advisable, when life is threatened, to make an incision of the pleura. Besides a macroscopic inspection, a careful microscopic examination of the fluid withdrawn is sometimes of importance. Besides the ordinary constituents—red and white blood-corpuscles, endothelial cells, and cholesterine crystals—we may sometimes find something of special diagnostic significance, such as bacteria in septic pleurisy, carcinoma-cells in cancerous pleurisy, etc.

We can not always judge from the beginning whether a pleurisy is tubercular or not; but we should never forget, as has already been affirmed, that in every case of pleurisy, even if it is apparently primary, there is a strong suspicion of tuberculosis. We must observe in particular the general habit and the nutrition of the patient, and inquire into the hereditary predisposition and any previous illnesses. In the further course of the disease persistent hectic fever, slowly increasing emaciation and pallor, fresh relapses, and the onset of pulmonary symptoms, point to the tubercular character of the pleurisy. Every double pleurisy, and every pleurisy associated with pericardial symptoms, leads us most decidedly to suspect tuberculosis. A hæmorrhagic effusion, as we have said, points strongly to tuberculosis. Tubercle bacilli are usually not present in the exudation of tuberculous pleurisy, because the tubercular nodules of the serous membrane scarcely ever ulcerate. On the other hand, we have often been able to demonstrate tubercle bacilli in the scanty expectoration of such patients, even when there were scarcely any pulmonary symptoms. The peculiarities of rheumatic pleurisy have been already detailed.

Prognosis.—The prognosis, as regards the immediate danger of the disease, depends entirely upon the severity of the symptoms, and especially upon the dyspnœa. The prognosis, as regards the further course of the disease, depends chiefly upon the nature of the pleurisy. Many secondary and also many apparently primary pleurisies, although extensive, recover completely and permanently after weeks or months. Unfortunately, we only too frequently have to give a doubtful or an unfavorable prognosis, especially if the tubercular nature of the pleurisy be probable or certain. The prognosis of empyema depends partly upon the underlying disease, but especially upon judicious and timely operative interference. In benign cases of empyema recovery is usually complete in a few weeks after operation, but sometimes months are required. The possibilities of a spontaneous rupture of empyema, internally or externally, have been mentioned above. With incomplete healing, which leaves a pleural fistula, we must fear the appearance of general amyloid disease in various organs.

In rare instances with large effusions sudden death occurs, an event which can not always be satisfactorily explained. Probably there are different factors in different cases, such as pulmonary embolism, cerebral embolism, sudden cerebral anæmia, weakness of the heart, or the onset of pulmonary œdema.

Treatment.—In the beginning of the disease the treatment is purely symptomatic. We try to alleviate the patient's symptoms, the pain and dyspnœa, by local applications, especially by mustard plasters, warm poultices, which are usually more grateful than cold applications, sometimes, too, by dry cups, also by embrocations with chloroform liniment, and, with severe symptoms, by morphine internally or subcutaneously. Unfortunately, we have but few remedies to check the inflammatory process in the pleura. If an ice-bag is well borne, it may be of service. The efficacy of the much-used painting with iodine is doubtful, but it may always be tried if there is a severe pleuritic pain. Perhaps more efficient is the application of iodized vasogen, or of iodoform ointment (1 to 15).

If a large serous effusion has formed, it is the universal custom to prescribe diuretics. The idea is to cause, or at any rate promote, the absorption of the exudation by increasing the excretion of urine. It is, however, very difficult to obtain any real proof of the usefulness of diuretics in pleurisy with effusion. They often are of no use whatever, but again, the absorption of the fluid may follow the administration of a diuretic, so that it does not seem impossible that the latter has a therapeutic influence. With regard to a choice of the diuretics to be employed we would name, first, salicylate of sodium, 75 to 120 grains (grammes 5 to 8) a day, because a specific influence has been ascribed to this drug in pleurisy. This applies particularly to cases of supposed rheumatic origin (*vide supra*). Many good observers assert that primary pleuritic exudations often run a remarkably rapid and favorable course, when treated with salicylate of sodium. Moreover, the drug has a direct diuretic effect. Besides this, the acetates of potassium and of sodium are frequently prescribed, as well as squills, and, of late, diuretine (sodio-salicylate of theobromine), given in doses of thirty grains (grammes 2) two or three times a day. We have sometimes found this remedy apparently very efficient. If there are signs of cardiac weakness and diminished arterial tension, digitalis must be prescribed, alone or with a diuretic. Other internal remedies are at present seldom employed in pleurisy with effusion. The influence of iodide of potassium as an " absorbent " is extremely doubtful. Drastic purgatives and such diaphoretic remedies as hot packs and pilocarpine usually affect the general condition unfavorably, and are of little use. Probably there are today few advocates of the so-called Schroth method of treatment, according to which the amount of fluid ingested is reduced to a minimum, so as to promote the absorption of the exudation. In many cases the operative treatment of pleurisy (introduced by Trousseau)—the evacuation of the effusion by puncture—is of the greatest importance. Many cases of pleurisy with effusion run a favorable course without it, and we consider it at least superfluous to puncture every effusion without sufficient grounds, but puncture is often one of the most serviceable therapeutic influences at our command. The first and most important indication for puncture is present when the effusion becomes so large as to be directly dangerous to life. As soon as the patient's dyspnœa reaches a dangerous degree, and the cyanosis becomes marked and the pulse weaker, a puncture must be made as a direct vital indication. When the exudation is of considerable size there may be a very sudden aggravation of the symptoms, so that in such cases one should not wait too long. It is much better to puncture too early than too late! Trousseau urged that tapping should invariably be performed when the dullness caused by the exudation involves not only the back, but also the whole or nearly all of the anterior wall of the thorax, a rule which the author almost always follows. The benefit of such a puncture is often pronounced. The second indication is a too protracted absorption of the effusion. Puncture is indicated if the effusion does not disappear after an apparent remission of the inflammatory symptoms, especially after the fever has gone. We often see the further absorption stimulated by such a puncture. It has been said that, if possible, tapping should be delayed until the fever has ceased, but to us this does not seem at all necessary. When the exudation was large, or the absorption was delayed, we have repeatedly aspirated even when the patient was still feverish, and have not infrequently found that the fever diminished remarkably upon the removal of the exudation (see the temperature chart, Fig. 37).

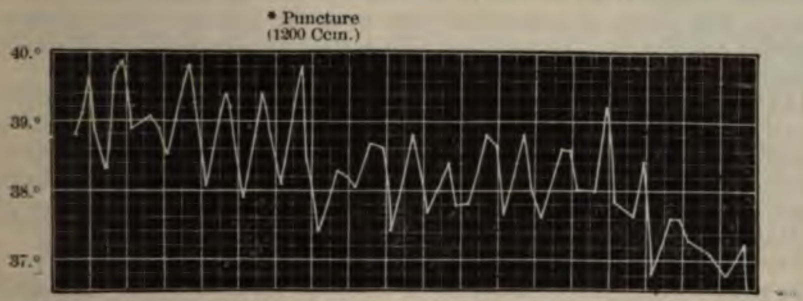


FIG. 37.—Temperature-curve in pleurisy with effusion before and after tapping. (Erlangen medical clinique.)

[There is considerable danger in delaying interference with a large effusion, especially if it has come on pretty rapidly, however comfortable the patient may be. The liability to sudden and fatal dyspnœa under these circumstances is now well recognized.]

As regards the performance of the puncture, we can not here go into all the numerous methods and forms of apparatus proposed. The distinctions are immaterial. The simpler the method, the easier it is to perform, and hence the better it is.

Every puncture must be preceded by an exploratory puncture in order to settle the diagnosis as to the presence and character of the exudation. A medium-sized trocar with a lateral opening, to which a rubber catheter can be fastened, serves to evacuate the fluid. Billroth's and Fräntzel's trocars are useful. Aspiration can be performed still more conveniently with a hollow needle, and best of all, according to our experience, with that proposed by Fiedler, which is now exclusively employed in the author's wards, because the point of this needle is guarded so that it can not scratch the tissues, and also, if it becomes plugged with clots of fibrine, these can be pushed out.* The instruments and the chest-wall at the point of puncture must be carefully disinfected. We usually choose a point for puncture, somewhere about the sixth intercostal space, in the middle or posterior axillary line. The patient sits up in bed, but is held and supported by another person, when it is possible. Before and during the puncture he takes a little strong wine, strophanthus, or similar stimulant. The pain of the operation may be mitigated by previously injecting morphine [or still better, by causing local anæsthesia with a half- to one-per-cent. solution of cocaine]. In the overwhelming majority of cases, and especially if the exudation is abundant, we can remove a large part of the fluid by simple puncture with siphonage. With few exceptions, the pleuritic exudation is under a positive pressure of 10 to 25 millimetres of mercury. The evacuating tube of the trocar must be previously filled with carbolized water and conducted under a layer of the same into the vessel prepared to receive the effusion. The evacuation of the effusion should always be slow and gradual. Many physicians advise stopping when 1,500 cubic centimetres have been removed, but if the fluid is allowed to escape slowly and everything is going well, this quantity may often be exceeded with impunity in the case of large exudations. While, as we have said, in most instances the exudation may be satisfactorily removed by simple puncture and siphonage, it will sometimes prove necessary to employ aspiration. Hence some physicians invariably do so, and to this there is no objection, even if it is unnecessary. The forms of apparatus most used for this are those invented by Dieulafoy and Potain. In puncture with aspiration we proceed more slowly and cautiously.

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* [Fiedler's instrument consists essentially of a sharp hollow needle, with an outer jacket, which can be moved so as to cover the sharp point after the chest-wall has been penetrated. If the lumen is stopped up, a pointed steel wire thrust directly through the attached rubber tubing displaces the obstruction.—TRANS.]

[The necessity for two punctures—one exploratory, the other distinctly operative—does not seem clear. The two can be perfectly combined, a fair-sized trocar or needle being as easy of introduction and producing really no more pain than a very fine one, and being more sure to give results on which reliance can be placed. An ordinary Davidson's syringe makes a very satisfactory pump and can always be obtained.]

Unpleasant incidents which may cause a cessation of the process are rare. If the patient complains of dizziness or faintness we must cease, or at any rate, pause. Usually everything goes well until the needle begins to rub against the pleura: then there is pain and generally a violent cough. It is well, then, to remove the needle at once. The cough can usually be promptly quieted by rest and a little morphine, but sometimes after aspiration there will develop a sort of pulmonary œdema, with the expectoration of a large amount of frothy serous sputum (*expectoration albumineuse*). This is perhaps due to the increased permeability of the vascular wall, or to feebleness of the left ventricle.

When the process is over, we close the little opening with a bit of sticking-plaster or with iodoform collodion. A regular surgical dressing is scarcely ever necessary.

If the exploratory puncture has shown a purulent effusion, we can first evacuate the pus by puncture, if the vital indication exists; but a permanent cure from tapping is exceptional. The pus almost always reappears. Empyema is like an abscess, which can not be cured until a permanent, free exit for the pus has been provided. We must, therefore, not only remove the pus, but institute drainage of the pleural cavity. The best method, and the one which is almost exclusively practiced in the surgical wards in Erlangen, is by incision of the pleural cavity, preceded by resection of a portion of a rib for the purpose of completely emptying out the pus, and obtaining a better final result. This comparatively simple method gives results so good and so entirely satisfactory that, to be frank, we can not understand why there has been so much discussion over the treatment of empyema. We are perfectly willing to allow that good results are likewise obtained with other methods, and in particular with the drainage by siphon action, advocated by Bülau. Yet there is not, in our opinion, any satisfactory ground for abandoning the ordinary surgical principles, relating to the treatment of internal suppuration, in the case of empyema. With regard to the minutiæ of the operation, we must refer to text-books on surgery.

In treating the chronic, contracted pleurisies with thickening, but without fluid effusion, methodical respiratory efforts, "lung-gymnastics," are of use. Besides these we should strengthen the general condition as much as possible. We should advise the patient to breathe deeply, and prescribe cold sponging of the chest daily. Inspiration of compressed air by means of a pneumatic apparatus is often accompanied by good results. Well-to-do patients, who have had a severe pleurisy, should be sent to a suitable climatic health-resort.

CHAPTER II

PERIPLEURITIS

UNDER the name of "peripleuritis" Wunderlich was the first to describe a rare form of disease, which consists of an inflammation of the connective tissue between the costal pleura and the ribs, and which terminates in the formation of an abscess. Similar cases have since been repeatedly observed, and all were characterized by the lack of any discoverable ætiology. There is neither a previous injury, nor a primary disease of the ribs or the pleura. Nevertheless the cause must be sought in an invasion of micrococci, which excite the suppuration. A knowledge of the particulars, however, can only be gained from future investigations. They will determine whether peripleuritis can be regarded as an independent disease or not.

The disease occurs chiefly in men. It usually begins suddenly with a chill, and runs its course with quite a high fever. In pronounced cases the local symptoms have the greatest similarity to those of an empyema, but the greater protrusion of the chest-wall is striking. The ribs are crowded apart by the abscess, and there is often spontaneous rupture externally, scarcely ever into the pleura. Percussion gives no symptoms of displacement of the neighboring organs, a distinguishing point from empyema. It is of diagnostic significance that we can often discover normal lung-tissue containing air below the abscess. The mobility of the lower border of the lung is also usually retained, contrary to what is the case in empyema. Another important sign was first brought to notice by Bartels: the wall of the abscess relaxes on inspiration and becomes tense on expiration. We may also mention that acute nephritis has often been observed among the complications.

From these points we may be able to make the diagnosis during life, at least in many cases. The prognosis is quite unfavorable, but recovery does occur. The treatment can be only operative, and it is quite analogous to that for empyema.

CHAPTER III

PNEUMOTHORAX

(*Pyo-pneumothorax. Hydro-pneumothorax*)

Ætiology.—Pneumothorax—that is, a collection of air or gas in the pleural cavity—arises, in an overwhelming majority of cases, from the penetration of air into the pleural cavity through an opening in the pleura. The opening may be in the external chest-wall from a penetrating wound of the chest or an empyema operation, or it may be in the pulmonary pleura. Pneumothorax is by far most frequently associated with phthisis, when a cavity lying beneath the pulmonary pleura perforates into the pleural cavity. This is more apt to happen in comparatively acute phthisis than in very chronic forms, because the extensive adhesions and contractions in the latter hinder its development. It usually appears in quite far advanced cases, but it may sometimes arise with but slight changes in the lung.

Pulmonary gangrene or abscess, as well as phthisis, may cause pneumothorax by perforation into the pleural cavity. It may also arise from the rupture of an empyema into the lung. In some cases a perforation of the œsophagus or stomach into the pleura, as in gastric ulcer, has been observed, with the formation of pneumothorax.

The development of this condition from severe injuries, as from laceration of the previously healthy lung, without injury to the chest-wall, is rare. Forced respiratory movements, associated with physical exertion, seem especially capable of exciting such a process. We have ourselves seen pneumothorax develop suddenly in a previously healthy woman while hanging out her washing, and another time in a young man during very labored rowing. Both cases recovered rapidly and completely.

All the last-named causes, however, are far less important than pulmonary tuberculosis. We should mention that in phthisis, too, there is sometimes a definite exciting cause—severe coughing, vomiting, or muscular exertion—which may favor the development of the pneumothorax.

Many authors maintain that, by decomposition of a putrid pleuritic effusion, gas may be produced, and thus we may have pneumothorax; but such an event is extremely rare, if it ever happens.

Pathological Anatomy.—On opening the pleural cavity a part of the air usually rushes out, sometimes with an audible noise. We then look into a large cavity filled with air, and find, in total pneumothorax, the lung completely retracted and lying compressed against the vertebral column. If, however, the air fills only a part of the pleural cavity, as a result of extensive adhesions of the pleuræ, we speak of a circumscribed or sacculated pneumothorax. The amount of air contained in the pleural cavity may reach 2,000 cubic centimetres. The pressure which it is under is almost always positive—on an average five or ten centimetres of water.

In the cases of pneumothorax arising from perforation of the pulmonary pleura we can usually make out the point of perforation in the lungs. This is more frequently situated in the upper lobe than in the lower. Sometimes it is already grown over or is covered by a layer of fibrine, and can no longer be found. The opening is usually quite small, but it may reach the size of a ten-cent piece. Left-sided pneumothorax seems to be somewhat more frequent than right-sided.

The pleura itself is only rarely normal. Usually agents of inflammation have entered it with the air, and hence it is found in a state of inflammation. A part of the cavity is then filled with effusion. This is usually wholly purulent—pyo-pneumothorax—or sero-purulent, but it may even be serous or sero-fibrinous—sero-pneumothorax, or hydro-pneumothorax.

The neighboring organs, especially the heart and liver, are found pushed out of their normal position, as in large pleuritic effusions.

Symptoms and Course.—The onset of pneumothorax (we speak in what follows especially of pneumothorax in connection with pulmonary tuberculosis) is quite often made known by a sudden pain, usually associated with an increase of the dyspnœa and of the general symptoms. There is sometimes collapse. The temperature sinks below normal, the pulse rises to 140 and over. The patient looks pale and cyanotic. He usually sits upright or is in a half-sitting position in bed, either more on the affected side, in order to use the normal lung as much as possible for breathing, or more on the sound side on account of the tenderness. If the pneumothorax has come on as a result of the rupture of an empyema into the lungs, there is at the same time a very abundant expectoration of pus.

Although in many cases the symptoms mentioned lead to a suspicion of pneumothorax, yet a positive diagnosis can be made only after a physical examination.

Inspection gives a very marked distention of the affected side. The intercostal spaces are stretched out, or even protruded. In some cases, as we have ourselves noticed, there is a marked elastic "air-cushion feeling" on palpating the intercostal spaces. On respiration, the affected side is almost entirely motionless, while the excursions of the other side are the more marked. The displacement of the heart is often evident from the visible displacement of the apex-beat.

Percussion gives over the pneumothorax a remarkably loud, full note, abnormally deep, but usually not tympanitic, on account of the tension of the walls. It is especially important to note that this resonance extends beyond the normal limits of the lung on the right down to the seventh or eighth rib, and on the left to the fifth or sixth rib, and sometimes even to the edge of the thorax. The lower limit of this abnormally loud percussion resonance shows no change of position with respiration.

The displacement of the neighboring organs can also be made out by percussion. With right-sided pneumothorax we find the lower border of the liver dullness abnormally low, and the left border of the cardiac dullness pushed over to the anterior axillary line. In left-sided pneumothorax the cardiac dullness is usually entirely absent from its normal place, and is found instead to the right of the sternum. The left lobe of the liver is pressed downward, and in the "semilunar space" we find, instead of the normal tympanitic resonance, the same deep, loud, and usually non-tympanitic note as in the upper parts of the thorax.

Upon auscultation we are struck by the entire absence of respiratory murmur. This is in special contrast to the clear resonance on percussion. In other cases, however, we hear a number of metallic sounds, at least in many places and at many times, which are very characteristic of pneumothorax. First among these is amphoric, metallic respiration. This arises in open pneumothorax (*vide infra*) from the direct passage of the air in and out, but in all other cases it is the ordinary respiratory murmur, which has acquired a metallic timbre from resonance in the pneumothorax. In an analogous way arise the metallic-sounding râles ["metallic tinkling"], and the metallic resonance of the cough and voice. Heubner has devised a particularly beautiful and practically important method for demonstrating the metallic sound in pneumothorax. If we strike lightly on a pleximeter with a little rod, usually the handle of a percussion hammer, while we auscult near it—"rod percussion"—we very often hear quite a distinct high metallic sound.

The vocal fremitus over a pneumothorax is usually diminished, but it may be felt in spite of quite a large collection of air.

A number of special physical signs are found if a purulent or serous effusion be added to the pneumothorax. In the first place, the resonance is thereby rendered dull, to a greater or less extent, in the lower parts of the chest. The boundaries of the fluid by percussion show a very evident change with the patient's change of position, because the fluid in pneumothorax can move easily in all directions. Inasmuch as the shape of the remaining air space must change correspondingly, there is not infrequently a change in the pitch of any metallic sounds which may be produced, whether by percussion or respiration, varying with the posture of the patient (Biermer's change of note). In many cases, if the ear is applied to the chest-wall there is heard, on every motion of the fluid, produced, e. g., by gently shaking the patient, a metallic gurgling sound, so-called Hippocratic succussion.

Forms of Pneumothorax.—According to the condition of the perforation during life, we distinguish three kinds of pneumothorax (Weil). We speak of an "open pneumothorax," if the point of perforation remains open, so that the air on respiration constantly passes in and out of the pleural cavity. If the perforation is completely closed, we have a "closed pneumothorax." The third and most frequent form is the "valvular pneumothorax," in which air enters the pleural cavity at each inspiration, but on expiration there is a valve-like closure of the perforation, and thus the air can not escape again; but as soon as the pressure in the pleural cavity increases so that no more air can enter it on inspiration, the valvular pneumothorax becomes closed. In open pneumothorax the pressure in the pleural cavity must be the same as the atmospheric pressure. A positive pressure in the pleural cavity can exist only in a closed or a valvular pneumothorax.

A clinical diagnosis of the form of pneumothorax is not always possible, and has usually no great practical importance. The very loud, metallic, amphoric respiratory murmur, which may be heard in open pneumothorax, must be mentioned, and Wintrich's change of pitch (see page 243) can sometimes be heard in this form. It is worthy of mention that symptoms of displacement of the neighboring organs must also arise in open pneumothorax. The predominant atmospheric pressure here is positive in contrast to the negative pressure in the other pleural cavity, and to the normal negative pressure which previously acted on the upper surface of the diaphragm. A very marked protrusion of the affected side, and great displacement of the heart and liver, however, speak most strongly against an open pneumothorax. Some authors have tried to find a point of distinction for the different forms of pneumothorax in the composition of the gas in the pleural cavity, but the results of chemical analysis are still contradictory. According to Ewald, we find in open pneumothorax not over five per cent. of carbonic acid and about twelve to eighteen per cent. of oxygen; in closed pneumothorax, however, fifteen to twenty per cent. of carbonic acid and ten per cent. at most of oxygen. If in an open pyo-pneumothorax or hydro-pneumothorax the point of perforation lies below the level of the fluid, there sometimes arise on every inspiration metallic sounds, since the bubbles of air drawn in rise and come up through the fluid—" the water-pipe sound," " metallic tinkling." A peculiar sipping and short snapping sound on inspiration, heard by us in one case, seems to point directly to the existence of a valvular pneumothorax.

Course of the Disease.—In many cases the occurrence of pneumothorax causes such a high degree of respiratory disturbance that death ensues in a few hours or days. In other cases the patient improves, and may feel quite well for a long time in spite of the condition. We have ourselves frequently observed patients who passed the entire day out of bed with scarcely any inconvenience. Usually, however, the disease which gives rise to the pneumothorax, most often pulmonary tuberculosis, leads in a shorter or longer time to death. Sometimes, again, the patient recovers. This almost always occurs in those rare cases above mentioned, when a healthy lung has been torn, but sometimes, also, in cases of pulmonary tuberculosis. The healing takes place in this way, that the air is first replaced by a fluid effusion, and then the latter is gradually absorbed, but the air may also be directly absorbed in whole or in part. It depends upon the origin of the lesion, then, and upon the intensity of the underlying disease, whether the recovery is permanent or not.

Diagnosis.—The diagnosis of pneumothorax is usually easy with careful examination, but the symptoms may sometimes be of so little prominence as to excuse overlooking it. It is very difficult and often quite impossible to make a differential diagnosis between very large cavities and a saccular pneumothorax, since both conditions must have in part precisely the same symptoms. We may mention as the chief points in distinction: A cavity is apt to be situated in the apex, pneumothorax in the lower part of the thorax; over a cavity the chest-wall is often sunken in, over pneumothorax it is prominent; the vocal fremitus is usually marked over a cavity, weak over pneumothorax. Signs of the displacement of organs point to pneumothorax, as does distinct succussion, while a metallic respiratory sound and metallic resonance on rod percussion may also be heard over large cavities with smooth walls. Besides the above signs, we should also carefully regard the general course of the disease. On careful questioning we shall almost always find that the severe symptoms depending upon the development of a pneumothorax, set in with more or less suddenness.

Treatment.—Whenever pneumothorax has developed we must seek to obtain perfect quiet for the patient, as well as diminution of his sufferings, by means of morphine given subcutaneously or internally. The results of the administration of this drug, in a sufficient though careful dosage, is often very satisfactory. We must also stimulate cardiac activity, if it is impaired, by means of strophanthus or camphor. It is exceptional to find operative interference necessary because of the danger to life in the first days of the attack; and even later on we should usually wait to see how much spontaneous improvement of the condition may take place. If the symptoms remain severe and the displacement of organs is great, we should try to remove by aspiration as much air as possible from the pleural cavity. Likewise if there is an abundant exudation it is always useful to puncture and remove the fluid, repeating the operation if necessary. If the exudation is purulent, the treatment is precisely the same as for empyema, except that in hopeless cases of far-advanced tuberculosis we may limit our efforts to purely symptomatic treatment, or to the simple evacuation of the pus by puncture.

CHAPTER IV

HYDROTHORAX. HÆMATOTHORAX

1. **Hydrothorax.**—We term the occurrence of a serous transudation into the pleural cavity, independent of an inflammation of the pleura, hydrothorax, or thoracic dropsy. The cause of hydrothorax is in rare cases a local hindrance to the outflow of venous blood or lymph from the thorax, as in compression of the veins or of the thoracic duct by tumors; but in the great majority of cases the hydrothorax is part of a general dropsy, occurring especially in cardiac or renal disease. Hydrothorax is often first developed after marked œdema of the subcutaneous cellular tissue and ascites, but it may sometimes be one of the first symptoms of dropsy, particularly in renal cases. It is usually bilateral, but it is often unilateral, or at least much greater on one side than on the other. The pleura itself is normal or else œdematous. We often find it traversed with a network of dilated lymphatics. The serous fluid in hydrothorax is distinguished from an inflammatory serous effusion by the smaller amount of albumen in it, and a correspondingly low specific gravity (usually below 1018), by the scanty number of cell-elements, and by the absence of or the slight tendency to spontaneous coagulation.

The clinical importance of hydrothorax lies in the hindrance to respiration which it causes. As a result of this the hydrothorax may be regarded in many cases, especially in renal disease, as the chief cause of death. The objective evidence of it comes from the physical examination, which must, in general, give dullness, diminished vocal resonance, and dislocation of neighboring organs, the same as in pleuritic effusion. We desire to emphasize also the bronchial respiration from compression in hydrothorax, which is often very loud, although at the same time usually high and sharp, and which may even given rise to a confusion with pneumonic infiltration in the lungs. This very loud respiratory murmur, contrasting with that of pleuritic effusion, is explained by the normal condition of the lungs and the absence of all adhesions. For the same reason, too, the change in the boundary of the dullness, as a result of the patient's change of position, is usually more marked in hydrothorax than in pleuritic effusion. We often hear a few fine crepitant râles over the hydrothorax, which arise in the retracted and partly atelectatic lung. The main factor, however, in distinguishing hydrothorax from a pleuritic exudation is, in every case, the consideration of the primary disease. It should also be noticed that hydrothorax is usually bilateral, while pleuritic exudation is usually unilateral.

Treatment is directed, first of all, to the primary disease. If we succeed in strengthening and regulating the action of the heart, or in restoring the secretion of urine, the hydrothorax often disappears with the other dropsical symptoms. If the dyspnœa caused by it reaches a dangerous degree, we often see great relief from aspirating the fluid. The nature of the underlying condition, of course, renders the benefit in many cases only transitory.

2. **Hæmatothorax.**—Effusions of blood into the pleural cavity (hæmatothorax) arise most frequently from traumatic lacerations of blood-vessels, rarely from the bursting of an aneurism of the aorta into the pleural cavity, from erosion of an intercostal artery in caries of the ribs, from the rupture of a cavity into the pleura in phthisis, if it simultaneously opens a blood-vessel, etc. In many such cases a typical exudative pleurisy follows the effusion of blood. The physical signs are the same as in other pleural effusions. Severe dyspnœa may demand the removal of the blood by puncture, or even by an incision.

CHAPTER V

NEW GROWTHS OF THE PLEURA

The majority of new growths occurring in the pleura are of a secondary nature. We sometimes find single metastatic nodules of cancer in the pleura after primary carcinoma of other organs, especially of the mammary gland and the lungs, but most carcinomata of the pleura arise from primary carcinomata of the lungs and are due to a direct invasion of the pleura by the new growth.

Of the primary new growths in the pleura, only one is of great importance—the endothelial carcinoma, first described by E. Wagner. This develops *de novo,* in a diffuse manner, from a proliferation of the endothelial cells of the lymphatics and the connective tissue. Metastases occur in the lungs, in the lymph-glands, in the liver, in the muscles, etc.

Single secondary nodules of cancer in the pleura cause no special clinical symptoms, but the cases of diffuse cancer of the pleura as a result of primary cancer of the lungs are important, inasmuch as the symptoms of disease of the pleura often quite predominate over the pulmonary disease. The dullness is intense, the respiratory murmur and the vocal fremitus diminished. In one such case we saw a proliferation of the cancer upon the ribs in front so that there was externally a very marked circumscribed swelling. The character of the sputum is the only thing that can give us definite information as to the origin of the new growth in the lungs (see the chapter on cancer of the lungs).

Primary endothelial carcinoma of the pleura runs a course similar to chronic pleurisy. As we sometimes find a co-existing fluid effusion in the pleural cavity, displacement of the neighboring organs may occur. The affection goes on for a long time without fever, or with slight and irregular elevations of temperature. Most cancers of the pleura are associated with severe pain.

The diagnosis of new growths in the pleura can usually be made, if at all, only in the more advanced stages of the disease. At first almost all the cases are regarded as simple or tubercular chronic pleurisy. The diagnosis is founded less upon the physical signs than upon the general course of the disease, the habit of the patient, and the evidence of metastases in the glands and other organs. In some cases characteristic elements of the new growth can be found by the microscope in the cloudy fluid obtained by an exploratory puncture.

The prognosis is absolutely unfavorable, the treatment purely symptomatic. In endothelial carcinoma we might perhaps try the long-continued use of arsenic.

CHAPTER VI

MEDIASTINAL TUMORS

In the anterior mediastinum, in quite rare cases, extensive new growths occur, which are of importance on account of their severe clinical symptoms. The point of origin for the tumor is either the mediastinal lymph-glands, or the connective tissue, or sometimes the remains of the thymus gland. In their anatomical character the tumors are almost always sarcomata, usually lympho-sarcoma, rarely alveolar sarcoma. They usually occur in youth or middle age, and are somewhat more frequent in men than in women. The special ætiological factors are unknown. In some cases an injury is stated to be the cause of their development.

The first clinical symptoms are usually of a very indefinite nature. The patient complains of general languor, headache, pain in the chest, and slight difficulty in breathing, and only gradually do severe subjective and objective symptoms develop in the chest.

The symptoms are in part due directly to the tumor, but in larger part they are symptoms of compression from the gradually increasing pressure of the tumor on a number of neighboring organs.

The pain in the chest, which is located chiefly in the sternal region, and is associated with a marked feeling of oppression, may be very severe. It sometimes shoots into the lateral portions of the chest and into the arms, showing pressure on the brachial plexus.

The dyspnœa may finally increase to an extreme degree. A patient with lympho-sarcoma under our observation could, in the last days of her life, breathe only while standing. The dyspnœa is due to a compression of the heart and lungs, and sometimes to actual stenosis of the trachea or a primary bronchus. Paralysis of the dilators of the glottis may also occur from a pressure paralysis of the recurrent nerves. Paralysis of one vocal cord has been repeatedly observed. In the case mentioned above a marked goître developed, as a result of vascular stasis, which further increased the dyspnœa by pressure on the trachea. A hydrothorax from local venous stasis may also aid in increasing the dyspnœa.

Pressure on the œsophagus, and disturbances of deglutition due to it, are rare. Pressure on the vagus nerve and the sympathetic sometimes causes anomalies in the rate of the pulse—either marked acceleration or slowing of the pulse. If the sympathetic is involved there is inequality of the pupils. In some cases, by pressing on the tumor, an artificial dilatation of the pupil can be excited at will. By pressure on the vessels, especially on the superior vena cava, the subclavian vein, etc., œdema and cyanosis may arise in the corresponding parts of the body.

Objective examination of the chest gives a marked diffuse prominence of the sternal region in a part of the advanced cases; in other cases this swelling is absent. The discovery of an abnormal dullness in the anterior part of the chest is of diagnostic importance; this usually joins the cardiac dullness on the left, and on the right it extends a varying distance beyond the right border of the sternum. The heart is often pushed somewhat to the left. We heard over the pulmonary artery in our case a marked systolic murmur, caused by compression of the vessel. A dissimilarity of the pulse on the two sides is not infrequent.

The diagnosis of a mediastinal tumor is usually possible in cases with well-marked symptoms, but in other cases it is difficult and uncertain. The differential diagnosis between mediastinal tumors and aneurism of the aorta (*q. v.*) causes especially great difficulty. Tumors may also be confounded with abscesses in the anterior mediastinum.

The prognosis is in all cases absolutely unfavorable. The disease terminates fatally, sometimes after a duration of six months or a year.

The treatment can be merely symptomatic. Internally we may try iodide of potassium or arsenic. In the last stages of the disease we must try to alleviate the patient's great distress by narcotics.

CHAPTER VII

ACTINOMYCOSIS OF THE THORACIC CAVITY

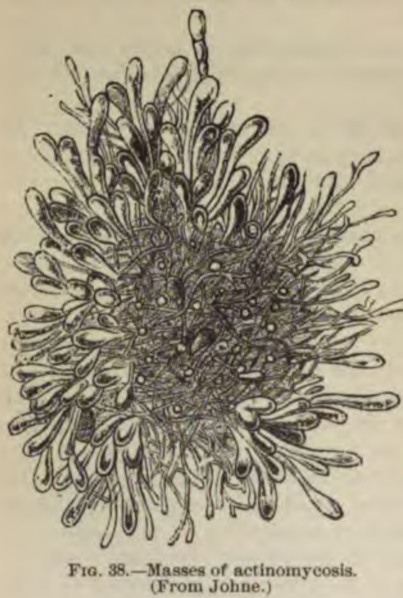

FIG. 38.—Masses of actinomycosis. (From Johne.)

BOLLINGER and others have described a peculiar tumor affecting the jaw-bones of cattle, and occasioned by the presence of a special form of fungus, known as the actinomyces or ray-fungus. More recently a class of diseases has been studied in human beings, occasioned by the same fungus (Ponfick, Israel, and others). These diseases may, as in cattle, affect the jaws, the floor of the mouth, and the neck; but in these cases they are mainly of surgical interest. The actinomycotic diseases of the internal organs, however, possess a great clinical importance; and, inasmuch as the lungs and pleura are the most frequently affected organs, it will be well to present briefly here the most important facts which have as yet been learned with regard to actinomycosis. The botanical position of actinomyces has not yet been definitely settled. Cohn and O. Israel regard it as a mould fungus. Boström, on the other hand, classes it with the algæ, and namely with the variety cladothrix. In its growth, the fungus forms small or moderate-sized gray or sulphur-yellow nodules which may be distinguished with the naked eye in the pus of the diseased tissue (see below), and which upon microscopic examination resolve themselves into a tangle of mycelium. It is an especial characteristic that many of these mycelia bear on their ends a club-shaped swelling. These are placed for the most part like radii on the periphery of a nodule, and so surround the entire mass like a circlet of rays (see Fig. 38). In nature actinomyces seems to appear especially upon plants, for example upon the beard of spikes of wheat. Thus is explained the frequency of infection in the plant-eating cattle, and a similar direct infection seems occasionally to be possible in man. It is worthy of note that the fungus seems to locate itself often in carious teeth. Thus apparently arises the above-mentioned disease in the buccal cavity; while on the other hand, the fungus may be carried from its nidus in the mouth by inspiration into the respiratory tract, or by swallowing into the *primæ viæ*. Of course it may also be directly swallowed or inhaled.

Wherever the fungus fastens itself in the body it occasions first a new growth of granulation-tissue, which has a tendency to break down into a whitish or brownish pasty mass. The brown color is occasioned by the hæmorrhage which frequently occurs. Very often actinomycosis goes on to suppuration; still, this probably depends upon the influence of pathogenic germs which have secondarily infected the part. Of especial importance is the tendency of the disease to extend from the lungs to the pleura and from the pleura to the peripleuritic connective tissue, and still further to the wall of the thorax. Thus arise not only extensive abscesses and wide-branching fistulous tracts, but also a very characteristic, extremely tough cicatricial infiltration of the affected part. Not infrequently there is at last a perforation reaching the outer surface of the body.

The entire process, as a rule, is slow and insidious, but constantly progressive. The symptoms consist at first in slight thoracic discomfort, pain, cough, and expectoration. Physical examination will often detect changes in the lungs, but the correct interpretation of the signs found is of course at first difficult, if not impossible. The more the disease spreads the greater is the distress. Usually there is hectic fever, which may assume a pyæmic character if there is extensive suppuration. The patient gradually loses flesh, and in repeated instances amyloid degeneration of the liver, spleen, and kidneys has been observed. If a focus breaks into a pulmonary vein, the disease may be developed by metastasis in other internal viscera. Moreover, there may be a direct extension of the disease to the pericardium, or through the diaphragm into the peritoneal cavity.

The diagnosis of actinomycosis is at first difficult. It is established when the characteristic fungus is found in the sputum, but this has occurred in only a few cases as yet. If very extensive peripleuritic and pericostal suppuration has taken place, and the process has spontaneously broken outward or been laid open by surgical means, the demonstration above described in reference to the fungus is easy.

The treatment can be only symptomatic, unless the diseased spot can be reached by operation, and then the treatment becomes surgical. Permanent cure has so far been attained in but rare instances. [Iodide of potassium has proved curative in a considerable number of cases, and it should certainly be tried, in doses of forty to sixty grains a day.—V.]

DISEASES OF THE CIRCULATORY ORGANS

SECTION I

Diseases of the Heart

CHAPTER I

ACUTE ENDOCARDITIS

(*Endocarditis verrucosa. Endocarditis ulcerosa*)

Ætiology.—Organized excitants of inflammation of different sorts, which circulate in the blood, may settle on the endocardium, especially on the valves of the heart, and there give rise to an acute endocarditis. Endocarditis, therefore, in its ætiological relations, is not to be regarded as a single disease. Pathogenic micro-organisms have been injected into the blood (the streptococcus pyogenes, staphylococcus aureus, and others), and in this way an artificial endocarditis has been set up in animals. The experiments are more apt to succeed if the valves or the inner coat of the vessels have been subjected to some slight injury before the injection, thus promoting the settling of the germs upon them (Orth and Wyssokowitsch, Ribbert). Most cases of endocarditis in man, both the vegetative and ulcerative forms, appear to be caused by the staphylococcus pyogenes aureus. Streptococci are much less frequent than staphylococci, and it is only in rare cases that endocarditis is occasioned by the pneumococcus, gonococcus, and perhaps the diphtheria bacillus.

In accord with these statements is the fact that in human beings acute articular rheumatism, which is probably to be regarded as a staphylococcus infection, is that one of the infectious diseases which is especially apt to have acute endocarditis as a frequent and important complication. The staphylococcus infection need not appear, however, in the first place, as a typical acute polyarthritis. An acute endocarditis may appear as a sequel of many cases of apparently primary pleurisy, tonsillitis, or other disease. Indeed, it is sometimes impossible to determine the gate of entry of the infection, and the whole disease takes the shape of an apparently primary endocarditis. In such cases the nature of the disease is made evident by the later appearance of multiple articular swellings. We also regard as allied to polyarthritis certain forms of infection called the "acute hæmorrhagic diseases" (e. g., purpura rheumatica), and also chorea (which see). It is therefore not surprising that acute endocarditis is not infrequently observed in association with these affections.

There are other acute infectious diseases with which acute endocarditis may be associated. Probably in these it is usually the result of a secondary mixed infection, generally with the staphylococcus. This explains the appearance of acute endocarditis in scarlet fever, small-pox, diphtheria, measles, and typhoid. Not infrequently mild cases of acute endocarditis, without clinical significance, are found in association with primary tuberculosis and ulcerating carcinoma. Again, acute and chronic nephritis may sometimes occasion a development of endocarditis. An especially important rôle in producing endocarditis is displayed by grave septic and pyæmic conditions (*vide supra*). In these cases the acute endocarditis appears as one symptom, but it is often so prominent that the whole attack is called endocarditis (*vide infra*). In such severe diseases we have often to do with a streptococcus disease, although it may be a staphylococcus infection of especial virulence. The ætiology of gonorrhœal endocarditis is not yet fully determined. Endocarditis as a sequel of gonorrhœa is fortunately very rare. It often seems to be an actual gonococcus infection, but in other instances is occasioned by some secondary germ.

Finally, we have still to mention the important fact that acute endocarditis quite frequently develops on the soil of an already existing chronic endocarditis—the so-called acute recurrent endocarditis. In women, pregnancy and the puerperal state sometimes seem to give the occasion for a recrudescence of the endocarditis.

Pathological Anatomy.—We usually distinguish an *endocarditis verrucosa*, with the formation of large or small papillary nodules on the endocardium, and an *endocarditis ulcerosa* (*endocarditis diphtheritica*), with ulcerations as a result of the destruction and wasting away of the superficially necrosed tissue. The malignant, invariably fatal form of severe septic endocarditis is chiefly ulcerative endocarditis. Endocarditis verrucosa is the milder form, which is seen especially in acute polyarthritis and allied diseases, but we can not draw either a sharp anatomical or a sharp clinical distinction between the two, since malignant cases of endocarditis verrucosa are also observed.

The endocardial growths are usually situated on the valves, especially on their edges of closure. More rarely we find them on the chordæ tendineæ and on the endocardium of the ventricle and auricle. In the mildest cases they are scarcely as large as the head of a pin, but in severe cases they may increase to quite large warty and glandular masses. Microscopically, the base of the nodule consists of a newly formed vascular tissue, infiltrated with small cells, which on its surface changes to a granular, coagulated mass. This last is formed partly of coagulated albumen, dead cells, and fibrine deposited from the blood, and partly of micrococci (*vide supra*). The micrococci are found without exception in all cases of ulcerative endocarditis—having been first discovered by Eberth. The endocardial ulcers arise from the destruction of the superficially necrosed nodules. If the thin valve in any place yields to the blood-pressure, we have the so-called acute valvular aneurism. Complete perforation of a valve, and tearing off of fragments of a valve and of the chordæ tendineæ, are also seen.

The great majority of cases of acute endocarditis are confined to the valves of the left side of the heart—the mitral and aortic valves. Endocarditis on the tricuspid valve is seldom seen except as a secondary affection in old cases of heart disease. In a case of acute ulcerative endocarditis in a grown man seen by us, the process was confined exclusively to the tricuspid valve, and there were very many embolic abscesses in the lungs. This may be considered a great rarity. In contrast to the ordinary localization of endocarditis we find fœtal endocarditis most frequently in the right side of the heart.

Many other organs may be affected by the endocarditis, through embolism. In the benign endocarditis verrucosa, the masses of fibrine deposited on the irregularities of the valve furnish the embolic material. They cause large or small infarctions in the kidneys and spleen, embolic softening of the brain, etc. In the malignant, ulcerative forms, however, large numbers of bacteria get into the circulation at the same time with the necrotic masses of tissue which have been torn off. Here, then, we have to do not merely with simple mechanical obstruction but with infectious emboli. The emboli in ulcerative endocarditis, therefore, either give rise to embolic abscesses in the cardiac muscles, the kidneys, the spleen, the lungs, the retina, etc., or they result in hæmorrhages, especially into the skin, but also into the kidneys, the brain, the retina, and the serous membranes. It is not yet known why in some cases abscesses are more frequent and in others hæmorrhages. The two, however, may be combined. In general, we may suppose that the development of abscesses is everywhere connected with the presence of bacteria, while hæmorrhages may also arise from toxic influences; but again changes in the vascular walls caused by bacteria might give rise to hæmorrhages. Embolic abscesses belong almost exclusively to the severe form of septic endocarditis. Hæmorrhages are seen in this form, and also—without co-existing abscesses—in certain severe forms of endocarditis occurring in the course of acute rheumatism and allied diseases.

Clinical History.—Since acute endocarditis is not ætiologically a distinct disease, and since its clinical course is very different in different cases, it seems advisable to us to describe, in what follows, the most important varieties separately; but it must be expressly noted that the separate classes can by no means be sharply defined, and that there are many intermediate forms.

1. *Slight endocarditis verrucosa* is quite frequently found in the cadaver, without the slightest signs of any affection of the heart during life. The little papillary excrescences on the valves of the heart in phthisis, and carcinoma, whose ætiology has been explained above, are to be classed under this head.

2. The typical form of *benign acute endocarditis* is most frequent, clinically, in the course of acute articular rheumatism. It is much rarer in other infectious diseases (*vide supra*). In a few cases it is also seen as an apparently primary disease. Here there is a constitutional rheumatic infection which has taken place in some way, and which settles directly on the valves of the heart instead of in the joints (so-called primary rheumatic endocarditis). Careful questioning will sometimes reveal, or at least render probable, the place of infection (a mild case of tonsillitis or some slight external injury). Very often there will be later the symptoms of articular rheumatism. It is seldom that endocarditis as such is associated with special local disturbances, such as pain in the region of the heart, palpitation, and dyspnœa. Ordinarily the heart disease is discovered only by physical examination. The impulse of the heart in many cases is abnormally strong and diffuse; the pulse is accelerated, but strong, often somewhat jerky (*pulsus celer*), and usually regular, but sometimes a little irregular. Percussion at first shows no deviations from the limits of normal dullness. On auscultation, we hear at the apex, more rarely at the base, a loud blowing, systolic souffle. Diastolic murmurs are rare in acute endocarditis. The pulmonic second sound is often accentuated. The physical signs referable to the heart are only slightly marked in many cases of acute endocarditis. This is understood if we remember that the occurrence of a heart-murmur depends wholly on the localization of the endocarditis, on the development of some valvular insufficiency, etc.

Besides the direct symptoms pointing to the cardiac affection, the onset of an acute endocarditis is often, but not always, associated with fever, or, if fever be already present, with an increase of it, and with a slight aggravation of the general disturbance. Embolic processes may occur in the brain, the spleen, the kidneys, and the extremities, but they are comparatively rare. Sometimes a pericarditis develops as a result of the endocarditis (*vide infra*).

It is hard to make any accurate statements as to the duration of this form of endocarditis. The physical signs may last for days, or for several weeks. Complete recovery is possible, but in the majority of cases this variety passes into chronic valvular disease of the heart.

3. *Malignant, non-septic form of acute endocarditis* ("rheumatoid endocarditis" of Litten). In many cases this form is perhaps only a quantitative increase of the preceding form, but in other cases it is probably distinct from it

ætiologically. The severe general infection is usually quite prominent here, and the disease resembles in many particulars grave septic endocarditis. The objective signs in the heart are the same as in the preceding form, but more intense and extensive. The subjective cardiac symptoms, such as palpitation and distress, may be quite pronounced, but they may also be almost wholly absent in this form. The general condition, however, is usually bad. There is sometimes high fever with an irregular or intermitting course, but in many cases the fever is remarkably low in spite of quite severe constitutional symptoms.

The constitutional infection is very often manifested in these cases by the appearance of small or large hæmorrhages in the skin, sometimes in the mucous membranes, as in the conjunctiva and the soft palate, and rarely in the retina. Secondary articular swellings often develop; they are always of a serous character, and never purulent. Renal hæmorrhages and acute hæmorrhagic nephritis are quite frequent. Large emboli may also occur in the different organs in this as in every other form of endocarditis.

The duration of the disease extends over many weeks. In severe cases death ensues with a gradual aggravation of the general condition, and often with severe cerebral symptoms, such as stupor and delirium. In milder cases, however, the patient may finally get well.

Regarding the occurrence of this form, we see it most frequently in acute articular rehumatism; also, in rare cases, in gonorrhœa, when it comes on some three or four weeks after the beginning of the urethral affection; also in nephritis, chorea, peliosis rheumatica, etc. The apparently primary cases of this sort usually belong to the recurrent form of acute endocarditis.

4. *The recurrent form of acute endocarditis* consists of an acute increase of the endocardial process, brought on by some exciting cause, in a patient already suffering from chronic endocarditis. The acute disease may show all the gradations from the mildest to the severest. The mild cases often run their course without any special symptoms. To this form we may frequently refer the more or less temporary elevations of temperature which we often see in patients with chronic valvular disease of the heart. In rarer cases the recurrent endocarditis comes on quite suddenly in the form of a severe acute attack. This sometimes seems to be clinically a primary, independent disease, especially if the previous chronic heart disease has up to that time caused no special symptoms. The patient has general malaise, headache, chills, and fever. The last may be quite high—104° (40° C.) and over—or moderate, varying between 100° and 102° (38°-39° C.), or it may be entirely absent. In many cases it is intermittent, when the return of fever is often associated with a chill. The cardiac symptoms may be quite pronounced, but in this form, too, they may be obscure and indefinite. In the further course of the disease we meet with cutaneous hæmorrhages, retinal hæmorrhages, articular swellings, large renal hæmorrhages, or typical hæmorrhagic nephritis—in short, just the same general type of disease as in the other malignant forms of acute endocarditis. The course is rarely rapid, and often lasts for weeks. Severe cases almost always end fatally.

5. The *severe septic ulcerative endocarditis* has already been described as a complication of a general septic state. We therefore refer to the appropriate chapter (see page 116) for all particulars. Septic endocarditis is probably entirely distinct ætiologically from the forms thus far described, and is manifested by quite a rapid fatal course, with severe typhoidal or pyæmic symptoms. It is characterized anatomically, apart from the cardiac affection, by the appearance of metastatic abscesses in the various organs, but in many cases, as we have said, abscesses and hæmorrhages are combined.

Diagnosis.—The diagnosis of an endocarditis, coming on secondarily in the course of articular rheumatism and other diseases, can be made only by a physical examination of the heart. We must therefore give constant attention to the condition of the heart in diseases which we know may give rise to endocarditis.

The diagnosis of the malignant form of endocarditis often causes great difficulty, especially if the patient is not seen until the later stages. It is confused with typhoid, meningitis, or acute miliary tuberculosis. Examination of the heart may furnish positive evidence, but sometimes, as we have said, there are no physical signs of cardiac disturbance, or the signs present are indefinite. Of other factors in diagnosis a special importance attaches to the secondary swelling of the joints, and also to the hæmorrhages into the skin and retina, for these are much more infrequent in the diseases with which this form of endocarditis may be confounded. The acute hæmorrhagic nephritis, too, in connection with the other symptoms, is, at least to a certain degree, characteristic of malignant endocarditis. The course of the fever is of diagnostic value only when it is decidedly intermittent. A careful search for some ætiological factor is very important for diagnosis in all cases. For other points the reader is referred to the consideration of septic diseases (page 116).

Prognosis.—In the description of the course of the disease we have already mentioned the prognosis of the different forms. The severe cases of acute endocarditis usually, and the cases of severe septic endocarditis always end fatally. Here, however, the cause of death is to be sought rather in the accompanying systemic infection than in the endocarditis itself. In mild cases recovery is possible, but the process of repair is often so incomplete that chronic valvular disease of the heart develops from the acute endocarditis.

Treatment.—The chief requisite in the treatment of every endocarditis is as complete rest as possible for the patient. If ice is well borne, the continuous application of an ice-bag to the cardiac region is of service. If there are signs of cardiac weakness, such as a small, rapid, and irregular pulse, we must employ cardiac stimulants, strophanthus, camphor, and, above all, digitalis. It must be confessed that the effect of these is not very great. If there are marked subjective symptoms (such as dyspnœa) narcotics, particularly morphine, are indispensable. Effort is made to combat the systemic infection by salicylic acid, salicylate of sodium, and similar remedies, including phenacetine and salipyrine. Quinine is usually entirely without effect, even when the fever is of an intermittent character. Arsenic, however, seems to be useful, particularly in the more protracted cases.

[For remarks upon the alkaline treatment of rheumatism, see page 686.]

CHAPTER II

VALVULAR DISEASE OF THE HEART

(Chronic Endocarditis)

Ætiology.—A large number of cases of chronic valvular disease of the heart proceed from acute endocarditis. Hence the frequent statement in the history of chronic valvular disease that the patient has had articular rheumatism, once or many times. In a collection of 163 cases of undoubted valvular heart disease, we were able to ascribe 86 to a previous attack of acute articular rheumatism. Following the acute valvular endocarditis, which is often associated with this disease, marked thickening of the valves occurs, due to the growth of connective tissue. There are also changes in the way of contraction, adhesion, and finally often of considerable calcification. The unavoidable result of all these processes is that the altered valves are rendered incapable of fulfilling their physiological function of regulating the circulation. Inasmuch as the mitral valve is most often attacked by endocarditis when associated with acute articular rheumatism, we find mitral disease predominating among rheumatic valvular troubles; but lesions of the aortic valves, rheumatic in their origin, are by no means rare.

If we find a valvular lesion in a patient who has never suffered from articular rheumatism we may, in some cases, be able to refer the origin of the valvular trouble to a previous attack of acute endocarditis, excited by one of the other causes above mentioned. Thus it is well known that acute endocarditis may occur in the course of scarlet fever, diphtheria, and typhoid fever, and eventuate in chronic disease, but this is, according to our experience, very rare.

In quite a large number of cases of heart disease, however, we can not obtain a history of acute endocarditis. We have to do here with an endocarditis which is chronic from the start, which also leads gradually to thickening, contraction, adhesion, and calcification of the valves.

The causes of this chronic fibrous endocarditis are probably the same as of acute articular rheumatism, but in such cases they act from the start in a chronic manner. Perhaps often there may have been an incipient acute endocarditis, whose course was unperceived. We not infrequently learn from patients with chronic heart disease, who have never had an attack of acute articular rheumatism, that in former years they did suffer repeatedly from mild rheumatic pains, to which they paid little attention. Furthermore, it is by no means exceptional to observe that such patients with a well-developed cardiac valvular lesion later on undergo one or more attacks of acute polyarthritis. Heart disease is also rarely associated with genuine chronic arthritis deformans. In other cases of valvular disease we must consider the possibility of other lesions, some infectious, some perhaps of a chemical and mechanical nature. Here belong, in the first place, those cases of cardiac disease which are associated with general arterio-sclerosis (atheroma of the blood-vessels). Indeed, atheromatous degeneration of the aorta seems sometimes to extend directly to the aortic valves, and thus cause a valvular lesion. Every ætiological factor favorable to general arterio-sclerosis is, therefore, important in the ætiology of valvular disease. This includes advanced life, excessive physical labor, alcoholism, and genuine gout. Another important cause is syphilis. Of late years, since we have paid more attention to this last factor than formerly, we have observed many cases of valvular disease which were almost certainly of syphilitic origin. In particular, lesions of the aortic valves, when there is no evidence of other causation, must awaken our suspicion of syphilis. There remains to be mentioned the influence of chronic nephritis upon the development of cardiac valvular disease, although in the not infrequent cases in which chronic nephritis, particularly of the interstitial variety, is associated with chronic endocarditis, it is not always easy to determine whether both conditions are related in the way of cause and effect, or whether they are both secondary, and both alike the result of some third unfavorable influence. A hereditary predisposition to heart disease is not very frequent, but yet it can be made out with certainty in many cases. We have ourselves seen five members of the same family who have suffered from chronic heart disease, some from pure valvular disease and some from severe so-called idiopathic hypertrophy. Perhaps the very frequent occurrence of heart disease in many families is also connected with a special family predisposition to rheumatic affections, the occurrence of which predisposition can not, in our opinion, be denied. Finally, a small number of cases of heart disease, especially in the right side of the heart, depend upon anomalies of development of the heart—congenital heart disease.

Valvular disease of the heart occurs at every age of life. The time of origin of most cases, corresponding in part to the occurrence of acute articular rheumatism, falls in youth and middle age, somewhere between eighteen and forty; but severe valvular disease is not infrequent even in children; while late in life the clinical picture of valvular disease is often confused by the simultaneous presence of general arterio-sclerosis, pulmonary emphysema, or kidney trouble. Heart disease is said to be rather more frequent in the female sex than in the male. Women with heart disease not infrequently date their symptoms from pregnancy and the puerperium.

General Pathology of Valvular Disease of the Heart.—Every valve of the heart, in order to fulfill its physiological task, must, on the one hand, open perfectly at the right time in order to furnish a free passage to the blood-current through the appropriate orifice, and must, on the other hand, close firmly and perfectly at the right time in order to make any abnormal backward flow of blood impossible. In both relations the function of the valves may be disturbed by chronic endocarditis, the disturbance being the result of their anatomical changes. If the tips of the valves are shortened on their free edges by contraction, or if the complete unfolding of the auriculo-ventricular valves is hindered by a shortening of their chordæ tendineæ, the closure of the valve can not be complete. At the moment when the closure of the valve is necessary a fissure remains open between its apices. We call this condition an insufficiency of the valve. On the other hand, the valves may lose their capability of free and sufficient separation from one another, as a result of thickening and calcification of the connective tissue, and also as a result of adhesions of the points of the valves with one another. At the moment when the blood-current should pass freely through the open orifice, the valve remains a stiff, narrow ring, through which the blood must force its way—stenosis of the orifice. The changes in the valves are often of such a sort that they cause at the same time both an insufficiency of the valve and a stenosis of the orifice. The thickening and calcification of the valves in stenosis cause, as a rule, a valvular insufficiency at the same time; but an insufficiency, set up by a contraction of the edges of the valves, may occur without a coincident stenosis of the orifice.

The abnormal and injurious effect of a valvular lesion upon the circulation in the heart is felt in two directions. Either the obstacles to the circulation are increased in certain places, or there is a greater diastolic distention of certain portions of the heart. Both circumstances of course demand greater cardiac effort. If the heart were to labor with only the same degree of energy as under normal conditions, in spite of the increased resistance, or in spite of the greater distention, there would speedily be an impairment of circulation, incompatible with the continuance of life; for, if the increased resistance were not overcome, or if the abnormally distended cavity were not properly emptied, there would at once be a rapidly increasing congestion behind the diseased valve, and beyond it a constantly diminishing pressure. We can speak of a circulation of the blood only when, in a given interval of time, exactly as much blood is driven out of the heart as flows into it. The slightest difference in this regard would, in a very brief period, produce such a congestion of blood in the veins and such a diminution of blood in the arteries that the demand of the tissues for oxygen could no longer be satisfied, and death would be the necessary result. A normal circulation is maintained so long as the amount of arterial blood, which flows into the organs in a given interval of time, is sufficiently great and also capable of immediate increase, proportional to any temporary increase in the demands of the organs, as, for example, when there is bodily exertion. The amount of blood flowing through the organs in a given time depends upon the degree of distention, the frequency of contraction, and the complete emptying of the left side of the heart. This amount ("the size of the circulation") may be diminished, and yet the circulation continue as such; but that the circulation can, in spite of the disturbance occasioned by a valvular lesion, still be maintained in a satisfactory manner is due to the capability of the heart of overcoming the obstacles to the circulation by means of increased work. It is one of the wisest contrivances in our organism, that the heart has control of a reserve fund of strength, which comes into action, if need be, in a way to compensate as far as possible for any disturbance of the circulation. This explains why a man with valvular disease of the heart may be almost perfectly well for a long time, while the increased work of certain portions of his heart suffices to keep up an approximately normal circulation in spite of the existing valvular disease. We call a heart disease, in which there is at least no marked disturbance of circulation, a compensated heart disease.

The abnormal increase in functional activity of certain portions of the heart, associated with every cardiac lesion, and referable in every case, as we have said, either to increased resistance or to increased distention, results in a hypertrophy of those particular portions of the heart, just as in the case of any other muscle. This hypertrophy does not consist of an increase in thickness of the individual muscular fibers, but chiefly of an increase in number. The total bulk of the cardiac muscle increases, and thus its capacity for work naturally becomes greater. It goes without saying that increased nutritive processes and a large supply of nourishment for the heart are necessary to bring about such a hypertrophy, by which alone a compensation of the heart disease is possible for any length of time. Hence we find the secondary hypertrophy of the heart absent, or at least only imperfectly developed, in feeble individuals, especially in those who have suffered from some other chronic wasting disease besides the heart disease, such as phthisis or carcinoma.

Although the compensatory processes in the heart may prevent for a long time any marked disturbance of the circulation, the already overburdened heart can no longer completely satisfy any excessive demands upon it, even in a compensated heart disease. Hence patients with a compensated heart disease are free from subjective disturbance only when they remain at rest, while the signs of a disturbed circulation usually become quite apparent on slight physical exertion.

The hypertrophied cardiac muscle can seldom supply permanently the abnormally great drafts made upon it. There finally comes a condition of "fatigue," of "cardiac insufficiency." The cause lies either in the increase of the valvular disease, so that the hindrance to the blood-current caused by it can no longer be completely overcome, or in the fact that the nervous and muscular elements in the heart have their powers gradually impaired by a disturbance of circulation in the heart itself. In short, in every heart disease the moment may finally come when the capacity of the heart has reached its limit, and hence the compensation of the heart disease ceases. The results of stasis now appear with increasing severity in the different organs, as we shall learn to recognize later on, and the patient finally succumbs to them, unless some intercurrent event puts an end to life.

After these general remarks, which will be understood better on reading what follows, we will pass on to the special description of the different forms of heart disease and their physical signs.

1. Insufficiency of the Mitral Valve

Mitral insufficiency is one of the most frequent forms of heart disease. It develops in acute or chronic endocarditis of the mitral valve, from contraction of the free edges of the valve or from shortening of the chordæ tendineæ. In rare cases it comes on from partial adhesion of the valves to the walls of the ventricle.

The closure of the mitral valve occurs normally at each systole of the left ventricle. It prevents the return of blood from the left ventricle to the left auricle. If the mitral valve is insufficient and its closure is incomplete, at every systole of the left ventricle a part of the blood is thrown back from it into the left auricle through the open space of the ostium venosum. This abnormal backward wave encounters the blood-current coming in an opposite direction into the left auricle from the pulmonary veins. The meeting of these two opposing currents, as well as the stream of regurgitant blood pressing through the open chink in the mitral valves, sets the blood in a whirl, and this impinging upon the tense edges of the valves produces a loud, blowing, systolic murmur. We hear this murmur loudest at the apex of the heart, corresponding to the laws of conduction in the thorax; yet it usually is propagated so far that it may often be heard at the other cardiac orifices, although not so distinctly. A loud systolic mitral murmur can also be heard sometimes in the back, on the left, and occasionally on the right. In some few cases the systolic murmur of mitral insufficiency is heard best in the second left intercostal space. This is probably because the murmur occasioned by the commotion in the left ventricle is conducted by the left auricular appendix to the anterior chest-wall (Naunyn). Curschmann has pointed out that this is particularly apt to happen in cases of incipient mitral insufficiency; but, as a rule, even in these cases, the murmur is found at the apex of the heart. In most instances the systolic muscle-sound of the left ventricle, the so-called first sound of the heart, can also be heard besides the systolic murmur. It can be heard rather better if the ear is slightly removed from the ear-piece of the stethoscope [meaning the mon-aural instrument]. Exceptionally the sound may be completely obscured by the murmur. The second sound is often not to be heard at the apex, probably because it is obscured by the relatively protracted murmur.

Since the left auricle, at each systole of the ventricle, receives blood from two sides—its normal quantity from the pulmonary veins, and, besides that, the abnormal blood-wave from the left ventricle—it becomes much dilated. At the next diastole of the left ventricle the whole amount of blood collected in the auricle under increased pressure pours into the left ventricle through the mitral valve, which is now wide open (supposing a pure insufficiency of the valve without any stenosis). We see, then, that in pure mitral insufficiency the left ventricle must be filled beyond the normal amount during the diastole. The left ventricle must also expel in the following systole an abnormally large amount of blood. Although by this contraction only a part of the blood reaches the aorta in the direction of the normal blood-current while a part pours back into the auricle, the work of the left ventricle is of course excessive. Thus, in pure mitral insufficiency, the left ventricle is dilated as a result of its increased filling in diastole, and is hypertrophied as a result of its increased labor. The general arterial tension remains approximately normal. It is not increased, since a part of the abnormal amount of blood, which pours out of the left ventricle at every systole, flows backward into the auricle. So long as the left ventricle is completely emptied by vigorous contractions, the aorta receives about the normal amount of blood, and the radial pulse remains, therefore, in cases of pure mitral insufficiency, of about the normal strength and tension.

The anomalies in the movements of the blood in mitral insufficiency produce still other effects. We have already seen that the left auricle is dilated from its overfilling. It also becomes hypertrophied, so far as its weak muscular structure permits, but it is not in itself capable of compensating for the disturbance which the pulmonary circulation suffers from the mitral insufficiency, for the back current from the left ventricle, and the consequent high pressure in the left auricle, must plainly offer an abnormal hindrance to the flow of blood from the pulmonary veins. This stasis sets back through the pulmonary capillaries and arteries into the right ventricle. This may be recognized, on physical examination, by the change in the pulmonic second sound, which is louder, more valvular, and "accentuated," since the closure of the semilunar valves in the pulmonary artery now takes place under the abnormally high pressure which prevails in the arteries of the lungs. The right ventricle has the task of overcoming this abnormal stasis in the pulmonary circulation. It can overcome the abnormal resistance in the pulmonary circulation by increased work, and as a result it becomes hypertrophied. So long as the hypertrophy of the right ventricle suffices to maintain the normal pulmonary circulation, the stasis extends no farther backward, but in the later stages of heart disease we see the right ventricle becoming paralyzed, and more and more dilated as a result of stasis. The flow of venous blood from the body into the right auricle and ventricle is now rendered more difficult. The signs of venous stasis become manifest; the patient has a cyanotic hue, congestive œdema appears in the face and the extremities, symptoms of passive congestion of the liver, spleen, and kidneys appear, and, in short, there is developed the picture of an uncompensated heart disease.

If we now sum up the physical signs of mitral insufficiency, the different methods of investigation give the following results:

INSPECTION.—The cardiac region often seems rather prominent, as a result of the hypertrophy of the heart. This protrusion is most marked in young persons with a yielding thorax. The apex-beat is, as a result of the dilatation and hypertrophy of the left ventricle, displaced toward the left and sometimes downward into the sixth intercostal space. It is more extensive and stronger than normal ("heaving"). The apex-beat is somewhat displaced toward the left as a result of the hypertrophy and dilatation of the left ventricle, and it is quite marked. Besides that, we often see a diffuse pulsation in the whole cardiac region. In the epigastrium we sometimes see an epigastric pulsation proceeding from the hypertrophied right ventricle. In cases which are no longer perfectly compensated the stasis in the veins of the body is rendered apparent by the general cyanotic appearance of the patient and the marked filling of the jugular veins in the neck. Undulatory or pulsating movements often occur in the latter (see tricuspid insufficiency, below).

PALPATION.—By palpation likewise we perceive the abnormal vigor and extent of the apex-beat, and the displacement of the same toward the left; and often, also, an extensive diffuse pulsation over the cardiac region, and in particular a distinct epigastric pulsation, due to the right ventricle. We often feel a systolic thrill at the apex of the heart—a "cat's purr"—by laying the hand flat on the chest. The same whirl of blood, which is audible as a murmur, may be perceived as a fine tremor of the chest-wall.

The radial pulse is quite strong and usually regular. The sphygmographic tracing of it shows nothing characteristic in mitral insufficiency.

PERCUSSION.—This usually gives at first only a moderate increase of the heart's dullness to the left, and a little upward dilatation of the left auricle and left ventricle, but in the later stages there is at the same time an increase of the heart's dullness to the right, caused by hypertrophy and dilatation of the right ventricle. The whole area of cardiac dullness may finally extend two fingers' breadth beyond the right edge of the sternum, and to the left it may reach the mammillary line, or even pass far beyond it.

AUSCULTATION.—At the apex of the heart we hear a loud, quite long, pure systolic blowing murmur, limited to the systole, either replacing the first sound or accompanying it. The second sound is often obscure or inaudible at the apex, but the pulmonic second sound is increased and accentuated. Auscultation of the vessels gives nothing characteristic.

2. Stenosis of the Mitral Orifice (Mitral Stenosis)

Mitral stenosis often develops in chronic endocarditis of the mitral valve, as a sequel to a previous insufficiency. The valve constantly grows stiffer and more rigid, and the signs of stenosis gradually predominate over those of insufficiency. Hence we very often find stenosis and insufficiency of the mitral valve combined, but often the signs of stenosis are so much more prominent that we can properly speak of a pure mitral stenosis.

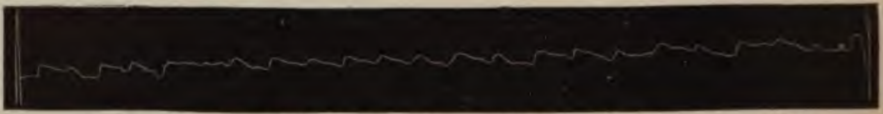

FIG. 39.—Pulse curve in marked mitral stenosis.

The disturbance which the circulation suffers in mitral stenosis is much greater than in mitral insufficiency. In mitral stenosis the orifice may finally become so narrow that it scarcely admits an ordinary lead-pencil. The influx of blood into the left ventricle is accordingly much impeded. During the diastole of the left ventricle the blood must force its way through the stiff and narrow ring of the mitral valve. In this way, again, are caused irregular whirling movements in the blood, and abnormal vibrations of the mitral valve, giving rise in most cases to an audible diastolic murmur. In mitral stenosis the left ventricle receives less than its normal amount of blood, and therefore it has no direct occasion for hypertrophy, and in fact it is sometimes found at the autopsy to be comparatively small and to be crowded backward by the enormously dilated and hypertrophied right heart. Nevertheless, we do often find hypertrophy of the left ventricle in cases of mitral stenosis, and for this reason, that mitral stenosis usually develops gradually from a previous insufficiency of the valves. That is, the chronic endocarditis occasions, probably in every case, first, an insufficiency of the valve, which is later followed by stenosis as the change progresses. We have already seen that insufficiency of the mitral valve always leads to hypertrophy of the left ventricle, and so we find it to persist even at a time when stenosis has become the prominent lesion. In other cases of stenosis, hypertrophy of the left ventricle is due to certain associated conditions, such as arterio-sclerosis or chronic nephritis. And finally we should also consider Friedreich's theory, that severe venous congestion may extend into the capillaries, and thence finally occasion abnormal resistance to the arterial circulation.

The radial pulse in mitral stenosis is approximately normal, as long as the ventricle is sufficiently filled with blood during diastole. In spite of the stenosis of the mitral valve, the left ventricle may be satisfactorily filled, and this is more likely if the action of the heart is slow, allowing a longer diastole; and if, also, the left auricle is still capable of vigorous contraction. If, however, the action of the heart is hurried, and the left ventricle is no longer sufficiently distended with blood during the diastole, the radial pulse becomes small and of low tension. Marked arhythmia is very often present in mitral stenosis, probably because of the insufficient amount of arterial blood supplied to the myocardium and its ganglia.

The hindrance to the flow into the left ventricle in mitral stenosis soon leads to a marked stasis, which extends to the right side of the heart through the left auricle, and the pulmonary veins, capillaries, and arteries. The left auricle is dilated first (often to an enormous extent), and its walls are hypertrophied, but it can overcome only a very small part of the resistance at the mitral orifice. The right ventricle can, by more work, so increase the pressure in the pulmonary vessels that, in spite of the narrowed orifice, an approximately sufficient quantity of blood may pour into the left ventricle. Hence we find in mitral stenosis a very marked hypertrophy and dilatation of the right ventricle. The stasis in the pulmonary circulation, manifested objectively by the accentuation of the pulmonic second sound, has as a result a gradually developing ectasis of the pulmonary capillaries. Thickening of the intima of the pulmonary arteries and veins also usually develops. (See the chapter on brown induration of the lungs.)

The results of physical examination are as follows:

INSPECTION.—The whole cardiac region may seem slightly prominent, as a result of the hypertrophy of the heart. This protuberance is most marked in children with their yielding thoracic walls. The heart's action is usually extended over a larger area, but in pure mitral stenosis the apex-beat is no stronger than usual, though often displaced to the left. We have frequently noticed a marked pulsation in the epigastrium, produced by the right side of the heart. The jugular veins are apt to be prominent, and show the different forms of undulatory and pulsating movement.

PALPATION.—This also gives signs corresponding to the more extended action of the heart. We sometimes feel the pulsation of the dilated right ventricle even to the right of the sternum. In some cases we feel a diastolic thrill at the apex of the heart, which alone may almost establish the diagnosis of mitral stenosis. This thrill arises from the same vertiginous currents in the blood which form the basis of the diastolic murmur (*vide infra*). The radial pulse is small in every case of severe mitral stenosis, and is very often irregular.

PERCUSSION.—Percussion shows, in the first place, an extension of cardiac dullness toward the right, reaching to the right edge of the sternum, or far beyond. Dullness also extends as a rule further to the left than normal. This is in part due to the hypertrophy of the left ventricle (*vide supra*), in part to a dilatation of the right side of the heart, so great as to push the left ventricle backward and to the left. The great distention of the left auricle causes an enlargement of the cardiac dullness upward. We observed one case in which the left auricle was so enormously dilated that it extended behind and beyond the right auricle toward the right, and was one factor in the increase of cardiac dullness toward the right, as observed during life.

AUSCULTATION.—The characteristic auscultatory sign of mitral stenosis is the diastolic [pre-systolic] murmur at the apex. This is never so loud and blowing as the systolic murmur of insufficiency, but it usually sounds more rolling or rippling. It is loudest at the apex, and it is transmitted only slightly toward the base. Since, as has been said, the left ventricle in mitral stenosis is sometimes pushed to the left and backward by the very much enlarged right ventricle, in looking for the murmur we must often go far to the left, in order not to auscult the right ventricle only.

The origin of the murmur is easily explained. In the diastole of the left ventricle the blood-current must force its way through the narrow mitral orifice, whence vertiginous movements arise in the blood, and produce the murmur. Since the blood flowing through the narrow orifice has a current of relatively slight intensity, the murmur produced by it can not be very loud. Indeed, in the most extreme cases of mitral stenosis, the murmur is particularly apt to be very faint, and when the heart's action is hurried and irregular, entirely inaudible. Not infrequently the murmur is not heard until the second half of diastole. That is, when the contraction of the left auricle gives a fresh impulse to the current of blood streaming through the stenosed orifice. A murmur of this sort, which is audible only at the end of diastole, and passes with a distinct crescendo directly into the loud systolic sound and terminates with this, is called a pre-systolic murmur. This murmur can often be plainly felt if the hand is laid upon the apex of the heart, as a pre-systolic thrill.

It is not very exceptional to find no murmur audible in cases of extreme mitral stenosis. If such cases do not come under observation till the last stage of the disease, the mitral stenosis may readily be overlooked. We have ourselves repeatedly found, in cases of mitral stenosis, that as the lesion grew worse the distinct diastolic or pre-systolic murmur gradually and completely disappeared. This is explained by the fact that as the chink in the valve grows narrower and the weakness of the heart greater, the blood is not forced through the narrow orifice with sufficient vigor to cause audible vibrations of the thickened valves. If the left ventricle is wholly displaced backward by the enormous increase in size of the right ventricle, we have also less favorable conditions for the propagation of the waves of sound from the mitral valve to the ear.

The first sound at the apex is maintained in pure mitral stenosis. Indeed, it is often noticeably loud and valvular. All the later observations confirm the view that the systolic sound is a muscle sound, and consequently it is probable that this vigorous first sound is due to the contraction of the left ventricle, which, as we have seen above (page 314), is often hypertrophied, and also, as a result of the stenosis, only imperfectly filled. The strength of the first ventricular sound in mitral stenosis affords, at any rate, a marked contrast to its weakness in aortic insufficiency (*vide infra*). We have a strong sound when the ventricle is ill-filled, diminution and dullness of sound when the ventricle is over-filled. If insufficiency of the valve co-exists, we may hear a systolic murmur with the first sound or instead of it.

The very marked accentuation of the pulmonic second sound, the result of the abnormally high tension in the pulmonary artery, is almost constant. It fails only in very anæmic, weak people, or in co-existing insufficiency of the tricuspid valve (*vide infra*). The second sound at the base is very often "divided" or reduplicated. The closure of the semilunar valves in diastole does not happen at the same time in the pulmonary artery and in the aorta, on account of the unequal tension in the two vessels, so that consequently the two sounds are heard, one shortly after the other. Although this division of the second sound is particularly frequent in mitral stenosis, it is by no means a pathognomonic sign of it.

Mitral stenosis is one of the severest forms of heart disease. It almost always causes greater subjective disturbance than mitral insufficiency. Hypertrophy of the right ventricle may, indeed, maintain for a time an approximately complete compensation, but the signs of marked stasis in the pulmonary circulation, and further in the veins of the body, are apt to appear quite early. It must be admitted that the disturbances of compensation which occur in mitral stenosis are particularly susceptible of treatment, so that for many years there may be times of improvement, alternating with times of aggravation of the symptoms; but finally the time comes when it is impossible to regulate the circulation. The dyspnœa grows worse, and finally death occurs, usually preceded by dropsy.

3. Insufficiency of the Semilunar Valves of the Aorta

Insufficiency of the aortic valves is due most frequently to contraction of the free edges of the valves. Tears, perforations, or adhesions of the valve to the wall of the vessel more rarely lead to insufficiency. The cause of all these changes is either a valvular endocarditis, which is usually a sequel of articular rheumatism, or a general arterial atheroma, which extends gradually from the intima of the aorta to the valves. We have already referred to syphilis as a not very infrequent cause of aortic lesions. A question of practical importance is turbance occasioned by a valvular lesion, still be maintained in a satisfactory manner is due to the capability of the heart of overcoming the obstacles to the circulation by means of increased work. It is one of the wisest contrivances in our organism, that the heart has control of a reserve fund of strength, which comes into action, if need be, in a way to compensate as far as possible for any disturbance of the circulation. This explains why a man with valvular disease of the heart may be almost perfectly well for a long time, while the increased work of certain portions of his heart suffices to keep up an approximately normal circulation in spite of the existing valvular disease. We call a heart disease, in which there is at least no marked disturbance of circulation, a compensated heart disease.

The abnormal increase in functional activity of certain portions of the heart, associated with every cardiac lesion, and referable in every case, as we have said, either to increased resistance or to increased distention, results in a hypertrophy of those particular portions of the heart, just as in the case of any other muscle. This hypertrophy does not consist of an increase in thickness of the individual muscular fibers, but chiefly of an increase in number. The total bulk of the cardiac muscle increases, and thus its capacity for work naturally becomes greater. It goes without saying that increased nutritive processes and a large supply of nourishment for the heart are necessary to bring about such a hypertrophy, by which alone a compensation of the heart disease is possible for any length of time. Hence we find the secondary hypertrophy of the heart absent, or at least only imperfectly developed, in feeble individuals, especially in those who have suffered from some other chronic wasting disease besides the heart disease, such as phthisis or carcinoma.

Although the compensatory processes in the heart may prevent for a long time any marked disturbance of the circulation, the already overburdened heart can no longer completely satisfy any excessive demands upon it, even in a compensated heart disease. Hence patients with a compensated heart disease are free from subjective disturbance only when they remain at rest, while the signs of a disturbed circulation usually become quite apparent on slight physical exertion.

The hypertrophied cardiac muscle can seldom supply permanently the abnormally great drafts made upon it. There finally comes a condition of "fatigue," of "cardiac insufficiency." The cause lies either in the increase of the valvular disease, so that the hindrance to the blood-current caused by it can no longer be completely overcome, or in the fact that the nervous and muscular elements in the heart have their powers gradually impaired by a disturbance of circulation in the heart itself. In short, in every heart disease the moment may finally come when the capacity of the heart has reached its limit, and hence the compensation of the heart disease ceases. The results of stasis now appear with increasing severity in the different organs, as we shall learn to recognize later on, and the patient finally succumbs to them, unless some intercurrent event puts an end to life.

After these general remarks, which will be understood better on reading what follows, we will pass on to the special description of the different forms of heart disease and their physical signs.

1. Insufficiency of the Mitral Valve

Mitral insufficiency is one of the most frequent forms of heart disease. It develops in acute or chronic endocarditis of the mitral valve, from contraction of the free edges of the valve or from shortening of the chordæ tendineæ. In rare cases it comes on from partial adhesion of the valves to the walls of the ventricle.

The closure of the mitral valve occurs normally at each systole of the left ven-causes an extension of the whole heart to the right, or because the right ventricle is also hypertrophied. The latter change occurs in pure aortic insufficiency when the compensation is no longer complete, and the stasis extends backward from the left ventricle, through the pulmonary circulation, into the right side of the heart.

It may also be remarked here that, in insufficiency of the aortic valves, the ascending aorta is often considerably dilated by the marked impulse from the amount of blood pouring into it. A moderate degree of dullness is found over the dilated aorta, which may sometimes be made out at the sternal end of the second right intercostal space.

Auscultation.—Insufficiency of the aortic valves is characterized by a long-drawn, loud, blowing diastolic murmur, the origin of which has been explained above. The place in which the murmur is heard loudest is not the sternal end of the second right intercostal space, the ordinary point for auscultation of the aorta, but it almost always lies farther to the left. Corresponding to the backward current of blood toward the left ventricle, which begets the murmur, we hear the latter loudest over the upper part of the sternum or even at its left border. In some cases the murmur assumes a marked "musical character"—that is, there is a definite high musical tone, which is due to a tendinous fiber arising from a wearing away of the valve, and set in vibration by the diastole, or to some similar cause. The diastolic murmur is often audible at the apex, but it is faint there. It is only in a few exceptional cases that there is no diastolic murmur in aortic insufficiency. Sometimes we hear not only the murmur, but also the diastolic sound of the closing valve. During systole we scarcely ever hear over the aorta a pure, loud, first sound, but almost always a short systolic murmur. This may, of course, be due to accompanying stenosis of the aortic valves, but yet is very common when there is insufficiency alone. It is explained by O. Rosenbach as being due to the fact that at the beginning of the systole of the left ventricle, the diastolic flow of blood has not yet completely ceased, so that the emerging blood-stream encounters this opposing current. This meeting of the two blood-currents in the root of the aorta during systole causes the vibrations which give rise to the short systolic murmur. It is to be noted that this circumstance also may perhaps have some influence upon the development of the hypertrophy of the left ventricle.

It is very interesting and important that, as Traube pointed out, we find the first sound at the apex scarcely ever loud and pure, but often very indistinct and muffled; or else we hear a short systolic murmur instead of it. This veiling of the first sound at the apex of the heart has theoretic interest, because it has been employed as an argument against the view that the first mitral sound is a muscular sound; for it is not, in fact, at once apparent why the hypertrophied left ventricle should so often fail to produce a distinctly audible tone by its contraction. But, as we have already pointed out (see page 316), the probable explanation lies in the previous over-distention of the left ventricle during diastole. This renders the systolic contraction difficult and somewhat slow, and may be the cause of the indistinctness of the muscle-sound. In later stages of the disease we may also adduce parenchymatous degeneration of the myocardium, as an explanation of its feebleness. The systolic murmur, often heard at the apex in aortic insufficiency, may depend upon a co-existing true mitral insufficiency, but it is probably often due to a relative insufficiency of the mitral, since the valves, which are normal in themselves, can no longer cause a perfect closure of the left mitral orifice now that the left ventricle is dilated.

Symptoms in the Peripheral Arteries.—Such remarkable symptoms are found in the peripheral arteries in aortic insufficiency that they demand a brief special description. The first striking symptom is the strong pulsation not only of the larger but also of the smaller arteries, even those the pulsation of which is not generally visible. We see and feel not only a strong pulsation in the carotids, but also in the tortuous brachial artery, in the radial, ulnar, temporal, dorsalis pedis, etc. We sometimes feel an arterial pulse in the liver through the abdominal walls.

The rapid decline of the pulse—the *pulsus celer* [Corrigan's pulse]—is most characteristic of aortic insufficiency, and is to be felt especially in the radial artery, but also in the femoral, dorsalis pedis, and other vessels. An abnormally large quantity of blood is thrown into the arteries from the hypertrophied and dilated left ventricle; hence the high ascent of the pulse; but since the distended artery quickly contracts again, and particularly as at the next diastole of the ventricle the blood escapes in two directions, into the capillaries and back into the ventricle, an abnormally rapid and deep decline of the pulse follows the high ascent of its wave—a condition which explains the "jumping," "springing" pulse (*pulsus celer*) of aortic insufficiency. The quality of the pulse may be plainly recognized also in the sphygmographic tracing (see Fig. 40). The abnormal backward wave may even be detected in the capillaries. We often see a marked pallor of the finger-nails at every diastole of the heart in patients with aortic insufficiency—Quincke's capillary pulse.

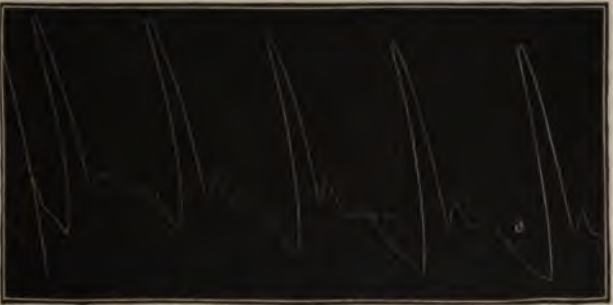

FIG. 40.—Pulse curve in aortic insufficiency.

The auscultatory phenomena over the arteries are connected partly with the changing conditions of tension of the arterial walls. We very often hear a short, rough, systolic murmur in the carotid. The second sound, which is well known to be the transmitted aortic second sound, is absent. Instead of it we sometimes hear faintly transmitted the aortic diastolic murmur. The sound of the medium-sized and smaller arteries is very characteristic. By applying the stethoscope lightly we hear over the femoral, the brachial, and often over the radial, the ulnar, the palmar arch, and the dorsalis pedis, a marked valvular sound, which is changed by pressure on the artery, especially in the larger arteries, to a loud stenotic murmur. The quicker the pulse, the more certain are we to hear these sounds in the arteries. In the most marked cases of Corrigan's pulse these vascular sounds are so loud that we may hear almost anywhere below the knee, by means of a stethoscope, a valvular sound. The double sound in the femoral (Traube's double sound) is quite a frequent phenomenon, about the origin and significance of which there has been much discussion. The double sounds either follow each other shortly, so that the first seems something like a preparatory blow for the second, or they are separated from each other by a longer interval, like the two sounds of the heart. Traube explained the origin of the first sound by the sudden tension of the vessel-wall, as in the simple femoral sound, and the second sound by the sudden relaxation of it. Friedreich has pointed out in regard to this that, in co-existing tricuspid insufficiency, a sound may also be produced in the femoral vein by tension of the venous valves. The double sound in the femoral may probably have different causes of origin. It is, of course, by far the most frequent in aortic insufficiency, but it has also been repeatedly observed in other forms of heart disease, as in mitral stenosis. The so-called Duroziez's double murmur in the femoral is more rare, and it is noticed almost exclusively in aortic insufficiency. This is when we hear, by pressing the stethoscope on the femoral, two murmurs plainly distinct from each other, of which the first comes from the passage of the systolic blood-wave, and the second from the passage of the abnormal backward wave coming from the periphery of the vascular system through the artificially contracted vessel. While the well-marked Corrigan's pulse and the arterial sounds associated with it are so characteristic, they do not appear with great distinctness in every case of aortic insufficiency, but only in many; while in other and apparently similar cases they are indistinct or quite absent. Probably this difference depends at least in part upon a difference in the elasticity of the arterial walls. At any rate, we have seen well-marked Corrigan's pulse and sounding arteries in youthful patients; while in elderly persons with accompanying arterio-sclerosis or similar changes, these phenomena are not apt to be striking.

Aortic insufficiency is a comparatively favorable form of heart disease, since it may be almost perfectly compensated for years by hypertrophy of the left ventricle. Many patients with moderate aortic insufficiency feel perfectly well, and are even capable of quite hard work. They have not the slightly cyanotic hue which almost all patients with mitral disease exhibit, but they have a normal or even a pale complexion. If, however, the signs of disturbed compensation once appear in aortic insufficiency the severest sequelæ may develop quite rapidly. In aortic insufficiency it is exceptional to see such repeated changes from bad to good, and good to bad, as are often observed, for instance, in mitral stenosis. If the left ventricle becomes enfeebled, it can no longer satisfy the excessive demands made upon it. Passive congestion ensues, extending backward through the pulmonary circuit into the systemic veins, even while the pulse may still seem to be powerful. The average arterial tension becomes subnormal, dyspnœa increases, and there are attacks of cardiac asthma. Œdema appears, and the patient dies with the symptoms of anasarca. We will speak more fully below of certain intercurrent events in aortic insufficiency, such as cerebral hæmorrhage and pericarditis.

4. Stenosis of the Aortic Orifice

Except for the mild forms of aortic stenosis, which often come on with aortic insufficiency, aortic stenosis is a rare disease. It arises from marked thickenings and calcifications, and especially from adhesions of the aortic valves to one another. The stenosis may become so considerable that the orifice is finally reduced to a mere fissure, through which the left ventricle must force the blood at its systole. The fluttering of the valves and the vertiginous movements in the blood thus arising produce a loud systolic murmur. The left ventricle is compelled to do greater work in consequence of the increased resistance of the aortic orifice, and hence becomes hypertrophied. In spite of the increased effort, however, comparatively little blood reaches the arterial system, and consequently the radial pulse is small and the arteries contracted.

Inspection and Palpation.—Upon physical examination of the heart we find, in the first place, the apex-beat displaced outward as a result of the hypertrophy of the left ventricle, and also often more powerful than normal. It may, however, be noticeably feeble, perhaps because of the slowness of the systole. A former explanation of this feebleness was the diminution of the recoil of the apex (the Gutbrod-Skoda theory of the apex-beat).

Percussion.—Percussion gives an extension of the heart's dullness to the left. The right ventricle is also dilated and hypertrophied to a moderate degree in the later stages, if the stasis extends backward through the pulmonary circulation.

Auscultation.—On auscultation, we hear over the aorta a very loud "sawing," long-drawn, systolic murmur, which is usually transmitted to the right, corresponding to the course of the aorta, in distinction from the diastolic murmur of aortic insufficiency. It is usually to be heard loudest at the sternal end of the second right intercostal space, but it is audible to a lesser extent over almost the whole heart. It is usually quite loud over the carotids. The systolic sound at the apex is apt to be feeble. The second aortic sound is likewise faint or even inaudible. If there is co-existing insufficiency of the valve, the second aortic sound is replaced by a diastolic murmur.

The pulse has been already described. It is small, and often surprises one by its contrast with the strength of the apex-beat. In well-compensated cases it is regular, and often moderately or even extremely slow. This slow pulse of aortic stenosis is often explained as a compensatory change in the heart's action, appropriate to the existing lesion—the systole being lengthened, an increased amount of blood can be driven through the narrow aortic orifice. But the slowing of the cardiac action is really, in main part, a prolongation of the diastole, and therefore the slow pulse is probably due chiefly to the fact that the wall of the left ventricle is ill-supplied with blood, just as in the case of sclerosis of the coronary arteries. The sphygmographic tracing of the radial pulse (see Fig. 41) shows a low wave and a comparatively slow rise and fall.

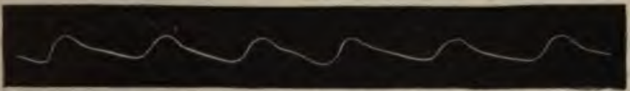
FIG. 41.—Pulse curve in stenosis of the aortic orifice.

Aortic stenosis of slight or moderate degree may be tolerably well borne by the patient. We have even seen a man with well-marked aortic stenosis who for years did not have the slightest subjective symptoms of heart-disease, until he finally died with an acute recurrent endocarditis. When the stenosis is more complete we sometimes have a most peculiar clinical picture. The pulse is very infrequent, as low as thirty to twenty-four beats in a minute. From time to time there are attacks of vertigo or syncope, the patient often falls, and has epileptiform attacks. These seizures, which may be repeated for months or even for some years, are probably connected with a sudden anæmia of the heart and brain. We have observed this remarkable group of symptoms particularly in elderly persons with aortic stenosis, due to arterio-sclerosis. In other respects the course of aortic stenosis is similar to that of the acute valvular diseases, and in the same way terminates in general circulatory derangement with its results.

5. Insufficiency of the Tricuspid Valve

Insufficiency of the tricuspid valve is extremely rare as an independent disease of the heart, but a secondary insufficiency of the tricuspid is quite frequent, and is therefore of practical interest, as it complicates other already-existing valvular diseases in the left side of the heart. It arises either from a secondary endocarditis, affecting the tricuspid, in quite an analogous manner with mitral insufficiency, or it is a so-called relative insufficiency. This name we give to that form of insufficiency which develops when the edges of the tricuspid valve, normal in themselves, at last fail to meet one another, from the increasing dilatation of the right ventricle, or at any rate from the inability of the enfeebled right ventricle properly to close the valve.

The necessary result of tricuspid insufficiency is, that in every systole of the right ventricle a backward current passes through the open tricuspid orifice into the right auricle, and thence into the veins of the body. The tricuspid insufficiency ensuing in other forms of heart disease must therefore increase the stasis murmur. This murmur can often be plainly felt if the hand is laid upon the apex of the heart, as a pre-systolic thrill.

It is not very exceptional to find no murmur audible in cases of extreme mitral stenosis. If such cases do not come under observation till the last stage of the disease, the mitral stenosis may readily be overlooked. We have ourselves repeatedly found, in cases of mitral stenosis, that as the lesion grew worse the distinct diastolic or pre-systolic murmur gradually and completely disappeared. This is explained by the fact that as the chink in the valve grows narrower and the weakness of the heart greater, the blood is not forced through the narrow orifice with sufficient vigor to cause audible vibrations of the thickened valves. If the left ventricle is wholly displaced backward by the enormous increase in size of the right ventricle, we have also less favorable conditions for the propagation of the waves of sound from the mitral valve to the ear.

The first sound at the apex is maintained in pure mitral stenosis. Indeed, it is often noticeably loud and valvular. All the later observations confirm the view that the systolic sound is a muscle sound, and consequently it is probable that this vigorous first sound is due to the contraction of the left ventricle, which, as we have seen above (page 314), is often hypertrophied, and also, as a result of the stenosis, only imperfectly filled. The strength of the first ventricular sound in mitral stenosis affords, at any rate, a marked contrast to its weakness in aortic insufficiency (*vide infra*). We have a strong sound when the ventricle is ill-filled, diminution and dullness of sound when the ventricle is over-filled. If insufficiency of the valve co-exists, we may hear a systolic murmur with the first sound or instead of it.

The very marked accentuation of the pulmonic second sound, the result of the abnormally high tension in the pulmonary artery, is almost constant. It fails only in very anæmic, weak people, or in co-existing insufficiency of the tricuspid valve (*vide infra*). The second sound at the base is very often "divided" or reduplicated. The closure of the semilunar valves in diastole does not happen at the same time in the pulmonary artery and in the aorta, on account of the unequal tension in the two vessels, so that consequently the two sounds are heard, one shortly after the other. Although this division of the second sound is particularly frequent in mitral stenosis, it is by no means a pathognomonic sign of it.

Mitral stenosis is one of the severest forms of heart disease. It almost always causes greater subjective disturbance than mitral insufficiency. Hypertrophy of the right ventricle may, indeed, maintain for a time an approximately complete compensation, but the signs of marked stasis in the pulmonary circulation, and further in the veins of the body, are apt to appear quite early. It must be admitted that the disturbances of compensation which occur in mitral stenosis are particularly susceptible of treatment, so that for many years there may be times of improvement, alternating with times of aggravation of the symptoms; but finally the time comes when it is impossible to regulate the circulation. The dyspnœa grows worse, and finally death occurs, usually preceded by dropsy.

3. Insufficiency of the Semilunar Valves of the Aorta

Insufficiency of the aortic valves is due most frequently to contraction of the free edges of the valves. Tears, perforations, or adhesions of the valve to the wall of the vessel more rarely lead to insufficiency. The cause of all these changes is either a valvular endocarditis, which is usually a sequel of articular rheumatism, or a general arterial atheroma, which extends gradually from the intima of the aorta to the valves. We have already referred to syphilis as a not very infrequent cause of aortic lesions. A question of practical importance is whether violent bodily exertion may suddenly occasion the partial laceration of an aortic valve. Many clinical observations, including a recent case of the author's, seem to show the possibility of this extremely rare occurrence.

The function of the aortic valves is to close tightly at the time of diastole of the left ventricle, in order to prevent any return of blood from the aorta into the ventricle. If these valves are insufficient—that is, if they do not close perfectly at each diastole—there is a return current of blood from the aorta into the left ventricle.

This regurgitant diastolic wave sets the tense edges of the valve in vibration as it passes over them. Furthermore, the two currents of blood impinging upon each other in the left ventricle, the one an abnormal regurgitation from the aorta, the other the normal stream from the left auricle, produce irregular whirling motions in the blood. All these vibrations are propagated to the surrounding structures, and produce the long-continued, blowing, diastolic, and remarkably characteristic murmur of aortic insufficiency.

Aortic insufficiency causes an immediate and great increase in the demands upon the left ventricle, because of its abnormal distention, for it receives, as we have already said, not merely its normal quantum of blood from the left ventricle, but also the blood which regurgitates through the leaking valves of the aorta. It is consequently overfilled at every diastole, and finally becomes permanently distended. Dilatation of the left ventricle forms a constant anatomical lesion in every case of aortic insufficiency, and is shown not only in the dilatation of the whole ventricular cavity, but also in the very characteristic flattening of the trabeculæ and of the papillary muscles. There is usually some fibrous thickening of the endocardium at the spot upon which the regurgitant blood-current is continally impinging. The left ventricle possesses sufficient reserve strength to discharge its contents completely for a long period by means of increased effort. This is indeed a task like that of Sisyphus, since the portion of the blood which is thrown into the aorta is constantly rolling back into the ventricle. The increased demands, however, lead necessarily at last to a hypertrophy of the left ventricle, often greater than is seen under any other conditions.

From the facts enumerated we can easily understand the physical signs of insufficiency of the aortic valves.

Inspection.—Great hypertrophy of the left ventricle often causes a marked protrusion of the whole cardiac region. The diffuse and very strong apex-beat, displaced downward and to the left, is especially striking. It may usually be seen in the sixth intercostal space, outside the left mammillary line, and sometimes even at the anterior axillary line. Besides, we often see a marked diffuse tremor of the whole cardiac region. There is marked pulsation of the carotid arteries in the neck. The jugular veins show undulation and pulsation, when at last compensation begins to fail.

Palpation.—We can appreciate the heart's action to a still greater extent by palpation than by inspection. The apex-beat is very resistant, massive, and plainly heaving—that is, the finger or stethoscope applied to the apex is lifted by the beat at every systole. In rare cases a diastolic thrill, corresponding to the diastolic murmur, can be felt over the base of the heart. In two such cases observed by us the murmur had a marked musical character (*vide infra*). The appearances in the arteries are given below.

Percussion.—Percussion gives an extension of the cardiac dullness to the left, beyond the left mammillary line and even to the anterior axillary line, caused by the hypertrophy and dilatation of the left ventricle. The upper boundary of the cardiac dullness is normal, or it may extend up to the third rib. The right boundary is in its normal place at the left border of the sternum, but it may also be pushed farther to the right, either because the large left ventricle of itself causes an extension of the whole heart to the right, or because the right ventricle is also hypertrophied. The latter change occurs in pure aortic insufficiency when the compensation is no longer complete, and the stasis extends backward from the left ventricle, through the pulmonary circulation, into the right side of the heart.

It may also be remarked here that, in insufficiency of the aortic valves, the ascending aorta is often considerably dilated by the marked impulse from the amount of blood pouring into it. A moderate degree of dullness is found over the dilated aorta, which may sometimes be made out at the sternal end of the second right intercostal space.

AUSCULTATION.—Insufficiency of the aortic valves is characterized by a long-drawn, loud, blowing diastolic murmur, the origin of which has been explained above. The place in which the murmur is heard loudest is not the sternal end of the second right intercostal space, the ordinary point for auscultation of the aorta, but it almost always lies farther to the left. Corresponding to the backward current of blood toward the left ventricle, which begets the murmur, we hear the latter loudest over the upper part of the sternum or even at its left border. In some cases the murmur assumes a marked "musical character"—that is, there is a definite high musical tone, which is due to a tendinous fiber arising from a wearing away of the valve, and set in vibration by the diastole, or to some similar cause. The diastolic murmur is often audible at the apex, but it is faint there. It is only in a few exceptional cases that there is no diastolic murmur in aortic insufficiency. Sometimes we hear not only the murmur, but also the diastolic sound of the closing valve. During systole we scarcely ever hear over the aorta a pure, loud, first sound, but almost always a short systolic murmur. This may, of course, be due to accompanying stenosis of the aortic valves, but yet is very common when there is insufficiency alone. It is explained by O. Rosenbach as being due to the fact that at the beginning of the systole of the left ventricle, the diastolic flow of blood has not yet completely ceased, so that the emerging blood-stream encounters this opposing current. This meeting of the two blood-currents in the root of the aorta during systole causes the vibrations which give rise to the short systolic murmur. It is to be noted that this circumstance also may perhaps have some influence upon the development of the hypertrophy of the left ventricle.

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SYMPTOMS IN THE PERIPHERAL ARTERIES.—Such remarkable symptoms are found in the peripheral arteries in aortic insufficiency that they demand a brief special description. The first striking symptom is the strong pulsation not only of the larger but also of the smaller arteries, even those the pulsation of which is not generally visible. We see and feel not only a strong pulsation in the carotids, but also in the tortuous brachial artery, in the radial, ulnar, temporal, dorsalis pedis, etc. We sometimes feel an arterial pulse in the liver through the abdominal walls.

The rapid decline of the pulse—the *pulsus celer* [Corrigan's pulse]—is most characteristic of aortic insufficiency, and is to be felt especially in the radial artery, but also in the femoral, dorsalis pedis, and other vessels. An abnormally large quantity of blood is thrown into the arteries from the hypertrophied and dilated left ventricle; hence the high ascent of the pulse; but since the distended artery quickly contracts again, and particularly as at the next diastole of the ventricle the blood escapes in two directions, into the capillaries and back into the ventricle, an abnormally rapid and deep decline of the pulse follows the high ascent of its wave—a condition which explains the "jumping," "springing" pulse (*pulsus celer*) of aortic insufficiency. The quality of the pulse may be plainly recognized also in the sphygmographic tracing (see Fig. 40). The abnormal backward wave may even be detected in the capillaries. We often see a marked pallor of the finger-nails at every diastole of the heart in patients with aortic insufficiency—Quincke's capillary pulse.

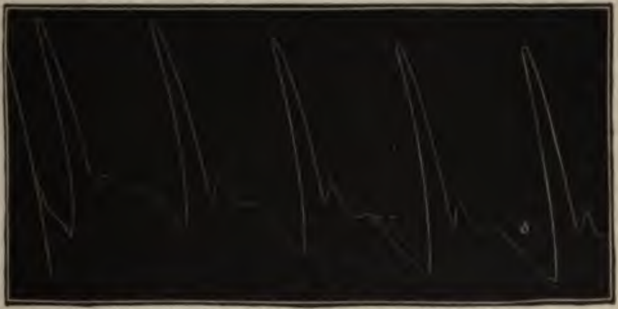

FIG. 40.—Pulse curve in aortic insufficiency.

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4. Stenosis of the Aortic Orifice

Except for the mild forms of aortic stenosis, which often come on with aortic insufficiency, aortic stenosis is a rare disease. It arises from marked thickenings and calcifications, and especially from adhesions of the aortic valves to one another. The stenosis may become so considerable that the orifice is finally reduced to a mere fissure, through which the left ventricle must force the blood at its systole. The fluttering of the valves and the vertiginous movements in the blood thus arising produce a loud systolic murmur. The left ventricle is compelled to do greater work in consequence of the increased resistance of the aortic orifice, and hence becomes hypertrophied. In spite of the increased effort, however, comparatively little blood reaches the arterial system, and consequently the radial pulse is small and the arteries contracted.

Inspection and Palpation.—Upon physical examination of the heart we find, in the first place, the apex-beat displaced outward as a result of the hypertrophy of the left ventricle, and also often more powerful than normal. It may, however, be noticeably feeble, perhaps because of the slowness of the systole. A former explanation of this feebleness was the diminution of the recoil of the apex (the Gutbrod-Skoda theory of the apex-beat).

Percussion.—Percussion gives an extension of the heart's dullness to the left. The right ventricle is also dilated and hypertrophied to a moderate degree in the later stages, if the stasis extends backward through the pulmonary circulation.

Auscultation.—On auscultation, we hear over the aorta a very loud "sawing," long-drawn, systolic murmur, which is usually transmitted to the right, corresponding to the course of the aorta, in distinction from the diastolic murmur of aortic insufficiency. It is usually to be heard loudest at the sternal end of the second right intercostal space, but it is audible to a lesser extent over almost the whole heart. It is usually quite loud over the carotids. The systolic sound at the apex is apt to be feeble. The second aortic sound is likewise faint or even inaudible. If there is co-existing insufficiency of the valve, the second aortic sound is replaced by a diastolic murmur.

The pulse has been already described. It is small, and often surprises one by its contrast with the strength of the apex-beat. In well-compensated cases it is regular, and often moderately or even extremely slow. This slow pulse of aortic stenosis is often explained as a compensatory change in the heart's action, appropriate to the existing lesion—the systole being lengthened, an increased amount of blood can be driven through the narrow aortic orifice. But the slowing of the cardiac action is really, in main part, a prolongation of the diastole, and therefore the slow pulse is probably due chiefly to the fact that the wall of the left ventricle is ill-supplied with blood, just as in the case of sclerosis of the coronary arteries. The sphygmographic tracing of the radial pulse (see Fig. 41) shows a low wave and a comparatively slow rise and fall.

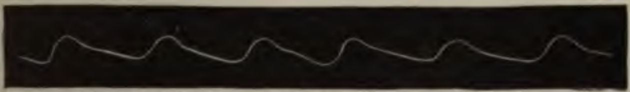
Fig. 41.—Pulse curve in stenosis of the aortic orifice.

Aortic stenosis of slight or moderate degree may be tolerably well borne by the patient. We have even seen a man with well-marked aortic stenosis who for years did not have the slightest subjective symptoms of heart-disease, until he finally died with an acute recurrent endocarditis. When the stenosis is more complete we sometimes have a most peculiar clinical picture. The pulse is very infrequent, as low as thirty to twenty-four beats in a minute. From time to time there are attacks of vertigo or syncope, the patient often falls, and has epileptiform attacks. These seizures, which may be repeated for months or even for some years, are probably connected with a sudden anæmia of the heart and brain. We have observed this remarkable group of symptoms particularly in elderly persons with aortic stenosis, due to arterio-sclerosis. In other respects the course of aortic stenosis is similar to that of the acute valvular diseases, and in the same way terminates in general circulatory derangement with its results.

5. Insufficiency of the Tricuspid Valve

Insufficiency of the tricuspid valve is extremely rare as an independent disease of the heart, but a secondary insufficiency of the tricuspid is quite frequent, and is therefore of practical interest, as it complicates other already-existing valvular diseases in the left side of the heart. It arises either from a secondary endocarditis, affecting the tricuspid, in quite an analogous manner with mitral insufficiency, or it is a so-called relative insufficiency. This name we give to that form of insufficiency which develops when the edges of the tricuspid valve, normal in themselves, at last fail to meet one another, from the increasing dilatation of the right ventricle, or at any rate from the inability of the enfeebled right ventricle properly to close the valve.

The necessary result of tricuspid insufficiency is, that in every systole of the right ventricle a backward current passes through the open tricuspid orifice into the right auricle, and thence into the veins of the body. The tricuspid insufficiency ensuing in other forms of heart disease must therefore increase the stasis in the veins of the body, and it is thus far an unfavorable complication. It has a compensatory significance only as it affords relief to the pulmonary circulation. Since a part of the blood passes back from the right ventricle into the veins, less blood than usual must reach the pulmonary arteries. The decrease in tension thus produced in these arteries makes itself apparent on auscultation, since the accentuation of the pulmonic second sound in valvular disease of the mitral orifice diminishes when tricuspid insufficiency takes place.

That tricuspid insufficiency must result in a hypertrophy of the right ventricle is explained in just the same way as the hypertrophy of the left ventricle in mitral insufficiency, from the increased influx of blood at increased tension into the right ventricle during diastole; but this effect of tricuspid insufficiency can rarely be made out in any individual case, since the right ventricle is usually already hypertrophied as a result of the disease in the left side of the heart.

The most important symptom from which we can diagnosticate tricuspid insufficiency is the venous pulse. The cause of this is the backward wave of blood produced at each systole of the right ventricle. So long as the venous valve above the bulbus jugularis is closed, we usually see only a "bulbar pulse," but very soon this valve also yields to the continued impulse of the blood, and then a strong, purely venous pulse is visible along the whole course of the jugular vein up to the vicinity of the mastoid process. The contraction of the right auricle very often causes a decidedly weaker elevation of the vein, which just precedes the marked pulsation caused by the ventricular systole (anadicrotic venous pulse). On account of the straighter course of the right innominate vein, the jugular venous pulse is often stronger on the right side than on the left. We must state, however, that the jugular venous pulse is not an absolutely certain sign of tricuspid insufficiency, since it may arise in hypertrophy of the right side of the heart without any insufficiency of the tricuspid, from the closure of the valves.

If there is pulsation in the bulb of the jugular vein and the jugular valve is still capable of closing, a low, audible, venous, valvular sound may be produced by its closure. A sound may also arise in tricuspid insufficiency, as has been already said, from the tension of the valves in the femoral vein. A visible pulsation in the larger veins of the extremities is very rare, but in tricuspid insufficiency we quite frequently feel a venous pulsation in the liver, which organ is usually enlarged by passive congestion. This may be quite apparent even in many cases in which the jugular venous pulse is absent, because the veins in the liver are without valves. [It is best appreciated by means of bimanual palpation.—V.]

Auscultation over the right side of the heart gives a systolic murmur in insufficiency of the tricuspid, arising from the regurgitating blood-current. This may be heard loudest over the lower part of the sternum, or at the sternal end of the right fifth rib. The significance of this murmur in diagnosis, however, is impaired by the fact that it can not always be separated from the systolic mitral murmur that often co-exists.

6. Stenosis of the Tricuspid Orifice

Stenosis of the tricuspid orifice is an extremely rare disease, and hence it is without practical significance. It has usually been observed, up to the present time, as a congenital form of heart disease, almost always combined with other anomalies of development in the heart.

The physical signs of tricuspid stenosis can easily be constructed theoretically. The first result must be a marked dilatation of the right auricle, and the occurrence of a diastolic or presystolic murmur over the right side of the heart. From the rarity and complex character of the cases, however, we have so far seldom had an opportunity to confirm these theories at the bedside.

The prognosis of this form of heart disease is very unfavorable, since a long-continued compensation by increased labor on the part of the right auricle is scarcely conceivable.

[Seventy cases of tricuspid stenosis have been collected by Bedford Fenwick, whose analysis affords good grounds for thinking that the lesion is often acquired. In fifty per cent. of the cases there was a clear history of rheumatism, and nearly all of the patients were more than twenty years of age at the time of death.

This lesion is never found alone, but invariably combined with mitral stenosis; all but eight of the cases were in women. Fenwick thinks that the influence of sex lies in the less onerous nature of the work of women than of men, the granulating edges of the valves being kept more in apposition, thus healing with adhesion and causing obstructions at the orifice.]

7. Insufficiency of the Pulmonary Valve

Insufficiency of the pulmonary valve is also a very rare form of heart disease. It occurs as a congenital anomaly, often combined with other failures of development, or as a disease acquired after birth. The anatomical changes in the valve, which lead to insufficiency, are precisely analogous to those which cause insufficiency of the aortic valve.

The physical signs of this form of valvular disease consist chiefly of a marked dilatation and hypertrophy of the right ventricle, to be made out by percussion, and of a loud diastolic murmur over the pulmonary valve. These signs are explained in just the same way as the precisely analogous signs in the left ventricle in aortic insufficiency.

In general, pulmonary insufficiency, like aortic insufficiency, seems to be compensated quite well for a long time by hypertrophy of the right ventricle. In many cases a co-existing patency of the foramen ovale also seems to be of favorable influence, as it lessens the stasis in the right auricle and the veins of the body, while it renders easier the filling of the left ventricle.

8. Stenosis of the Pulmonary Orifice (Pulmonary Stenosis) and the other Congenital Diseases of the Heart

1. **Congenital Pulmonary Stenosis.**—While the stenosis of the pulmonary orifice acquired in later life is so rare that it has only slight practical significance, the congenital pulmonary stenosis is of far greater importance. It is, on the whole, the most frequent of the congenital forms of heart disease. Its origin is to be referred either to an endocarditis of the pulmonary valves during fœtal life, or to anomalies in the development of the heart. The stenosis is often situated not merely at the pulmonary orifice itself, but farther back in the conus arteriosus, which seems to be narrowed by the formation of myocardial cicatrices. The pulmonary artery is often also narrowed as a whole. In the majority of cases we find, in addition, other anomalies of development in the heart, especially patency of the foramen ovale, great defects in the ventricular septum, and, in about half the cases, patency of the ductus Botalli, etc.

The symptoms of congenital pulmonary stenosis sometimes appear soon after the birth of the child. The first thing that strikes us is the marked cyanosis, which is constant, or else comes on with crying, or with movements of the body. Many children, however, reach a fair age, five or ten years, or even fifteen. In some cases the heart disease may be so perfectly compensated that the child may be comparatively well for a time, and severe disturbances may not appear for several years.

As a rule, children with congenital pulmonary stenosis present a very striking appearance. The cyanosis is especially noticeable in the face, the lips, the nose, and the hands and nails. The parts mentioned feel cool. The eyes are often somewhat prominent, and there is a slight œdematous swelling about them. The peculiar club-like thickening of the terminal phalanges of the fingers and toes, a result of stasis, as seen also in many cases of bronchiectasis, is very characteristic. The nails also present a characteristic claw-like curvature.

The whole development of the child is remarkably retarded. It often seems several years younger than it is. The muscles and fatty layer are slight. The gums are sometimes very spongy and disposed to bleed. In severe cases the child suffers from faintness, vertigo, etc.

On physical examination of the heart, we usually find the cardiac region rather prominent. Percussion gives an increase of the heart's dullness, especially toward the right. This extension of the dullness is due to the hypertrophy of the right ventricle, which must arise in the same way as hypertrophy of the left ventricle in aortic stenosis. On auscultation, we hear a loud systolic murmur, which is perceptible over the whole heart, but which has its greatest intensity at the sternal end of the second left intercostal space. The eddies of blood, which produce the murmur, may also often be felt by the hand as a systolic thrill. In some cases, however, we miss the murmur in pulmonary stenosis, just as in mitral stenosis. The pulmonic second sound is weak or inaudible, or it is replaced by a murmur if there is also insufficiency of the valve.

The course of congenital pulmonary stenosis is always unfavorable. As has been implied, few children get beyond the age of fifteen years. Death ensues, either with general disturbances of compensation evidenced by dyspnœa and dropsy, as in every other form of heart disease, or from complications. Among the latter, we may mention especially the very frequent development of phthisis.

2. **The Remaining Congenital Lesions of the Heart.**—Inasmuch as other congenital lesions than pulmonary stenosis have but slight clinical importance, we will limit ourselves here to a brief review of the same.*

(*a*) Patency of the foramen ovale is a comparatively frequent lesion, whether alone or combined with others. Physical signs are mostly absent. In a few cases a presystolic murmur has been heard. When mitral insufficiency co-exists with a patent foramen ovale, venous pulsation may be caused.

(*b*) Defects in the septum between the ventricles. These are most frequently situated in the posterior section of the anterior septum, and they likewise are associated with other anomalies, such as abnormal distribution of the arteries, pulmonary stenosis, or defects in the septum between the auricles. Sometimes the patency of the septum gives rise to a systolic murmur, but a diagnosis of the condition during life is scarcely ever possible.

(*c*) Persistence of the ductus Botalli. Inasmuch as this contributes blood from the aorta to the pulmonary circulation the pressure in the latter is raised, hence there is to be observed accentuation of the second pulmonic sound and hypertrophy of the right ventricle. There is sometimes also a loud systolic murmur.

(*d*) We have already spoken of congenital stenosis of the tricuspid valve. Congenital narrowing of the mitral valve and of the aortic valves may also occur, but they are extremely rare.

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* A more extensive presentation of the subject can be found in the article by Rauchfuss in Gerhardt's "Handbuch der Kinderkrankheiten," vol. iv, and in text-books on pathological anatomy—for example, Orth's.

9. Combined Valvular Diseases of the Heart

Although in what has preceded we have dealt with the several forms of valvular disease of the heart separately, in order to present them in a general way, yet in reality combinations of them often occur in great variety. We find especially, as has already been mentioned, stenosis of an orifice co-existing with insufficiency of the accompanying valve; but diseases of two or more different valves are not infrequent, combined in the most diverse manners. The physical signs of these "combined forms of heart disease" may, of course, be inferred from the signs of anomalies of single valves, but the phenomena are often so complicated that the diagnosis of combined heart disease is generally much harder than that of the simple forms. Sometimes the single forms neutralize one another in their action. For example, the left ventricle is usually small in pure mitral stenosis, but, if aortic insufficiency be also present, it is nevertheless found dilated, at least to a certain degree. The influence of an absolute or relative tricuspid insufficiency on the action of mitral disease, especially the decrease in tension in the pulmonary vessels caused by it, and also the diminished accentuation of the pulmonic sound, have been mentioned above.

In reference to the clinical symptoms of combined heart disease we may say, on the whole, that, in a large number of cases, the disease of *one* valve stands out as predominant in the whole picture of the disease. The other anomalies are only slightly noticeable, and often of later date. Hence, we may find at the autopsies of patients, who during life have shown the symptoms of disease of only *one* particular valve, unimportant changes on the other valves, which have been without clinical significance.

General Comparison of the most Important Physical Signs in Valvular Disease of the Heart

Form of Heart Disease	Inspection.	Palpation.	Percussion.	Auscultation.
1. *Mitral insufficiency.*	Strong apex-beat, often somewhat displaced outward.	Systolic thrill at the apex. Quite strong radial pulse.	Hypertrophy of the left, later of the right ventricle.	Loud systolic murmur at the apex. Pulmonic second sound accentuated.
2. *Mitral stenosis.*	Area of cardiac impulse enlarged, epigastric pulsation.	Diastolic thrill at the apex. Small and often irregular pulse.	Hypertrophy of the right ventricle.	Diastolic or presystolic murmur at the apex. First sound sometimes loud. Pulmonic second sound accentuated, and sometimes double.
3. *Aortic insufficiency.*	Apex-beat very strong, displaced downward and to the left. Visible pulsation of the medium-sized and smaller arteries.	Very strong, heaving apex-beat. *Pulsus celer.*	Marked hypertrophy of the left ventricle.	Loud diastolic aortic murmur, especially over the upper part of the sternum. Sounds in the arteries (femoral and brachial sounds, etc.). Sometimes a double sound or double murmur in the femoral.
4. *Aortic stenosis.*	Apex-beat displaced to the left.	Heart's action not very strong. Pulse small, infrequent, sometimes slow.	Hypertrophy of the left ventricle.	Loud systolic aortic murmur, transmitted to the right.

[Bramwell reports that of 131 cases with macroscopic valvular lesion, the tricuspid was implicated in 33.58 per cent.; in all but 12 per cent. of these the changes were recent. Hence he thinks that tricuspid endocarditis is generally recovered from, and this he attributes to the relatively small strain to which that valve is subjected. The obvious therapeutic deduction is the importance of rest in mitral endocarditis.]

GENERAL SEQUELÆ AND COMPLICATIONS OF VALVULAR DISEASE OF THE HEART

After having discussed, in what precedes, the mechanism of the single forms of valvular disease, and the physical signs derived from it, we must now describe a number of symptoms and sequelæ which may be present to a greater or less degree in all forms of valvular disease. With them we must also mention certain peculiarities of the individual forms.

1. **Subjective Symptoms.**—Fully compensated heart disease may exist, at least for a long time, without any subjective symptoms. This is especially the case in aortic insufficiency, more rarely in mitral insufficiency. Stenosis of the mitral or of the aortic valves almost always causes subjective symptoms. These symptoms often do not exist so long as the patient keeps perfectly quiet physically and mentally, but they develop under appropriate circumstances, especially upon bodily exertion.

The existing subjective symptoms in heart disease are by no means always referred, in the first place, to the heart itself. It sometimes happens that the patient comes to the physician complaining of various digestive disturbances, or in other cases of headache, vertigo, etc. The physical examination alone leads us to recognize the heart disease. As a rule, the patient's first and chief complaint is directed toward his difficulty in breathing. The shortness of breath, which increases on any physical exertion, comes on quite early in many cases. In the later stages it is almost always the most distressing symptom. The causes of dyspnœa in heart disease are manifold. In the first place dyspnœa results from congestion of the pulmonary circuit, with consequent slowing of the circulation in the lungs, and limitation of the exchange of gases in those organs. In later stages the anatomical changes in the lungs contribute also to an increase of the dyspnœa (*vide supra*, the chapter on brown induration of the lungs). Basch ascribes special importance to the fact that the distended capillaries encroach upon the alveoli, as a result of the passive congestion. This broadens the alveoli ("swelling of the lungs"), but at the same time impairs the mobility of the lungs ("rigidity of the lungs"), and thus embarrasses the exchange of gases in the lungs, just as in emphysema. Further investigations are necessary in order to determine the clinical importance of these conditions.

The secondary bronchitis of heart disease is a very great factor in the dyspnœa. This bronchitis is a frequent result of the pulmonary stasis. Often the respiratory distress increases and decreases simultaneously with corresponding variations in the bronchitis. A purely mechanical cause of dyspnœa is the compression of the lower portion of the left lung by great cardiac hypertrophy. The highest grade of dyspnœa is reached when finally hydrothorax, hydropericardium, and pulmonary œdema are developed. From what has already been stated, it is easy to see why lesions of the mitral valve which directly impair the pulmonary circulation lead sooner to shortness of breath than do lesions of the aortic valves. Finally, it is self-evident that the condition of the heart has the greatest influence upon the degree of dyspnœa present, for all the phenomena of pulmonary congestion must increase or diminish, according to the functional integrity of the heart, and particularly of the left ventricle. If the contractile power of the left ventricle grows less, dyspnœa must at once increase. Not infrequently conditions of cardiac weakness develop with considerable suddenness, and occasion attacks of dyspnœa which are termed cardiac asthma.

Palpitation is the first subjective symptom to be mentioned of those which are referred directly to the heart. It is not yet accurately determined under what circumstances the action of the heart is perceived by the patient himself. We sometimes see an uncommonly strong action of the heart, as in aortic insufficiency, which is not perceived at all subjectively. In other cases, where objectively the heart is not especially active, palpitation forms the patient's chief complaint. It usually first appears when the heart disease ceases to be fully compensated. It is increased or first excited by physical exertion or mental excitement. In many patients attacks of palpitation occur without any discoverable external cause, due apparently to nervous disturbance. They are sometimes associated with a striking acceleration of the pulse, the so-called tachycardia.

Pain in the cardiac region is only rarely present in heart disease. The patients more frequently complain of an indefinite feeling of pressure and oppression in the chest, but still there do occur, particularly in aortic insufficiency, attacks of violent pain in the front part of the chest and the region of the heart, radiating to the shoulders and arms, and associated with a general feeling of extreme anxiety and weakness. Such conditions are termed angina pectoris, or attacks of stenocardia (*vide infra*). Pains in the epigastrium and abdomen, which sometimes form the chief annoyance of the patient, usually depend upon passive congestion of the liver (*vide infra*), or upon the tension of the abdominal walls from ascites, œdema, etc.

We must finally mention here the rheumatoid pains in the joints and muscles, from which many patients with heart disease suffer.

The greatest subjective distress occurs in the last stages of heart disease, when general dropsy develops. The patient's helplessness usually culminates. All motions of the body are difficult, the dyspnœa and oppression in the chest constantly increase, until death finally releases the patient from his mournful condition.

2. **Sequelæ in the Heart Itself.**—We have already discussed the most important sequelæ of valvular disease in the heart itself, its hypertrophies and dilatations. It remains for us to describe the influence of the cardiac disease on the frequency and regularity of the heart's action, and also to discuss some secondary diseases of the cardiac muscle and of the pericardium.

In every well-compensated heart disease the heart's action may for a long time be of approximately normal frequency and regularity. We often find a constant and moderate acceleration of the pulse, which is easily increased from temporary causes. Obviously this increase in the number of heart-beats is often compensatory. If the left ventricle receives during diastole less blood than normal—for example, in stenosis—there is such an increase in the frequency of the cardiac contractions that the same amount of blood reaches the various organs in a given interval of time as before. Diminution of the pulse-rate (bradycardia) is exceptional in valvular heart disease, except when it is artificially caused by large doses of digitalis. It is most frequent in aortic stenosis, and here perhaps it has a compensatory effect (*vide supra*). Marked changes in the rate of the pulse are due to severe disturbances of the nervous apparatus of the heart, and hence they are often associated with arhythmia. Under such circumstances, the pulse-rate may be as high as 120–140 beats per minute. A rare but interesting symptom is the sudden onset of attacks of enormous rapidity of the pulse, up to 160–200 beats and more (tachycardia). This is especially connected with mitral lesions. In the intervals the action of the heart is quiet, with approximately complete compensation of the cardiac lesion. The acceleration of the pulse occurs rather suddenly, and is usually associated with a subjective sensation of palpitation and distress. It may last several hours and then vanish, usually with the same suddenness. The exact cause of these attacks is unknown. They suggest a temporary paralysis of the inhibitory apparatus of the heart. Very often a well-marked acute dilatation may be demonstrated by percussion, during the attack of tachycardia.

Arhythmia of the heart is of still greater importance than anomalies of the pulse-frequency. It always points to a severe disturbance of the nervous apparatus of the heart. The general circulatory disturbance which follows every valvular disease must of course make itself felt in the heart itself, and the nerves and ganglia of the heart can not remain undisturbed by it. Hence we generally see marked variations in the frequency and rhythm of the heart's action along with the other signs of beginning disturbance of compensation; but daily clinical experience teaches us that there is not a perfect parallelism between the two symptoms. We find often enough in heart disease a considerable irregularity of the pulse without any of the other signs of marked disturbance of compensation, and, on the other hand, we see in many patients an almost perfect regularity of the pulse up to death. In mitral disease, especially in mitral stenosis, arhythmia of the heart is much more frequent than in aortic disease.

We can not enter here into a complete discussion of the special varieties and symptoms of arhythmia. A distinction is made between the irregular pulse, in which the successive beats are separated by irregular intervals, and the unequal pulse in which the separate waves are of unequal height. In reality, both phenomena are often associated, for when the pulse is more rapid the left ventricle is imperfectly filled during diastole, and the pulse is consequently smaller. The irregularity of the cardiac activity may be extreme (*delirium cordis*); or it may be so modified as to have a certain periodic regularity of its own (*allorhythmia*). One of the most important and frequent forms of allorhythmia is the *pulsus bigeminus* (see Fig. 42). In this there is first a vigorous systole and then, before the ventricular diastole is completely finished, a second weaker contraction, followed by a pause. We feel alternately a strong and quite weak pulse. The latter may be imperceptible, so that it can be made out only by the sphygmograph. In such cases, with co-existing tricuspid insufficiency, we sometimes find the number of the venous pulsations twice as great as the number of the radial pulsations, because the second weaker contraction of the heart produces a visible venous pulse, but not an appreciable radial pulse. It is possible that in such cases we may have to do with an unequal contraction of the two halves of the heart, the right ventricle contracting vigorously, and the left feebly. If we listen to the heart when there is a pulsus bigeminus we hear four sounds in quick succession, the first two corresponding to the distinct radial pulse, the next two to the close-following systole of the heart which does not give rise to a distinguishable pulse. Continuous and regular pulsus bigeminus, as depicted in the preceding figure, is an infrequent phenomenon, but we very often have a few beats of this character at brief irregular intervals. It is certainly the most frequent cause of the intermittent pulse. The fingers on the radial artery from time to time fail to distinguish any pulse whatever, or else they perceive it merely as a short wave, closely following the preceding pulse; but if we listen to the heart at the same time, we recognize that four audible heart sounds correspond to every intermission of the pulse. On the whole, the pulsus bigeminus is a bad sign, since it always points to a marked disturbance of the cardiac innervation; but it may also pass away again and give place to a regular action of the heart.

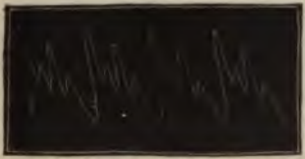
FIG. 42.—Pulsus bigeminus.

Chronic valvular disease of the heart is often combined with anatomical changes in the cardiac muscle, and sometimes in the pericardium.

Among the changes in the cardiac muscle, cloudy swelling, and especially fatty degeneration of the muscular fibers, are the most frequent. The fatty degeneration of the muscle occurs either in a diffuse form, or in little yellowish spots, which are plainly visible on the papillary muscles and trabeculæ. The opinion has often been expressed that fatty degeneration of the muscles is the cause of the disturbance of compensation; that the cardiac muscle performs its increased work until fatty degeneration ensues and reduces its strength. This theory does not entirely correspond to the facts. We have often seen the greatest disturbance of compensation in valvular disease when section of the cardiac muscle showed no fatty degeneration; and, on the other hand, we have seen great fatty degeneration of the heart, as in pernicious anæmia, when there were no signs of cardiac weakness during life. Anatomically, with our present aids to research, we can hardly ever decide with certainty whether the cardiac muscle is still capable of performing its normal functions or not. The usual state of the case is probably this, that fatty degeneration of the cardiac muscle is a result of the disturbance of compensation, and especially of the deficient supply of arterial oxygenated blood to the muscle (see the chapter on anæmia).

There is as truly a passive congestion of the heart as there is a passive congestion of the liver and kidneys. The circulatory disturbance in the heart itself is the chief cause of cardiac insufficiency, and the phenomena of disturbed compensation.

A frequent affection of the cardiac muscle in valvular disease is the presence of cicatricial changes and so-called myocarditic nodules in the substance of the heart. Chronic endocarditis may directly invade the subjacent parts of the cardiac muscles and set up a chronic inflammation there, but the cardiac cicatrices usually have another origin. The connective-tissue thickening beneath the endocardium is the result of a simple atrophy of the superficial muscular fibers due to the increased internal pressure of the blood, as in mitral or aortic insufficiency. The connective-tissue nodules within the cardiac muscle, however, depend in part upon a deficiency in the local supply of arterial blood. Simple sclerotic thickening of the coronary arteries, or complete embolism or thrombosis of a small branch of one of them, is the evident cause of these circumscribed cicatrices. On the other hand, it is also probable that disease of the myocardium is associated with the endocarditis, and is referable to the simultaneous action of the same causes, such as the infection of articular rheumatism, diphtheria, and syphilis. On careful microscopic examination we find, in almost all cases of valvular disease, quite extensive changes in the myocardium (Krehl), and in many cases these may certainly impair the functional powers of the heart. Still, we often find cicatrices of myocarditis without any signs of a previous disturbance in the compensation of the heart. A fuller discussion of this subject will be found in the next chapter.

Pericarditis is not very rare as a result of chronic valvular disease. It is always a dangerous complication, and it may cause death. Regarding its origin, we have found that almost all the cases of heart disease complicated with pericarditis show changes in the aortic valves. Hence it does not seem improbable to us that the secondary pericarditis in such cases is sometimes due to a direct invasion of the pericardium, by the excitants of inflammation passing from the aortic valves through the walls of the blood-vessel.

3. **Symptoms of Stasis in the Different Organs of the Body.**—As has frequently been mentioned in what precedes, the results of stasis of the blood make themselves manifest in heart disease in various organs. We have already spoken of the important results of blood stasis in the heart itself and the lungs. It remains for us to discuss the symptoms of stasis in the systemic veins.

As soon as the flow of venous blood into the right side of the heart is hindered, the venous stasis is shown by the cyanotic appearance of the patient. This cyanosis may exhibit any degree. In heart disease which is still, on the whole, well compensated, it is recognized only by the practiced eye of the physician as a slight bluish tinge of the lips, the alæ of the nose, the cheeks, or the nails. With the increase of the disturbance of compensation the cyanosis increases, if it be not modified by the co-existence of general anæmia. In mitral disease, especially in mitral stenosis, the cyanosis is usually more marked than in aortic disease. The large veins, also, become plainly visible as a result of their complete filling, especially the large external jugulars.

A further important symptom which follows the venous stasis is the œdema, the dropsy of heart disease. As we know from general pathology, every venous stasis, if it reaches a certain grade, leads to a transudation of the fluid of the blood from the capillaries. If the lymphatics can no longer carry this transudation away, it collects in the meshes of the tissues and leads to œdema. The œdema of heart disease, therefore, does not appear until the venous stasis has reached a certain degree, and the compensation of the heart disease has become impaired. It first appears in those parts where there is a particularly loose tissue, as in the scrotum, or where mechanical conditions favor its development. The legs usually swell first, especially about the ankles, because here the stasis of the venous blood is increased by gravity. At first, slight œdema appears only temporarily and by day, and disappears again while the patient is in bed at night; but, as the disturbance of compensation increases, the œdema also constantly grows worse, especially in the dependent parts of the body, until finally it may reach the highest degree of dropsy. Besides the œdema of the skin, transudations into the internal cavities occur, especially into the abdomen and the pleural cavity.

The relation between the degree of cutaneous œdema and the amount of dropsical transudation is variable; thus, in particular, we may have a large amount of ascites, with only moderate swelling of the legs. This is probably occasioned in most cases by special secondary changes in the liver (*vide infra*).

The patient's distress is decidedly increased by marked œdema, as has already been said. All the motions of the swollen extremities are considerably impeded. Hydrothorax and ascites increase the dyspnœa, the former by compression of the lungs, the latter by upward pressure on the diaphragm. The passage of urine may be rendered very difficult by œdema of the prepuce. Besides that, we must mention that the skin, when very œdematous, is quite apt to become the seat of furuncular and erysipelatous inflammations.

The results of stasis in the internal organs may be best seen in the liver, spleen, and kidneys.

Passive congestion of the liver is manifested by quite a considerable increase in the size of the organ. The lower boundary of the liver dullness extends several fingers' breadth beyond the edge of the ribs, and the anterior surface and lower border of the liver may often be plainly felt. The liver may also be enlarged in cases in which there are no other marked signs of passive congestion, such as dropsy. Quite severe pain in the hepatic region sometimes arises from the tension of the capsule of the liver. In later stages the liver may grow smaller again through a partial atrophy of the liver cells (atrophic nutmeg-liver). Indeed, there may even develop, as a result of the chronic congestion, a secondary cirrhosis of the liver, with a distinctly granulated surface. These are the cases in which marked ascites is especially apt to occur (*vide supra*).

Slight jaundice often develops in heart disease, as a result of passive congestion of the liver, or perhaps sometimes from a secondary duodenal catarrh. The peculiar mixture of a cyanotic and slightly jaundiced hue of the skin is very characteristic in many cases, especially in mitral disease. It should be added that the yellowish discoloration of the skin in heart disease is probably not always a genuine icterus, but it may be occasioned by the deposit of other pigments in the skin.

Passive congestion of the spleen arises if the stasis of the blood extends to the splenic vein. The spleen increases in size and becomes firm and dense. It is often hard to make out the congestion from the increase of the splenic dullness, because percussion of the spleen is uncertain if there be also ascites, hydrothorax, etc. We can often, however, plainly feel the enlarged spleen under the edge of the ribs on the left.

In the kidneys we find passive congestion, with the final development of "cyanotic induration." The slowness of the circulation and the diminution of the arterial tension in these organs lead to marked changes in the urine. In particular, there is a diminution in the quantity, especially if the patient is at the same time dropsical. The amount of urine falls as low as 800–500 cubic centimetres (27–17 ounces), or even less, in twenty-four hours. It becomes dark, concentrated, of high specific gravity, and abnormally acid, and hence it usually has an abundant sediment of urate of sodium. In marked degrees of stasis there is albumen in the urine as a result of the damage done to the glomerular epithelium. The amount of albumen is usually slight, but it may equal one third or one fourth of the volume of urine. Under the microscope we find, in the urine of simple passive congestion, only an occasional hyaline cast, and a few red and white blood-corpuscles.

Careful examination of the urine in cases of severe heart disease is of the greatest practical importance, for the character of the urine as shown by the color, specific gravity, and amount of albumen, is an excellent index of the vigor of the heart and the condition of the circulation. Any change for the worse in the circulation is directly shown in a diminution of the amount of urine, and an increase in its specific gravity, or, it may be, in the appearance of albuminuria. Any improvement in the circulation, whether spontaneous or due to remedies, is shown first and clearest by an increase in the daily excretion of urine, and a corresponding diminution in its specific gravity.

We not infrequently meet with acute and chronic nephritis, particularly arterio-sclerotic nephritis, complicating heart disease. It is often no easy matter clinically to form a correct opinion about such cases.

We may in part refer the numerous gastric and digestive disturbances, such as loss of appetite, vomiting, constipation, and diarrhœa, from which such patients often suffer, to the stasis in the blood-vessels of the stomach and intestines; but of course there are not infrequently diseases such as acute and chronic catarrh of these organs, which are to be regarded as complications.

4. **Embolic Processes.**—The slowing of the circulation, and the disturbances in the nutrition of the walls of the vessels, which result from it, often give rise in heart disease to the formation of thrombi. These are situated in the heart itself, on the diseased valves, in the recesses between the trabeculæ, in the auricles, etc., or else they form in the veins, especially in those of the lower extremity. From these thrombi fibrinous plugs may easily be set loose and enter the circulation, and thus give rise to embolic processes in distant organs. Some of the embolisms, whose clinical relations are especially important, have been more fully described elsewhere, and will be mentioned only briefly here.

Embolism of the pulmonary arteries, proceeding from venous thrombi or from thrombi in the right side of the heart, gives rise to hæmorrhagic infarction of the lungs. Its pathogenesis and symptoms have already been discussed in a previous section (see page 273).

Embolism of the cerebral arteries is a common cause of apoplectic attacks, which are not infrequent in heart disease, and usually lead to hemiplegia. The anatomical cause of the hemiplegia in these cases is the embolic softening of the brain which ensues. The details of this are given in the section on cerebral diseases (see page 1103).

Embolism of the larger arteries of the extremities, such as the femoral and the brachial, is much rarer than the forms mentioned. It leads to embolic gangrene of the extremities, unless an adequate collateral circulation can be established. The skin of the peripheral parts, the fingers or toes, first becomes cool, bluish, and at last, if the circulation be wholly checked, almost black. The gangrene advances slowly, usually occupying weeks. Ulcerations develop as the necrotic portions are thrown off. The affection is extremely painful. The patient soon becomes very miserable from the pain and the septic fever that usually attend the ulcerations, and extensive gangrene almost always ends fatally. Sometimes there is embolism of the abdominal aorta. This is usually located at its bifurcation, and occasions a sudden and almost complete paraplegia, speedy disturbance of sensation, abolition of the reflexes, and loss of electrical excitability. No pulse can be felt in the peripheral arteries; the feet are pale and cold, and soon there are signs of gangrene in both legs. The condition is probably invariably fatal.

Embolism of the renal arteries, with consequent anæmic or hæmorrhagic infarctions in the kidneys, may have no clinical symptoms at all, but it is sometimes indicated by sudden pains in the region of the kidneys and marked hæmaturia (see page 644).

Embolic infarction of the spleen is often marked by swelling of the spleen and by severe perisplenitic pains. In other cases it is wholly without symptoms.

Embolism of a mesenteric artery is a very rare event. Its symptoms consist of a sudden intestinal hæmorrhage, of severe colicky pains, general collapse, and peritonitis.

5. **Complications on the Part of the Nervous System.**—The most important complication on the part of the nervous system—embolic softening of the brain—has already been mentioned. We must also state that cerebral hæmorrhage may occur in heart disease. It is especially frequent in aortic insufficiency, either as a result of co-existing atheroma of the cerebral arteries, or perhaps of the abnormally high tension of the vessels during systole.

Mental disorders have been repeatedly observed in chronic valvular disease. They are the result of the disturbance of the circulation, and the consequent impairment of nutrition in the brain. Hence they usually make their first appearance in the last stages of heart disease, at the same time with the other disturbances of compensation. The psychoses in heart disease most frequently have the character of melancholia, but conditions of confusional insanity and excitement also occur.

6. **Secondary Affections of the Joints** are not rare in heart disease. As acute endocarditis develops in the course of acute articular rheumatism, so, on the other hand, rheumatic pains in the muscles and joints, and even acute swelling of the joints, associated with fever, appear in the course of chronic heart disease. Sometimes we have the complete picture of acute articular rheumatism. Probably no fresh infection from without is requisite for the development of these secondary lesions in the joints, but under certain circumstances there is an unusual development of infectious germs (staphylococci?) on the diseased valves, and large numbers of them passing into the circulation occasion a fresh constitutional infection.

7. **Constitutional Symptoms. Fever.**—In congenital and early acquired valvular disease, the general development of the child is ordinarily much retarded. In heart disease in adults, however, we by no means always see an injurious influence on the general nutrition. In many patients we even find a remarkably good development of fat. A marked general disturbance of nutrition, such as great

anæmia and general emaciation, often develops in the later stages, especially in aortic insufficiency. The wasting is of course often hidden by the œdema.

In general, chronic heart disease runs its course without fever, but periods often occur in the course of the disease when there is a moderate and usually irregular fever. Marked disturbances of the general condition may or may not be associated with it. The basis of the fever is probably an acute exacerbation of the endocarditis, except, of course, in accidental complications. All variations occur, from a mild febrile movement without further symptoms to a severe acute recurring endocarditis (*q. v.*). In other cases the fever is connected with secondary swelling of the joints, or with embolic processes.

GENERAL COURSE AND PROGNOSIS OF VALVULAR DISEASE OF THE HEART

The course of valvular disease of the heart is in most cases very chronic. It may last for years. So long as there is a complete compensation, the patient feels almost perfectly well; sometimes he even has no misgivings as to his trouble. The slight difficulty in respiration and the incapacity for physical exertion are noticed, but little attention is paid to them, because the patient is used to them. In other cases there is a moderate disturbance for a long time, but it may be borne quite easily if the patient is rational and prudent in his conduct.

We can not make any general statement as to the length of the stage of compensation, because cases differ very greatly in this respect. It depends in part upon the intensity of the heart disease, in part upon the external conditions under which the patient lives, and in part, certainly, upon the different individual capacity for work and power of resistance of the heart. Thus it happens that many cases last for decades, while in others severe sequelæ appear within a few months. External injurious agencies, acting on the patient, are of great influence on the course of heart disease. Severe physical exertion, an injudicious manner of living, intercurrent febrile disease, mental disturbances, care, and anxiety are often followed by unhappy consequences.

If the first signs of disturbed compensation appear, if severe dyspnœa, slight œdema of the ankles, etc., develop for the first time, these symptoms may disappear again completely under proper treatment. Severe disturbances of compensation even, great general dropsy, associated with very weak and irregular action of the heart, may abate, after a few weeks' duration, and the patient may feel quite well again. Exacerbations of the disease may come on several times and as often improve. Finally, of course, the improvement is incomplete. Persistent œdema and the other results of venous stasis ensue, the symptoms constantly increase in severity, especially the dyspnœa, until the patient dies after a long and distressing illness. Immediately before death in heart disease, certain irregularities in the innervation of the heart and in the respiration sometimes develop. Among these the so-called Cheyne-Stokes phenomenon deserves especial mention. It consists in a peculiar periodical variation in the respiratory movements. There will be, first, a complete pause in the respiration (apnœa), and this will be succeeded by feeble breathing gradually becoming stronger, then gradually abating, and finally ending in another complete cessation of the respiration. The patient usually becomes more comatose during the cessation of respiration; his pupils contract. During the hard breathing the patient recovers somewhat, and his pupils dilate again. The chief cause of this rhythmical breathing is probably to be sought in the decided lowering of the excitability of the respiratory center. During the apnœa a considerable amount of carbonic acid collects in the blood before the respiratory center is sufficiently stimulated to resume its activity.

We have seen the heart behave in a precisely analogous manner, but independently of any Cheyne-Stokes respiration. There was a series of rapid but regular pulsations, alternating with a series of slower contractions.

In regard to the particular forms of valvular disease, aortic insufficiency generally gives the best prognosis, inasmuch as it may be very perfectly compensated for many years, but if severe disturbance of compensation once occurs in this form of heart disease, it gives a very unfavorable prognosis, since, as a rule, we can not reinvigorate the heart. Mitral insufficiency is also quite a favorable form of heart disease, which may be compensated for a long time. Mitral stenosis is decidedly more unfavorable in its prognosis, and is associated with more disturbance; but in all mitral diseases very severe conditions may improve once or even repeatedly. Aortic stenosis is also capable of quite good compensation, and in this respect it is even more favorable for the patient than mitral stenosis, but it often causes persistent cerebral symptoms, such as headache and vertigo, depending on anæmia of the brain, or very likely upon simultaneous changes in its blood-vessels.

Whether established valvular disease of the heart is curable is a question which can not be answered unconditionally in the negative. Of course in the great majority of cases it is in itself incurable; only its sequelæ can be prevented or removed to a certain degree. In children and young people, however, cases sometimes do occur, as we ourselves have seen, in which there are all the signs of a pronounced heart disease, but after a long time recovery is complete. Of course it is very hard to decide whether we really have to do with a valvular disease that has been cured, because simple dilatation of the heart, relative insufficiency of the valves, anæmic cardiac murmurs, etc., may easily be confounded with pure valvular disease of the heart.

Among the dangerous intercurrent accidents in valvular disease we must make especial mention of embolic processes, which may occur suddenly and without warning. The different forms of embolism have been mentioned above, and also the possibility of cerebral hæmorrhage in heart disease. Intercurrent acute diseases, such as typhoid and pneumonia, often, but not invariably, take a very severe and dangerous course in patients with heart disease, because they make increased demands upon the heart.

TREATMENT OF VALVULAR HEART DISEASE

1. *Prophylaxis.*—Our means for preventing the development of heart disease are very limited. To avert endocarditis in articular rheumatism by the present method of treating acute rheumatism with salicylic acid is impossible. The probability of the onset of endocarditis may be lessened only so far as the whole duration of the disease is often considerably shortened by salicylic acid.

We can also do little in the way of prophylaxis against the development of heart disease that is chronic from the start, since the cause of the disease is in many cases wholly unknown to us. Those injurious influences deserve the most attention which may favor the development of arterial atheroma and its consequent chronic valvular disease. The chief factors in this connection are excessive physical exertion and high living, including the use of too much alcohol and tobacco; but the rôle which these play in the development of genuine valvular disease is certainly much less important than their influence upon certain myopathic and nervous diseases of the heart (see the following chapter).

[There is evidence that the alkaline treatment of acute rheumatism lessens the danger of cardiac complications (see page 686).]

2. *Treatment of Compensated Heart Disease.*—If we have to treat a heart disease which already exists, but which is at the same time fully compensated, our treatment must be chiefly hygienic. The patient must be made aware of his heart disease without making him needlessly anxious. He must be told that his further good health depends in great part upon his own conduct, his discretion, and his perseverance. The patient must avoid everything which makes great demands upon the heart, or which may have a directly injurious influence on it. All

violent bodily exertion, too intense mental work, and also all excesses in eating, drinking, smoking, etc., must be avoided. That the physician's directions will often collide with the demands of the patient's occupation, as well as with his favorite amusements and his habits, should not deter the physician from demanding the fulfillment of his prescriptions, at least so far as possible.

Treatment by drugs is usually unnecessary in compensated heart disease. We do not know a remedy which has a directly favorable action on heart disease. The protracted use of iodide of potassium, Fowler's solution, arsenite of antimony ("granules of antimony"), etc., has been recommended. The efficacy of these remedies is very questionable. We can always try them, if a mild disturbance makes a prescription desirable and other remedies are not especially indicated. Beyond this, the physician is usually contented with an endeavor to improve the appetite and nutrition of the patient by means of iron, quinine, and bitters. If there is a suspicion that the heart disease may be due to syphilis, a trial of iodide of potash may be made, but a brilliant result from the employment of antisyphilitic measures is hardly to be expected, because the mechanical imperfections of the valves, leading to regurgitation and stenosis, can scarcely be remedied.

The employment of baths in heart disease deserves special consideration. Numerous experiences go to prove that they are not only well borne by patients with heart disease, but that they exercise a peculiarly beneficial and invigorating influence upon the action of the heart. In this regard, the greatest reputation is possessed by the warm mud baths, which are rich in carbonic dioxide, particularly those at Nauheim. Even where there is incipient failure of compensation, there will be marked improvement upon the use of these or similar baths (e. g., Kissingen). Patients may also employ at home either simple, or, still better, salt baths, at a temperature of 88°–90° (25°–26° R.). Where there is no marked disturbance of compensation, the methodical employment of gymnastic exercises, the so-called Swedish movements, has a certain value in the treatment of heart disease. Regular muscular movements certainly promote the circulation of the blood, and thus lighten the task of the heart. If they are carried out cautiously and with a proper consideration of the individual case, they are not infrequently beneficial. The best measure for the amount of bodily exercise for patients with heart disease seems, to the author, to be the subjective sensation of dyspnœa. Any patient who has no special signs of failing compensation may walk at a slow pace until he experiences distinct dyspnœa. As soon as this happens he must stand still and rest. The author regards it as a great mistake to encourage patients to persist in their efforts regardless of difficulty in breathing. We should not forget that any disturbance of the circulation which occurs must involve the myocardium itself in its influence. That a change of climate may prove beneficial is self-evident. It is particularly appropriate that patients with a tendency to bronchitis or rheumatism should spend the winter South.

3. *Treatment of Ruptured Compensation.*—As soon as the compensatory activity of the heart, in a case of valvular trouble, begins to be impaired, as soon as there is marked dyspnœa, diminution in the excretion of urine, and œdema, we must promptly lighten the burden of the heart by complete bodily rest. Many cases, particularly of mitral disease, are completely restored, even when there are marked symptoms of cardiac embarrassment, by mere rest in bed with simple diet and without any other therapeutic measures; but if the disturbance of compensation is more marked and persistent, the physician must have recourse to digitalis, a remedy which possesses undoubted value when the powers of the heart are impaired. Digitalis has the properties of making the separate beats more powerful, of lowering the pulse-rate by lengthening the diastole, and above all of heightening the arterial tension. Digitalis is therefore indicated in every case of heart disease when there is persistent disturbance of compensation, and if the

pulse is small, of low tension, and, above all, frequent and irregular; the desired effect of digitalis is to make the pulse slower, more regular, and especially of higher tension. Under the influence of the increase in tension thus produced and of the acceleration of the circulation, the disturbances of compensation often disappear in a surprising fashion; there is more abundant diuresis, the scanty, dark, concentrated urine of passive congestion disappears, the daily amount of urine increases, and the urine therefore becomes clear and of lower specific gravity. The œdema then disappears, the dyspnœa ceases, the head becomes free, the general condition improves, and, in brief, there may again ensue a complete compensation of the heart disease. This change is sometimes accomplished in a comparatively short time, in a few days or weeks.

It is very important to prescribe digitalis in a correct manner in order for it to be efficient. Much experience has shown that it is best to give it at regular intervals of two hours, in doses of one grain and a half (gramme 0.1) of the powdered leaves, either in capsules or simply in water; so that the patient receives in the course of a day some five or six powders—that is, eight or ten grains (gramme 0.5–0.6) of digitalis. If the remedy is well borne we may continue it in this manner, but usually it is omitted during the night, to be resumed in the same way on the next day. As a rule, the distinct specific influence of digitalis is evident after the employment of ten to fifteen powders in the course of two or three days. This is recognized by the marked diminution in pulse-rate, the increasing strength and regularity of the pulse, and the improvement in the general symptoms. Instead of the powder we may employ an infusion of the strength of 1 or 2 parts to 150 of water. Of this a tablespoonful must be given regularly every one or two hours. In some cases, particularly if the patient has already become accustomed to the remedy, we may prescribe still larger doses, up to thirty or forty-five grains (grammes 2–3) and even more, daily. But in every case digitalis should be given in regularly repeated doses, so as to obtain by their combined action the full effect of the drug. It is entirely useless to prescribe digitalis in small doses at long intervals. The remedy must never be prescribed unless we can accurately watch the pulse and the heart's action, for only thus can we obtain clear indications as to the further employment or the omission of the drug.

Often some unpleasant incidental effects appear simultaneously with the beneficial influence upon the heart's action. Among these are nausea, vomiting, and specks before the eyes. If the digitalis is omitted these symptoms usually cease, while the beneficial effect upon the heart may last for a long time. With regard to these incidental and disagreeable symptoms, different patients vary greatly. Many bear digitalis very well, others very ill. From a therapeutic point of view it is particularly embarrassing if the nausea, vomiting, and other disagreeable symptoms appear before the digitalis has produced an effect upon the heart; but in such cases we should not be too hasty in abandoning the drug, especially if it is urgently indicated. If the patient can not bear the infusion we should administer the digitalis in powders, or *vice versa.* If the drug absolutely can not be given by the mouth, it should be injected into the rectum in the form of an infusion of the strength of 1 or 2 parts to 100 of water. This should be warmed to the temperature of the body and given, after a cleansing enema has been employed, once or twice a day. The subcutaneous employment of digitalis has also been recommended, in the form of an infusion of the strength of 3 parts to 100, of which two or three syringefuls may be injected daily (♏ 25–40), but with this we have had no personal experience. Sometimes, especially if the patient has not been carefully watched, the cumulative effect of digitalis is shown in severe symptoms of poisoning, such as collapse, very frequent pulse, disturbance of vision, and dilated pupils. Then such stimulants must be employed as camphor, ether, wine, and strong black coffee. How often digitalis is to be used in the case of heart disease can be determined by experience alone. Many patients, particularly cases of mitral disease, may undergo treatment with digitalis twenty to thirty times or more, with the best results. Every time that signs of impaired compensation return, we must try digitalis again. It must be confessed that it will often be necessary gradually to increase the dose. As is the case with so many other remedies, the patient becomes habituated to the drug. There is no maximum dose, and we must find out by experience in each case what the satisfactory amount may be. Some patients become at last genuine "digitalis eaters," and are unable to exist without large doses of digitalis. We have ourselves seen a patient take seventy-five grains (grammes 5) of the powder daily. In very many cases, however, the beneficial effect of digitalis even in the larger doses fails at last to appear. The remedy is no longer borne and it must be abandoned. This usually marks the last stage of the disease.

Not infrequently we see patients with distinct symptoms of passive congestion, in whom the character of the pulse seems at first to contraindicate the administration of digitalis: the pulse is perhaps frequent, but regular and strong; in other cases it is not rapid at all, but yet somewhat irregular; or it may be even infrequent and regular. Particularly in cases of aortic insufficiency is it often very difficult to determine whether we should give digitalis or not. With all patients of this sort it is, in general, worth while to make a proper trial of digitalis, since it may often be beneficial, and yet we should exercise especial caution and vigilance with regard to its effects.

The attempt to employ the active ingredients of digitalis in a pure form (viz., digitaline, digitaleine, and digitoxine) instead of the drug itself, have not yet proved very successful. We should admit that we ourselves, in common with other observers, have seen good results follow the use of digitoxine. This has been introduced quite recently, and is to be given in tablets containing $\frac{1}{250}$ of a grain (one quarter of a milligramme) every one or two hours. It has no special advantages over digitalis, but certainly deserves to be recommended as a substitute for digitalis when that drug is ill borne or ineffectual. Tincture of digitalis, vinegar of digitalis, and other preparations are far less efficient than digitalis leaves, and, in our opinion, may be disregarded. The best opportunity for their employment is when patients with slight disturbance of compensation need to take small amounts of digitalis persistently.

[Digitalis is used more commonly in this country in the form of the tincture. The urine affords a good guide as to the safety of the continuance of the drug; as long as the renal secretion is sufficient in quantity, and increasing rather than diminishing, there is no danger of the toxic effects. It is, consequently, a good plan to follow carefully the twenty-four-hour quantity of urine when this can be done.

There are cases in which digitalis must be taken for long periods, but it should then be given only twice a day, with twelve hours' interval between the doses, unless the patient has ready access to his physician; there is then less risk of toxic symptoms.

In mitral cases, with or without secondary tricuspid regurgitation, where the cyanosis and other symptoms show that the right heart is engorged with blood which it can not propel onward, the relief afforded by venesection, or by a dozen leeches in the hepatic region, may be very great. Until the veins are relieved either in this way or by free purgation, digitalis and stimulants are useless, and a resort to them results merely in a loss of time, and perhaps in the loss of a life which might be saved.]

Caffeine deserves the first mention among the substances having a similar action, which have of late been recommended as substitutes for digitalis by Lépine, Riegel, and others. Given in repeated small doses, a total of fifteen to twenty grains a day (gramme 1.0 to 1. 5), it often slows, regulates, and strengthens the activity of the heart, and also increases the arterial pressure. The salicylate of caffeine and sodium is chiefly used, three to five grains (gramme 0.2 to 0.3) of the powder, and also the benzocitrate of caffeine in the same or smaller doses. The latter is also given subcutaneously. Besides caffeine, we may also mention here adonis vernalis, convallaria majalis, and sulphate of sparteine; but none of these drugs is as reliable as digitalis.

On the other hand, in cases of simple acute or chronic weakness of the heart, the tincture of strophanthus sometimes proves decidedly beneficial. It is given in doses of ten drops several times a day. In many cases it is an excellent way to combine digitalis with some of the drugs already named, or with others, particularly diuretics. Thus, for example, we may often with advantage add one or two parts of the tincture of strophanthus to the infusion of digitalis, or we may prescribe powders containing a grain and a half (gramme 0.1) of digitalis and of diuretine fifteen grains (gramme 1.0) in each dose, or digitalis and calomel (*vide infra*); or digitalis and caffeine. A combination of this sort which is often very efficient is the following: Infusion of digitalis, 30 grains to 5 ounces of water (grammes 2 to grammes 150); sodio-salicylate of caffeine, 30 grains (grammes 2); tincture of strophanthus, 1 drachm (grammes 4); liquor potassii acetatis, 2 ounces (grammes 60); syrupi aurantii, 1 ounce (grammes 30). M. S. A tablespoonful every two hours.

4. *Symptomatic Treatment.*—Some symptoms which often occur in heart disease demand a special description.

Dropsy is a symptom of venous stasis, and disappears if compensation be restored spontaneously or by the use of digitalis. Complete rest in bed and elevation of the swollen parts serve as the chief aid in removing the dropsy. Dropsical patients ought also to change their position in bed frequently, if possible, that there may not be too much œdema collected in the dependent portions of the body. It is a good plan to wrap up the swollen arms and legs with flannel bandages under gentle pressure. Mild massage of the œdematous parts may sometimes be of advantage. The amount of liquid ingested is to be limited, if possible. Of internal remedies, besides digitalis, which should invariably be first employed, and drugs of similar action, the true diuretics are to be considered, such as acetate of potassium, acetate of sodium, and diuretine. They are sometimes ordered in combination with digitalis and sometimes alone, particularly when digitalis cannot be borne or it is not indicated. Calomel is sometimes especially efficient in cardiac dropsy. Its diuretic influence has lately been emphasized by Jendrassik and others. It is prescribed in powders of three grains (0.2 gramme) three to five times a day. Often a very marked diuresis will be caused after one or two days with rapid abatement of the dropsy. The administration of the remedy is stopped as soon as the diuresis begins. It is also omitted if stomatitis develops.

We have already mentioned the advantageous combination of calomel with digitalis (digitalis, one grain and a half, gramme 0.1; calomel, three grains; gramme 0.2), five such powders daily.

In the last stages of heart disease the patient's condition may be particularly distressing from the severe general œdema. It is then justifiable to remove the ascites or hydrothorax by puncture, or to let the œdema drain out by scarification of the skin—long incisions into the subcutaneous cellular tissue—in order to procure relief for the patient. The scarification of the skin, however, is dangerous, and is not to be employed without urgent indications, because erysipelatous inflammation, etc., is apt to ensue at the point of incision. We can recommend little silver capillary trocars (the so-called Southey's trocars), to which a thin rubber tube is attached. By the aid of these trocars we can drain off large amounts of serum. We must always use great cleanliness, however, and the utmost disinfection of the skin, wrapping the parts in sterilized gauze. In patients with heart disease it is not, as a rule, advisable to attack the dropsy by sweating, by hot packs, or pilocarpine.

The dyspnœa of heart disease is usually the most distressing symptom of all. Here, too, our chief task is of course to restore the compensation; but this failing, we must try to relieve the dyspnœa symptomatically. Morphine is most efficient in this respect. In general, morphine is, next to digitalis, the most indispensable remedy in the treatment of severe heart disease. It is usually well borne, and procures great relief, especially if given subcutaneously. If we have to do with the last stage of the disease, we need not spare large doses. Otherwise, of course, caution is necessary.

Chloral hydrate should be cautiously employed in heart disease. It is often, however, well borne and gives the patient rest and comfort. We have also prescribed chloralamide in cardiac cases, with good results.

In practice we must often prescribe external applications to the chest, mustard-plasters, hot poultices, and also hot foot-baths with mustard, ashes, etc. In severe cases their action is slight. Acetate of lead in large doses sometimes seems to have a favorable influence in severe dyspnœa, especially with threatening pulmonary œdema. We give the powder, up to a grain and a half (gramme 0.1), every two or three hours, and it is often a good plan to add half a grain or a grain (gramme 0.03 to 0.05) of opium. We can also frequently obtain decided relief for the patient, particularly if constipated, by a vigorous drastic purge, with compound infusion of senna or gamboge. Inhalations of nitrite of amyl seldom have a beneficial effect.

Palpitation, constant or paroxysmal, is treated by applying ice to the cardiac region; the "heart-bottles," made of tin, act very well. In patients with aortic insufficiency and very excited action of the heart, we may recommend the protracted use of ice. Hot poultices also are sometimes beneficial. The narcotics are the most efficient internal remedies, especially morphine, which, of course, we should reserve for severe cases. If the palpitation is of a lesser degree, we may try bromide of potassium, or bitter-almond water.

The subcutaneous use of morphine is again by far the most potent remedy in the anginous attacks, associated with pain and a feeling of distress. We may also use cutaneous irritation, mustard-plasters, etc., hot compresses or poultices, and perhaps strophanthus, nitrite of amyl, or nitroglycerine.

We may prescribe bitter remedies—tinctura amara (P. G.), or compound tincture of cinchona—and muriatic acid for the loss of appetite, in case this is not improved by regulating the activity of the heart. In addition, we must always endeavor to get a regular evacuation of the bowels.

For the attacks of faintness and vertigo, occurring especially in aortic stenosis, as a result of cerebral anæmia, we may prescribe a horizontal position, and stimulants—wine, ether, and Hoffmann's anodyne. If the cerebral symptoms depend upon venous stasis, we try to remove it by ice, mustard-plasters to the neck, and thorough derivation to the intestines.

Especial accidents and complications, like pulmonary œdema, infarctions, or apoplexy, are to be treated according to the usual rules.

CHAPTER III

DISEASES OF THE MYOCARDIUM

Preliminary Remarks.—In valvular disease of the heart a disturbance of the circulation takes place because of the functional impairment of the valves, or the obstruction of the orifices of the heart; but in the diseases which we are now to consider we have to deal with impairment of the structure and functional activity of the cardiac muscle itself, while the valvular apparatus of the heart remains intact. The expression myocardial diseases of the heart, in a strict sense, is too narrow, inasmuch as we probably have diseases of the ganglia and nerves of the heart associated with the muscular diseases. Indeed these nervous lesions may often be the essential change, but as yet our knowledge with regard to the pathological changes in the nervous apparatus of the heart is extremely limited; and, on the other hand, most of the organic diseases of the cardiac nerves probably involve some secondary changes in the cardiac muscle. We may therefore, in the classification and consideration of these conditions of the heart, limit ourselves provisionally to the disturbances of the cardiac function observed during the life of a patient, and the anatomical changes in the muscle of the heart found after death. It remains to be discovered later what is the part which the degenerations of the nerves and ganglia of the heart have in the development of these disturbances. We must also point out that in what follows we shall consider merely the primary muscular diseases of the heart, in which the change in the heart is the cause of all the essential symptoms. We shall quickly find how difficult it is in these matters invariably to draw a sharp distinction between secondary and primary disease.

1. Interstitial Myocarditis

(Fibrinous Degeneration of the Myocardium. Sclerosis of the Coronary Arteries)

Ætiology and Pathological Anatomy.—The anatomical changes of interstitial myocarditis result in irregular-shaped spots of a whitish luster, often very numerous, interspersed through the cardiac muscle and corresponding to a partial or complete destruction of the muscle-fibers and the substitution for them of a firm, fibrinous, cicatricial connective tissue. These places, which are best found by making horizontal sections of the myocardium, occur particularly in the left ventricle, and especially at its apex and in its anterior wall; but they may also be found everywhere, and especially in the papillary muscles. Often we may observe them as somewhat glistening retracted spots on the endocardial or pericardial surface of the heart. The origin of this interstitial myocarditis was explained by the researches of Weigert, Ziegler, Huber, and others. In a great majority of cases, though perhaps not in all, the changes just sketched are directly associated with changes in the coronary arteries and their branches. In almost every case we find in these vessels marked changes due to endarteritis, or arterio-sclerosis. In places where this change in the vessel causes marked diminution of its lumen, the corresponding portion of the myocardium is imperfectly supplied with arterial blood, and in consequence the muscular fibers gradually degenerate, lose their nuclei, and change into a friable, cheesy detritus. In place of the destroyed muscular fiber there is a new growth of connective tissue. With ordinary endarteritis these processes are slow and gradual, but under certain circumstances there may be a somewhat rapid occlusion of the branches of the coronary arteries, because of thrombosis or embolism of proximal origin. In such cases we have a genuine cardiac infarction, presenting a spot of anæmic necrosis, or sometimes a rather fresh brownish-yellow hæmorrhagic infiltration.

Of course, this limited necrosis and interstitial new growth will not occur if, in spite of the existence of arterio-sclerosis, the direct supply of blood remains sufficient, or, if insufficient, is eked out by the collateral circulation.

If the formation of connective tissue is extensive, the entire heart wall may become decidedly thinner, so that it yields to the internal pressure of the blood. This sometimes occasions in the left ventricle the bulging of a limited portion of the wall of the heart, so-called cardiac aneurism. A cardiac aneurism of this sort, and also an extensive fresh infarction, may exceptionally result in rupture of the heart with escape of blood into the pericardium, and sudden death. Of still greater importance, because far more frequent, is the formation, in places where the fibrinous process reaches to the endocardium, of parietal thrombi inside the heart. These sometimes give rise to embolism in distant organs.

As to further pathologic changes in the heart we find that certain portions of the heart are not infrequently dilated or hypertrophied. The dilatation may, at least in part, be due to the diminished resistant power of the cardiac walls, but in case of hypertrophy we must always seek for special causes, inasmuch as sclerosis of the coronary arteries as such can not lead to hypertrophy of a portion of the heart. As a rule, the cause is easily discovered, either in an associated arterio-sclerosis, or in those factors, such as dissipation, which may occasion, simultaneously with arterio-sclerosis, an idiopathic hypertrophy of the heart. Of course, we must also bear in mind the possible but exceptional presence of such complicating diseases as interstitial nephritis, or pulmonary emphysema. With regard to the right ventricle we know, also, that this portion of the heart must hypertrophy as a result of congestion in the pulmonary circuit, when the left ventricle is permanently weakened.

The causes of this most important, and by no means rare, form of myocarditis which we have just described, and which is due to sclerosis of the coronary arteries, must be the same as the causes of this latter affection. Often the sclerosis of the coronary arteries is merely a part of a general arterio-sclerosis, but we sometimes find comparatively marked changes in the coronaries, although there are no specially extensive atheromatous changes in the other arteries of the body, and again, when there is well-marked arterio-sclerosis elsewhere, the coronary arteries may show slight symptoms of disease, if any. In general, however, we have the same ætiological factors to explain sclerosis of the coronary arteries as arterio-sclerosis in general. Often we shall find the cause in high living, and particularly in chronic alcoholism. In other cases, habitual muscular overstrain seems to favor the development of arterio-sclerosis, and clinical experience shows that an important cause of sclerosis of the coronary arteries, particularly, lies in great mental effort and excitement, which indeed are not infrequently combined with the above-mentioned factors—for example, in the case of extraordinarily active business men, speculators, high officials, and physicians. All these considerations go to explain the fact that sclerosis of the coronary arteries is seen far more often in the male than in the female sex. That age plays an important part is universally acknowledged. It is true of interstitial myocarditis, as of arterio-sclerosis in general, that, as a rule, persons are attacked in the second half of life, from forty years of age on. Finally, we must add that hereditary tendencies to the development of arterio-sclerosis in general, and sclerosis of the coronary arteries in particular, can not be denied.

That form of disease of the coronary arteries deserves special mention which is of syphilitic origin, and therefore somewhat distinct from ordinary arterio-sclerosis, although the number of proved cases is not great. It is scarcely possible to doubt that there is a specific syphilitic endarteritis of the coronary arteries, giving rise to almost exactly the same symptoms as ordinary arterio-sclerosis. At any rate, this point should in every case be considered, for therapeutic reasons if for none other (*vide infra*).

Finally, it is to be added that there may be another form of chronic myocarditis not associated with primary chronic disease of the arteries, but with an antecedent acute myocarditis as a sequel of severe constitutional infectious disease, such as typhoid, diphtheria, sepsis, or scarlet fever. The careful histological examination of the myocardium by Krehl, Romberg, and others, in cases of disease of this sort, has demonstrated acute inflammatory processes in the cardiac muscle. It might very well be that chronic interstitial myocarditis, with all its results, should gradually develop from acute processes of this sort. Still, the matter needs further clinical study.

There is a secondary chronic myocarditis as a sequel of chronic endocarditis (valvular disease), but this is only exceptionally of any importance in itself. The endocarditic process may extend directly to the neighboring muscular layers of the heart, or there may occur, especially in association with aortic endocarditis, embolic infarctions, the development of which is the same as of the previously described thrombotic infarctions of the heart. Finally, there is the possibility that myocarditis and endocarditis might appear simultaneously. It has been maintained that myocarditis may develop as a sequel of acute articular rheumatism. The development of myocarditis in this way, if it ever occurs, is probably very exceptional.

Clinical History.—We must first mention that sometimes quite extensive cicatricial formation may be found in the cardiac muscle *post mortem*, without the occurrence of any manifest symptoms referable to the heart during life. We see, then, that the heart may, under some circumstances, undergo quite a considerable loss in its contractile substance without injury.

In many other cases, however, the heart's capacity for work suffers so much that the same symptoms arise as in valvular disease. The course of such cases may be very chronic. The symptoms begin quite gradually. The patient first has a slight dyspnœa or palpitation, and a feeling of distress in the chest, but only from external causes, such as slight physical exertion. Sometimes there is marked general weakness and languor. The patients have an unhealthy sallow look. They become easily tired, feel depressed, and scarcely capable of any great bodily or mental exertion. The symptoms gradually increase, and just the same results of disturbance of the circulation appear as in all the other forms of heart disease. The difficulty in breathing becomes more marked, œdema occurs, signs of stasis in the liver, intestines, and kidneys appear—in short, the well-known type of general circulatory disturbance develops.

Physical examination of the heart shows marked anomalies of the heart's action in all severe cases. The pulse is often irregular in regard to its rhythm and the intensity of its single beats, but the arhythmia may also be wholly absent in spite of the degeneration of the myocardium, as we have often convinced ourselves. The pulse is at first quite strong and full, later it becomes weaker, of lower tension, and at last sometimes very small and scarcely perceptible. It may be increased in frequency, but we quite often notice in chronic myocarditis, especially in the early stages, a very characteristic and persistent slowing of the pulse to 60, 50, or even less, in a minute. With this slowness of the pulse there is also frequently irregularity of the heart's action, especially the appearance of occasional double beats (pulsus bigeminus). Percussion usually shows an increase of the heart's dullness, due to dilatation or hypertrophy of the heart, the increase being either general or chiefly on one side. Auscultation shows the absence of any murmur, and hence the absence of valvular disease. The heart-sounds are distinctly audible, and sometimes quite loud and valvular, but in the later stages often low and obscure. The pulmonic second sound is accentuated, when there is stasis of the pulmonary circulation. In several cases we found the second sound for a long time very plainly divided—reduplicated. We must also mention that sometimes in pure myocarditis a systolic murmur is heard at the apex which is due either to a relative insufficiency of the mitral valve, or to its incomplete closure, as a result of defective muscular action of the left ventricle.

We must make particular mention of one symptom of sclerosis of the coronary arteries, which, although it is not pathognomonic, is by far most often seen in association with it and therefore has an important diagnostic significance. We refer to the attacks of so-called *angina pectoris* (stenocardia, heart-pang). These attacks of genuine angina pectoris (compare also the following chapter) consist in the sudden occurrence of pain in the region of the heart, extending into the back, the left shoulder, and the left arm even to the tips of the fingers. In severe cases the pain is extremely violent. It is as if the chest were squeezed in a vise. At the same time there is a decided feeling of anxiety and oppression, as well as complete loss of strength—a sense of impending death. The patient seeks some support, he can scarcely move, scarcely utter a few words in a whisper; the extremities grow cool, the brow moist and cold. The attack may be directly fatal, but this is the exception. As a rule, the symptoms pas away in a few hours, and the patient gradually recovers. In many cases of sclerosis of the coronary arteries attacks of this sort occur very often, in varying severity. Not infrequently an attack may be due to some special cause, such as the bodily exertion of a long walk or climb, errors in diet, or mental excitement. In such cases bad news may cause death.

With regard to the true essence of angina pectoris, we are confined to hypothesis. The ordinary supposition of a sudden paralysis of the left ventricle explains many symptoms, but not all. The intense pain indicates irritation of the sensory nerves. Sometimes during an attack the pulse grows small and irregular, but often also, as we have learned by experience, it may not be very greatly disturbed. There may or may not be, during an attack, acute dilatation of the heart.

Besides genuine angina pectoris, attacks of cardiac asthma are not infrequent in chronic myocarditis. This differs from angina pectoris in that the peculiar pain and constriction are not present, while the distress for breath, the paroxysmal dyspnœa, is prominent. In most cases of this sort we probably have really to do with sudden weakness of the heart. Another symptom that has been repeatedly observed in sclerosis of the coronary arteries is simple syncope, due to acute anæmia of the brain, and perhaps also referable to simultaneous sclerosis of the cerebral arteries.

The general course of the disease varies considerably in different cases. Much depends upon the patient—e. g., upon his ability to take good care of himself. Sometimes dyspnœa, œdema, and other general symptoms of passive congestion are prominent, and in such cases the patient is alternately better or worse. Other cases are characterized by attacks of angina pectoris. The termination is invariably unfavorable. It may either occur gradually with increase in the circulatory disturbance, or with complete suddenness like a stroke.

This important clinical fact of sudden apoplectiform death ("paralysis of the heart") in patients with sclerosis of the coronary arteries, demands consideration. It usually happens in elderly persons in comfortable circumstances and good livers, who up to that time have not regarded themselves as really ill; but they have repeatedly had slight attacks of vertigo, of oppression, etc. Suddenly a sort of apoplectic attack comes on, often after some definite cause, after a banquet, or after some physical exertion or mental excitement. Death follows in a few moments, or after a deep coma that lasts for several hours or even days. The diagnosis often remains in doubt in such cases, especially if we have not known the patient previously. The autopsy shows, as the sole pathological lesion, a sclerosis of the coronary arteries, with a more or less extensive cicatricial formation in the heart. Apparently in these cases the moment must suddenly arise when the supply of blood to the heart is insufficient, and thus death is caused. Experiments upon artificial closure of the coronary arteries, by Cohnheim and others, agree perfectly with the clinical facts above mentioned. Artificial narrowing of the coronary arteries may also be well borne for a long time, until suddenly both halves of the heart stand still in a condition of diastole. In such cases, also, the heart seems to stop suddenly, but sometimes there are frequent anginous attacks, and death occurs in one of them. Finally, it should be mentioned that sudden death in sclerosis of the coronary arteries may also be caused by embolism of the trunk of the coronary artery, or, as in one case seen by the author, by the bursting of a focus of myocarditis with hæmorrhage into the pericardial cavity.

Diagnosis.—The diagnosis of chronic myocarditis is by no means always easy and certain. We must first determine that there is a cardiac lesion of some kind. This is usually evident from the secondary symptoms of stasis, the condition of the pulse, the area of cardiac dullness, etc. Then the question arises whether we have to do with a valvular disease or with a myopathic disease of the heart. Here auscultation must chiefly decide. The absence of a heart murmur, in spite of other definite signs of heart disease, speaks against valvular disease, but not with complete certainty. All murmurs may be absent in the last stages, especially with a high degree of mitral stenosis, and hence we may easily confuse mitral stenosis with myocarditis, particularly when there is marked arhythmia of the heart. On the other hand, we have already stated that in pure myocarditis, with the valves intact, functional murmurs may be present, which may lead to an erroneous opinion as to valvular disease. If continued study of the case has enabled us to exclude valvular disease, it remains to distinguish between chronic myocarditis or sclerosis of the coronary arteries on the one hand, and the other diseases of the myocardium (*vide infra*) on the other. We regard it as impossible to make this distinction with absolute certainty. All the diseases named present the same clinical picture of cardiac insufficiency, but what the anatomical conditions may be which occasion this cardiac insufficiency we can at present only conjecture with more or less probability, during life. The following symptoms are most characteristic of coronary sclerosis: persistent infrequency (slowness) of the pulse, particularly if associated with arhythmia; attacks of genuine angina pectoris; and the evidence of arterio-sclerosis in the brachial, radial, temporal, and other peripheral arteries. Persistent rapidity and arhythmia of the pulse occur in interstitial myocarditis, just as in other myopathic diseases of the heart; but arhythmia is, in general, far more common when there is actual myocarditis than when there is simple muscular weakness of the heart. There is self-evident difficulty in the diagnosis of cases with sudden apoplectiform paralysis of the heart, and in distinguishing them from apoplexy, embolism, pancreatic hæmorrhage, and similar causes of sudden death.

Prognosis.—The prognosis is evident from what has been already said. Recovery is impossible, but even extensive cicatricial formation in the heart may probably last for years without causing much disturbance. We must always be prepared for disturbances of compensation, and the manifold sudden accidents to which patients with myocarditis are exposed, but we can not foretell the time of their occurrence.

Treatment.—The treatment of chronic myocarditis must be directed first to the dietetic and hygienic care of the patient. This is of the greatest importance. For obese persons accustomed to high living, a moderate, simple diet must be accurately prescribed. Alcoholic beverages must be greatly limited or wholly forbidden, and not more than two or three cigars allowed per diem. Moderate bodily exercise is beneficial, and indeed necessary, for the promotion of the circulation and the more rapid diminution of the obesity, but the patient must be earnestly warned against too violent exertion, nor can great mental effort be permitted. In summer, a quiet life in the country or some mountain region is to be advised, or, under suitable circumstances, the cautious use of the waters of Carlsbad, Kissingen, Marienbad, or Nauheim. Frequent tepid baths or mud baths may usually be employed even at home with advantage. Of internal remedies, iodide of potassium has been specially recommended to be used persistently, in the amount of eight to fifteen grains (gramme 0.5–1.0) or more daily. Iodide of potassium enjoys a reputation for an especial specific action upon arterio-sclerosis in general, and ought therefore to have a beneficial effect upon arterio-sclerosis of the coronary arteries. We have ourselves seldom seen very striking results from its use. Probably iodide of potassium is of benefit in cases of syphilitic origin, and we therefore recommend it particularly when there is any suspicion of a syphilitic taint. With disturbance of compensation, and with abnormally frequent, weak, and irregular action of the heart, digitalis and similar remedies are indicated, just as in valvular disease. In cases with an abnormally slow pulse, we may use them, provided great caution is exercised, but we must also be governed by the other prevailing symptoms. In attacks of angina pectoris (see the following chapter) the subcutaneous injection of morphine is by far the most efficient, and often an indispensable remedy. Also, the frequently enumerated cardiac stimulants, such as strophanthus, camphor, ether, and wine, ought to be borne in mind, and, finally, one may make trial of nitro-glycerine ($\frac{1}{60}$ to $\frac{1}{30}$ of a grain, gramme 0.001–0.002) in alcoholic solution, and of nitrite of sodium (two or three teaspoonfuls daily of a solution containing 1 or 2 parts to 120 of water). The inhalation of a few drops of nitrite of amyl is also sometimes beneficial, but more often it has no effect. In cardiac asthma, stimulants are indicated, but often narcotics as well. Mustard-plasters, the application of cold and of heat, hot foot-baths, etc., are also employed. Sometimes even in the worst cases we see good results from the use of digitalis.

With regard to regimen and mechanical treatment compare the following section on idiopathic hypertrophy of the heart.

2. So-called Idiopathic Hypertrophy of the Heart

(Mechanical Strain of the Heart)

Ætiology and General Pathology.—Not infrequently cases during life present all the symptoms of an uncompensated cardiac lesion, and yet the autopsy discloses merely hypertrophy of the heart with a greater or less degree of accompanying dilatation. There is no lesion of valves, coronary arteries, or myocardium. The cardiac hypertrophy, which involves the left ventricle chiefly, but often both ventricles, can not be regarded as secondary in the ordinary sense of the word, for in the heart itself and in the other organs we find nothing which can call forth a secondary hypertrophy of the cardiac muscle—no valvular disease, no chronic nephritis, no general arterio-sclerosis, and no pulmonary emphysema. Hence we term these cases "primary idiopathic" cardiac hypertrophy, in the sense that we can not discover any other primary disease.

Notwithstanding, we must in these cases also seek the cause of the cardiac hypertrophy in some excessive demand upon the cardiac muscle, and as there are no macroscopic anatomical or mechanical changes involving an increase in the cardiac activity, we must seek other circumstances occasioning a functional overstrain of the heart. Careful observation and investigation of cases of this sort have taught us a number of such causes.

Among the most frequent is habitual embarrassment of the circulation by excess in eating and drinking. There can be no doubt that every time food is taken in large amount there is a temporary increase in the activity of the heart, the frequency of the pulse, and the tension in the arteries. This is in part because the specific gravity of the blood is temporarily increased by the material taken into it, and in part because the products of metabolism in the blood have a direct stimulating action upon the heart or the blood-vessels, and finally, because an excessive ingestion of liquids temporarily increases the total amount of blood. In brief, there are numerous persons of the more favored classes who indulge too freely in eating and drinking, and for years go to excess in the pleasures of the table; and in the case of such persons there is, during a great part of their life, although not all the time, that condition which the older physicians termed plethora. All these circumstances which we have mentioned occasion an increase in the demands upon the heart, especially its left ventricle. The heart performs this extra work laid upon it, and consequently becomes gradually hypertrophied. From what has been said, it is easy to understand why this form of cardiac hypertrophy is especially frequent in the obese, and particularly in men from forty to fifty years old, though sometimes much younger. We have seen by far the most frequent examples of it in great beer-drinkers, and so in persons whose business tempts them to excessive indulgence in beer, such as landlords, brewers, hop-dealers, butchers, and many others. Persons of this sort often drink for years, almost daily, four or five, or even eight or ten, litres (quarts) of beer. Let the reader consider what an amount of liquid, and at the same time of nourishment (for one litre of beer contains about fifty or sixty grammes [two ounces] of carbohydrates), is in this way alone introduced into the blood. This explains the great frequency of cardiac hypertrophy which Bollinger has shown to exist at Munich; but the "Munich beer-heart" is seen with unfortunate frequency in other towns than Munich. The alcohol is probably not a factor in the development of the hypertrophy of the heart, but we may well suppose that it promotes, or at least hastens, the degenerative changes in the cardiac muscle, and particularly in the cardiac nerves, which at last render the heart's action inadequate and the circulation imperfect. For the development of cardiac hypertrophy in itself is a circumstance which does not impair the health, but rather preserves it; only, experience teaches us that no heart can uninterruptedly perform an amount of work which is physiologically excessive, and escape final exhaustion—the point of time depending upon the individual vigor and other associated factors.

A second cause of idiopathic hypertrophy is persistent muscular strain. Every muscular exertion increases the demands upon the cardiac activity. There is an increase in the frequency of the pulse and in the amount of blood passing through the heart. If excessive bodily exertion is habitual for a considerable period of time, there finally develops hypertrophy of the heart, usually affecting both ventricles, but especially the left. Thus is explained the hypertrophy sometimes seen in blacksmiths, locksmiths, baggage-carriers, laborers in vineyards ("Tübingen heart"), and soldiers after severe campaigns. Why this hypertrophy should sometimes occur and sometimes be absent, under apparently similar conditions, must be explained by the difference in the physiological capacity of the individual. Here, too, the abnormality of the condition does not become evident till the functional ability of the heart begins to fail ("heart overstrain," "irritable heart").

In the third place, it seems that in some few cases of idiopathic hypertrophy of the heart, in which none of the causes yet named are operative, it is abnormal nervous irritation of the heart which increases its activity, and hence, finally leads to its hypertrophy. This is perhaps the explanation of many cases of cardiac hypertrophy in persons who have long been exposed to many sorts of psychical excitement. The cardiac hypertrophy of exophthalmic goître (*q. v.*) may also be put in this category. Not infrequently we may conjecture in an individual case that several of the causes mentioned have been operative simultaneously in the development of the hypertrophy. Thus, idiopathic hypertrophy of the heart is seen particularly often in individuals who have to undergo great mental strain and excitement, and at the same time are heavy drinkers.

There is usually associated with the hypertrophy a dilatation, referable to the persistent over-distention of the ventricles during diastole. The weaker and more yielding the myocardium becomes with the course of time, and the more passive congestion affects the heart itself (particularly the auricles and right ventricle), the more the dilatation increases; and with it comes degeneration of the cardiac muscle, or, at any rate, functional weakness of the same, giving rise to symptoms of cardiac insufficiency.

Clinical History.—Idiopathic hypertrophy of the heart may certainly exist for a long time without causing the patient any subjective disturbance. We have already emphasized the fact that it is precisely the cardiac hypertrophy which protects the patient for a time from the onset of marked symptoms. Prodromata may occur for a long time before severe disturbance sets in. Among these are palpitation, a certain sense of discomfort in the cardiac region, and slight dyspnœa. The symptoms begin when the heart can no longer respond to the demands made upon it, and when it begins to fail. Then all the symptoms of cardiac insufficiency arise, in just the same way as in valvular disease and in chronic myocarditis. Hence we need not go into the details of the disturbances of compensation again. The whole series of symptoms of stasis, as well as the attacks of angina pectoris and cardiac asthma, described in the preceding chapter, also occur in idiopathic hypertrophies and dilatations of the heart.

Another subjective disturbance is the peculiar and often very annoying feeling of "fluttering" of the heart—that is, a momentary feeling of fluttering contraction of the heart. This symptom indicates, probably, abnormal conditions of contraction, and must by no means be confounded with the numerous sensations which hypochondriacal and neurasthenic patients refer to the heart. If the action of the heart is irregular, and especially if there is a tendency to bigeminal pulsation, many patients feel every bigeminal contraction as a jolt or stopping of the heart.

The general course of the disease differs considerably in individual cases. Sometimes there is moderate difficulty in breathing for a long time, especially on any physical exertion. The patient often complains of great languor, or nervous irritability, and sometimes of attacks of vertigo and faintness, and a tendency to perspiration. The appetite is poor, and there is very apt to be constipation. The condition may become quite suddenly worse after any marked injurious influence, especially after great physical exertion or mental excitement. Physical examination discloses all the signs of hypertrophy of the heart. In the first place, the left ventricle is usually enlarged, corresponding to the direct causes of the condition. This enlargement is often better appreciated by accurate observation of the apex-beat than by percussion. Later the right side of the heart also becomes hypertrophied; dullness on percussion then extends over the lower part of the sternum, with epigastric pulsation and undulatory movements in the veins of the neck. The heart-sounds are pure, at first valvular, later sometimes muffled and feeble. We lay especial value on the characteristics of the first sound of the heart. A dull, indistinct sound is probably a sign of considerable dilatation, while a normal valvular sound indicates a vigorous systole. In general, there is persistent frequency of the pulse, at least in patients who are led to seek the physician because of beginning symptoms. Infrequency of the pulse probably indicates, in most cases, co-existent sclerosis of the coronary arteries (*vide supra*). The frequent pulse may for a long time remain regular, but it is not always so. So long as the left ventricle does its work well the pulse may be tense, and the second aortic sound valvular; but if the heart becomes insufficient the pulse grows small, feeble, often irregular; the heart-sounds grow faint, and not infrequently that bad sign appears which is known as *bruit de galop* or gallop rhythm, and consists in a peculiar reduplication of the first sound of the heart; * for each beat of the pulse we hear three sounds over the heart. At the same time the dyspnœa and oppression in the chest increase, the amount of urine diminishes, and œdema appears in the legs. We now have the complete picture of an uncompensated heart disease. With proper treatment the symptoms may disappear again; but, sooner or later, they return. Death finally ensues from general dropsy or from some complications or intercurrent attacks, among which we may mention embolic processes.

Cases of sudden death (paralysis of the heart) from heart-failure occur not very infrequently, as we know from experience, in idiopathic hypertrophy of the heart independently of chronic myocarditis.

Diagnosis.—It is by no means an easy task for the physician to make an absolute diagnosis of idiopathic hypertrophy of the heart. Sometimes, when the subjective symptoms suggest an examination of the heart, it is easy to make out an increase in the cardiac dullness and a displacement of the apex-beat; but in other cases the presence of emphysema, or obesity, or even of dropsy, makes the physical examination very difficult. If an enlargement of the heart has been demonstrated, we must then exclude valvular disease, especially mitral stenosis, and also secondary hypertrophy, as a result of chronic interstitial nephritis, or other causes. It may be difficult to rule out nephritis if the patient is suffering from passive congestion when he first comes under observation, for then it is often hard to say whether the albuminuria which may be present is to be referred to actual renal disease, or to passive congestion. Even if we can exclude nephritis we have still to determine whether there is simple hypertrophy of the heart, or an interstitial myocarditis. To settle this question is, as we have already said (page 344), very difficult. Ætiology is always important—for example, if there has been an excess in beer, or in muscular exertion. Frequent attacks of angina pectoris, arhythmia, and a lowering in the rate of the pulse, suggest sclerosis of the coronary arteries. Not infrequently diseases of the myocardium are confounded with chronic obliterative pericarditis. When we consider this latter condition we shall refer again to the differential diagnosis.

Treatment.—The main points in the treatment of idiopathic hypertrophy of the heart are, in general, exactly the same as for valvular disease and interstitial myocarditis. We may, therefore, refer the reader for these to the preceding chapters. There is, however, one point which we would like to discuss in more detail here, because it is especially prominent in the treatment of diseases of the myocardium. We refer to the so-called dietetic and mechanical treatment of circulatory disturbances. Much of what we shall say here applies also to other forms of chronic heart disease.

Efforts have long been made, but with fresh zeal of late years, because of Oertel's recommendation, to promote and improve the circulation by mechanical means as well as by such internal remedies as digitalis. The following considerations lead one to regard such an influence as possible:

1. *Diminution of the Amount of Fluid in the Body, and particularly in the Blood* (*“Desiccation”*).—This is the point upon which Oertel lays the greatest

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* This sign is not infrequently found in idiopathic hypertrophy of the heart, in myocarditis, and particularly in secondary cardiac hypertrophy following chronic nephritis. It has not yet been fully and definitely explained. Perhaps we have to do with an audible muscle-sound originating in the auricle, perhaps with peculiar irregularities in the contraction of the heart [for example, the contraction of the right and left ventricles not being simultaneous.—V.].

stress. He believes that he can diminish the amount of blood in the body by withdrawing liquids, and thus lighten the task of the heart and restore the normal circulation. On this view rest the proscription of excessive drinking and the limitation of liquid nourishment, such as soup. We must confess that it is difficult for us to decide upon this point. Numerous well-known physiological experiments prove that the body maintains its amount of blood with great tenacity at a certain constant ratio. Inasmuch as the body is able by means of numerous ways (secretion and absorption of liquids) to make speedy compensation for variations arising through changes in the amount of water ingested, it is by no means proved that the total amount of blood in patients with circulatory derangements is increased; and if actually there is a retention of fluid in the body (as indeed is certainly the case where œdema has developed), yet the liquid does not collect in the vessels, but in the lymph-spaces of the interstitial tissue, or possibly in the cells of the parenchyma itself. That the total amount of water in the system may be subjected to great variations must certainly be admitted; but this assumption, that in circulatory disturbances there is an increase in the amount of water contained in the blood, is not proved. On the other hand, there are facts which contradict it, such as the actual number of blood-corpuscles and the specific gravity of the blood. That there should be any increase in the labor demanded of the heart, requires that a large amount of fluid should be added to the blood in a relatively short space of time, so that an actual, although extremely temporary, hydræmic plethora should exist. If this process is very frequently repeated, it will surely result in a permanent hindrance to the circulation. These circumstances are, however, actually found only in certain patients, particularly in great beer-drinkers. In them, of course, to forbid the ingestion of fluid means to forbid the drinking of beer, and is therefore of the greatest benefit; and we can not insist too strongly upon the necessity of this injunction in the case of corpulent beer-drinkers who are beginning to have some dyspnœa. It is not, however, in our opinion, permissible to apply this rule about liquids, as is sometimes done, to every patient without individualizing. For those who live temperately and are of spare figure, the amount of fluid ingested needs no special attention from the physician.

2. *Strengthening of the Cardiac Muscle and Promotion of Compensatory Hypertrophy by Increased Physical Exertion.*—That the fulfilling of this indication may be of great importance is certain, and Oertel lays the greatest stress upon inciting the heart to more vigorous contractions by means of suitable bodily exertion, and especially by methodical mountain-climbing, in order by these means to promote as much as possible the development of cardiac hypertrophy. This view is probably fully justified and of obvious utility in many cases of simple muscular cardiac weakness. If, however, we apply it to those cases of circulatory disturbance in which there is valvular disease, or some other actual mechanical hindrance to the circulation, or in which the heart is already suffering from functional strain, the matter seems to be entirely different. For we must consider that we can not transfer unreservedly to the cardiac muscle our current views relating to the voluntary muscles of the body with regard to exercise and invigoration. The functional activity of the heart is minutely regulated by means of an especial reflex apparatus, independently of our volition. We know that every increased demand upon the heart's activity is in most cases directly fulfilled by an increased cardiac effort. Under proper conditions the most marked cardiac hypertrophy may develop in a completely bedridden patient. We must therefore consider carefully whether, in cases of this sort, the further increase of the demands upon the heart is judicious; whether it may not, on the contrary, contribute to a premature exhaustion of the myocardium. It certainly seems to us, and our opinion is fortified by practical experience, that the prescription of increased

bodily exertion, such as mountain-climbing, should always be given with great caution, and with consideration of the individual circumstances, if the physician desires to avoid unhappy consequences. It may indeed be claimed that a certain measure of bodily exercise is very suitable for many patients with valvular and other similar cardiac lesions, but we hold that the benefit lies less in the resultant "invigoration of the cardiac muscle" than in the promotion of the venous circulation occasioned by the motion of the extremities and the deeper inspirations; or, in the obese, in the increased metabolism of fat occasioned by the increased muscular effort. Nevertheless, the inconsiderate prescription of muscular exertion has worked much harm. Long mountain trips have a great and unique value for those circulatory disturbances alone which are occasioned by obesity, or associated with simple weakness and slight dilatation of the heart.

3. *Promotion of the Circulation by Massage, Passive Movements, Gymnastics, and Baths.*—It can not be doubted that the first three of these are suitable means for the promotion of the circulation, especially in the veins. We ought not to overestimate their value, but we are fully justified in the assertion that they may sometimes be employed with decided benefit in circulatory derangements. There are patients whom we dare not permit to take general bodily exercise, and for these there is sometimes great benefit in Swedish movements methodically and regularly pursued, especially in the form of what is called resisted movements with the arms and legs. The employment of massage is likewise useful in appropriate cases, in which there are such symptoms as slight œdema and moderate dyspnœa.

Thermic stimulation and baths are considered to have a strengthening influence upon the heart. About this we have as yet little physiological knowledge, but we can not ignore the fact of the beneficial influence upon many cardiac patients, from the use of lukewarm, or warmer baths, and in particular the salt baths and natural brine baths, containing carbonic-dioxide gas, as employed at Nauheim and Kissingen, and other places.

To sum up what has been said, we must, in every case of beginning impairment of the circulation, give full weight to the considerations just enumerated. We must find out the patient's mode of life, and determine and regulate his diet and exercise according to the individual circumstances. In this we must always pay great regard to the patient's constitution, whether he is obese or spare; whether he is full-blooded or anæmic; whether his muscular strength is great or small. It scarcely seems necessary to go into every detail in this place, since all the points are repeatedly discussed in other portions of this book. We would refer particularly, however, to the chapter on obesity.

3. Hypertrophy of the Heart, Associated with Congenital Smallness of the Systemic Arteries

(Hypoplasia of the Aorta)

For a considerable time, physicians have occasionally noticed cases, of the sort to be described, in which weakness of the heart and disturbance of the circulation appear at a comparatively early age. The patient complains of palpitation, dyspnœa, and slight œdema. On examination we usually observe anæmia, combined with more or less cyanosis. The cardiac dullness is extended toward the left, and the apex of the heart is displaced in the same direction. There is no marked dilatation of the right ventricle till the later stages of the disease. The heart-sounds are perfectly clear, unless there may be a murmur due to relative insufficiency of the mitral valve, or due to imperfect muscular contraction. The heart's action is usually regular, but considerably accelerated; the pulse small and the arteries contracted, but often of high tension. We observe the ordinary symptoms of increasing circulatory disturbance, and finally death. Upon autopsy we find the heart hypertrophied and usually dilated. The valves are normal, but the entire aorta and, in most cases, probably, the other large arteries, are hypoplastic, though otherwise of normal structure. Such cases are termed congenital hypoplasia of the systemic arteries, and it is believed that the hypertrophy of the heart is due to the increased effort to drive the blood through these small vessels.

In our opinion, this condition of the blood-vessels deserves full consideration in forming an opinion about idiopathic hypertrophy of the heart; but, ordinarily, still other factors are potent at the same time, for we sometimes find this hypoplasia at the autopsy of persons who showed no material disturbance of the circulation during life, and in whom the heart is of normal or even less than normal size. Virchow has called special attention to the fact that this sort of hypoplasia of the aorta and its branches is sometimes found in association with chlorosis, or, perhaps more correctly, with constitutional anæmia. In these cases the smallness of the reservoir diminishes the volume of blood contained in it, but it does not have any marked influence upon the heart, which indeed has a less than normal burden rather than an excessive one; so that we believe that in those instances of congenital hypoplasia of the aorta in which there is severe circulatory disturbance there is either coincident smallness and feebleness of the heart itself (*vide infra*), or an association of the hypoplasia with other influences unfavorable to the heart. There have been repeatedly observed cases of cardiac hypertrophy due to muscular strain (for instance, in soldiers), or due to excessive drinking, in which congenital hypoplasia of the aorta and its branches has been found in association with the hypertrophy of the heart; and in the cases which we have ourselves seen and in which the diagnosis was confirmed by autopsy, there have almost always been some other factors unfavorable to the heart besides this congenital abnormality of the blood-vessels. Indeed, one can easily understand that the same injurious influences which a normal circulatory apparatus might endure for a considerable time without much damage, would occasion premature symptoms if the vascular system were abnormally developed.

The recognition of hypoplasia of the systemic arteries during life is probably in every case difficult, and scarcely ever absolutely certain. Important factors would be the existence of anæmia and a tendency to shortness of breath and palpitation from early youth; and also the discovery by palpation that the circumference of the arteries was less than normal. The condition of the heart is to be determined in the ordinary manner.

Prognosis and treatment must likewise be governed by the same principles as in other forms of heart disease.

4. Primary Weakness of the Myocardium

(*Congenital Weakness of the Heart; Weakened Heart; Acute Muscular Strain of the Heart; Toxic Weakness of the Heart*)

If there is a myopathic heart disease, and the heart is at the same time hypertrophied, this indicates that for a considerable time the heart has done an unusual amount of work. In such cases, therefore, it can not be that the heart has been feeble from the start. It must indeed have been able to do more than is normally demanded of it. It is not until a later period, when its power is impaired, that we can say that the heart is relatively or finally absolutely feeble. There are cases, however, in which the heart is originally feeble—that is, its functional powers are below normal. This weakness is certainly, in many instances, congenital. It may be expressed in the structure of the heart, the organ being unusually small with thin walls, or merely in physiological incapacity, the organ being apparently of normal structure. In either case the heart can not satisfy even the ordinary demands made upon it. Patients of this sort complain, upon the least exertion, of palpitation, shortness of breath, and a sense of pressure over the heart. The pulse is usually frequent or, at any rate, very easily accelerated; a short but rapid walk, for instance, may raise the pulse-rate to 120 or 140 beats per minute.

In many cases there are never any severer symptoms. The patient remains through life feeble, and "with a weak heart"; but if his mode of life is favorable the heart manages to maintain the circulation. Not so, however, if unusual demands are made upon it—as, for example, in the course of military service, or in mountain climbing, or in unsuitable modes of life. Then more threatening symptoms of cardiac insufficiency appear, either suddenly or gradually, and express themselves objectively in dilatation (or stretching) of the heart. In some cases the heart is not able permanently to satisfy even the ordinary demands made upon it. We observe all the symptoms of circulatory disturbance, and at last upon autopsy find a heart which is dilated, but not much hypertrophied. Cases of this sort are not very frequent, but they certainly do occur; their recognition during life, however, is not easy, for it is difficult to distinguish simple dilatation from insufficiency which is subsequent to hypertrophy. We shall probably be able to determine that there is some muscular heart disease because of the increase in cardiac dullness, the smallness and frequency of the pulse, and the ordinary tokens of disturbed circulation, such as dyspnœa, palpitation, œdema, and the urine of passive congestion. If we consider carefully the history of the case and the presence or absence of special ætiological factors, we may be able while the patient is still alive to make a probable diagnosis of simple dilatation of the heart, as a result of muscular weakness.

Acquired muscular weakness of the heart should be distinguished from the congenital variety. It is occasioned by the action upon an originally normal heart of influences which damage the myocardium or its nervous apparatus. We often see temporary conditions of muscular weakness of the heart in anæmia, or following severe attacks of acute disease. Even in these cases there are such differences as to indicate a difference in the original vigor of the heart. The same is true of acute muscular strain of the heart. Acute attacks of cardiac weakness are seen in the case of soldiers at maneuvers, or in laborious mountain climbing, such as has of late been so often undertaken in an ill-considered manner; and these attacks are usually associated with acute dilatation. There is a sudden and enormous increase in the demands made upon the heart; the amount of blood which, in a given period of time, must pass through the muscles is increased; and the heart is unequal to the task. It yields to the increased tension and dilates. The pulmonary circulation becomes congested, the pressure in the arteries falls, and we have all the clinical phenomena of cardiac insufficiency, including dyspnœa, cardiac asthma, and sometimes angina pectoris. If there is prompt cessation of the effort and medical assistance, the condition may be restored to normal and so remain. This, for example, we lately saw in the case of a young and healthy person who escaped drowning only by desperate efforts. But sometimes there remains behind a permanent weakness of the heart, whether because the single excessive strain caused a permanent damage to the heart, or because the heart was already of less than normal vigor, and betrayed its weakness for the first time when this excessive demand was made upon it.

Certain forms of chronic intoxication are among the causes which frequently lead to acquired weakness of the myocardium, or its nerves. The most important of these, from a clinical standpoint, is chronic alcoholism, the noxious influence of which upon the heart is universally recognized. Less frequent, but still of practical importance, is chronic nicotine poisoning, or, to speak in a more general and perhaps more correct manner, the influence of excessive smoking. This is especially evident in persons who have smoked many strong-flavored Havana cigars. The symptoms consist of an unpleasant subjective sensation in the region of the heart (fluttering, pressure, or palpitation), of slight dyspnœa, and objectively of a frequent, irregular, or intermittent pulse. Other objective cardiac signs are not present, at least at first; but there may be still other symptoms of chronic nicotine poisoning, such as specks before the eyes, disturbance of vision, and dyspepsia. If smoking is stopped in due time, the symptoms may vanish. Otherwise, there follow severer disturbances of the heart, although in most cases there are other ætiological factors also present, such as alcoholism and mental strain.

With regard to prognosis and treatment, there are few special statements to be made. If there are signs of congenital weakness of the heart we must strengthen the constitution in every way possible, and we must seek to give the patient, on the one hand, the proper amount of protection, and, on the other hand, cautious and moderate exercise of the cardiac muscle, by means of medical gymnastics; and in other respects regimen and hygienic precautions are of first importance, from a prophylactic and therapeutic point of view. The treatment of acute and chronic weakness of the heart by such remedies as cardiac stimulants and digitalis is controlled by the ordinary rules. Finally, we may mention in this connection that obliteration of the pericardial sac by chronic pericarditis, or as a sequel of antecedent acute pericarditis (*vide infra*), sometimes occasions atrophy of the myocardium, with resultant feebleness and dilatation of the heart. Cases of this sort may readily be confused with primary cardiac dilatation.

5. The So-called Fatty Heart

Ætiology and Pathological Anatomy.—By the name of "fatty heart" we often mean, at present, two quite distinct conditions of the heart—the one an abnormal deposit of fat in the heart, and the other a fatty degeneration of the muscular fibers of the heart.

Fatty overgrowth and fatty infiltration of the heart are usually merely symptoms of great general obesity. At the autopsy of very fat people we sometimes find the heart entirely inclosed in a thick capsule of fat. The fat is situated chiefly in the external pericardium and beneath the visceral pericardium. It is usually abundant along the course of the larger vessels within the grooves of the heart, but in marked cases the fat also involves the muscular substance, so that many groups of fat-cells are interspersed between the muscular fibers. The heart itself is otherwise quite normal or somewhat hypertrophied or dilated. There are in some instances also present sclerosis of the coronary arteries and indurations due to myocarditis.

We have already mentioned fatty degeneration of the muscular substance of the heart as a frequent result of valvular disease. In myocarditis and idiopathic cardiac hypertrophy, and in the hypertrophy secondary to chronic nephritis and pulmonary emphysema, we also meet with fatty degeneration. We often find fatty degeneration of the heart, as well as of other organs, in severe acute infectious diseases, in phosphorus poisoning, and in all marked primary and secondary anæmias. Under the microscope we find the muscular fibrillæ studded with little drops of fat, which may be so numerous that the nuclei and the transverse striation of the fibers are quite concealed by them. We often find, besides the fatty granules, albuminous granules, which disappear on the addition of acetic acid ("cloudy swelling" of the cardiac muscle). If the fatty degeneration is of high degree, we can easily recognize it with the naked eye. Beneath the endocardium, especially on the trabeculæ and papillary muscles, we see very fine and delicate yellow points and striæ. With great fatty degeneration, as in phosphorus poisoning and pernicious anæmia, the whole cardiac muscle is manifestly yellow, and also soft and flabby. It is claimed that rupture of the heart may occur as a result of marked fatty degeneration.

In fatty degeneration of the heart the fat comes from the decomposition of albumen in the muscular cells. The occasion of it is probably a defective supply of oxygen, which has either a general cause, as in anæmia and phosphorus poisoning, or a local cause, as disturbed circulation in the heart in heart disease. The details of this are given in the chapter on anæmia (page 708).

Clinical Symptoms.—Fatty degeneration of the heart has no characteristic clinical symptoms. In the conditions under which we know it is apt to occur, we can usually suspect it during the lifetime of the patient, but we can not diagnosticate it. We must also mention that the frequently expressed opinion, that fatty degeneration of the heart invariably occasions general cardiac weakness, is very often incorrect. In pernicious anæmia there may be quite a strong and a perfectly regular pulse up to death in spite of the most marked fatty degeneration, and many personal observations lead us earnestly to dispute the view that fatty degeneration of the heart is to be regarded as the regular cause of cardiac insufficiency, and the consequent disturbances of compensation. We have made microscopic examinations in many cases, and failed to find any sign of fatty degeneration in the myocardium, although during life the signs of cardiac insufficiency were well developed. In general, with our present knowledge, it is impossible to establish definite relations between the histological condition of the muscular tissue of the heart and its functional activity during life.

We can not say much that is certain in regard to the clinical symptoms of a deposit of fat in the heart. "Fatty degeneration of the heart" always plays a far larger part in popular speech than it does in reality. It is certainly a fact that difficulty with the heart and respiration is very often observed in fat people. Examination of the heart, which, however, is decidedly impeded by the thick panniculus adiposus, often shows in such cases an increase of the cardiac dullness, a small and sometimes irregular pulse, and faint but clear heart-sounds. The disturbance may be very considerable, attacks of angina pectoris and cardiac asthma may come on, and death may follow with increasing dyspnœa and general œdema.

If one has opportunity to make an autopsy in such cases, there will be found no single, constant anatomical change as the cause of the cardiac disturbance, but usually idiopathic hypertrophy (*vide supra*), or, less often, myocarditic changes with sclerosis of the coronary arteries, and the like. Sometimes, but by no means invariably, there is, of course, a marked deposit of fat upon the heart itself, but the question arises whether this can directly and seriously embarrass the cardiac activity. The fact is that we often have seen similar well-marked cases of fatty heart which during life presented no special cardiac symptoms. There would be more reason in ascribing an unfavorable influence to the fatty infiltration of the cardiac muscle; but in such cases there is almost always a coincident atrophy of the muscular structure, so that it is questionable whether the fatty infiltration is to be regarded as actually the primary pathological process. We ourselves are far more inclined to the view that there is primary atrophy of the myocardium, with this fatty infiltration as a secondary phenomenon, similar to the frequent and well-known occurrence of secondary lipomatosis of atrophic voluntary muscles. At any rate, we are as yet entirely unable to recognize cases of this sort of fatty heart during life, and it is certain that they are much less frequent than the other myopathic diseases of the heart.

Therefore we can not associate with the term "fatty heart" any uniform anatomical and clinical conception. It would be better to speak of the "heart of obesity"—that is, of all the manifold injuries to which the heart of obese persons is exposed.

According to our experience, simple idiopathic hypertrophy of the heart is certainly the most frequent of these conditions, and it would be very desirable if physicians would have more regard to the facts of pathology, when they are inclined to make an off-hand diagnosis of fatty heart.

Treatment.—A great part of the disturbance of respiration in fat people depends not upon the cardiac weakness, but on the corpulency itself. The great bulk of the body, and the hindrance to the activity of the respiratory muscles, are very important factors. Treatment directed against the respiratory disturbance must hence attack the obesity chiefly, and thus in many cases we also assist the action of the heart. The detailed description of the hygienic methods of cure to be employed here is to be found in the chapter on obesity (page 784).

In regard to the special treatment of the cardiac symptoms in the obese, this does not differ from the rules that obtain in other forms of heart disease.

CHAPTER IV

CARDIAC NEUROSES

1. **Angina Pectoris (Stenocardia).**—Angina pectoris is not an independent disease, but a group of symptoms, to which we have had frequent occasion to refer already; but for a considerable time this symptom-complex may be the only sign of disease. It is the paroxysmal occurrence of an extremely violent pain below the sternum and in the region of the heart, associated with a feeling of the greatest distress and oppression ("præcordial anxiety," "sense of impending death"). The pain is usually described as cramp-like, boring, and constricting It not infrequently radiates from the heart into the shoulders and the left arm, sometimes as far as the tips of the fingers. The beginning of the attack is usually absolutely abrupt. It may last a few minutes or half an hour, or longer. The attacks may be infrequent with intervals of weeks or months, or they may be repeated several times a day. Respiration during the attack is often unaffected, in which point genuine angina pectoris differs from cardiac asthma. Many patients indeed seek to lessen their pain by holding the breath, and sometimes even in angina pectoris the respiration is hurried and irregular. It must be pointed out that the symptoms of "pure" angina pectoris and the symptoms of cardiac asthma may be clinically much intermixed and confused. The pulse is usually rapid and small during an attack, and not infrequently irregular. Sometimes, however, we find that, in spite of the most violent pain and distress, the pulse remains strong, quiet, and apparently normal. If the hand is placed upon the heart a peculiar wavy motion and "fluttering" is felt in a few cases, and this symptom the patient also perceives subjectively. The countenance is usually pale during an attack; the hands feel cool. Toward the end of an attack there is often abundant perspiration.

We are as yet entirely ignorant as to the internal processes which are the foundation of angina pectoris. The condition is often compared with neuralgia, and spoken of as "cardiac neuralgia." Other investigators suspect that there is a painful contraction of the blood-vessels of the heart. It is certain that angina pectoris rarely occurs as an independent disease, or pure neurosis. Nothnagel described a vaso-motor form of angina pectoris ("angina pectoris vasomotoria"), in which there were at the same time distinct signs of vascular contraction in the extremities, as indicated by pallor, cyanosis, and paræsthesiæ. In the case of excessive smoking, also, angina pectoris sometimes appears as a result of chronic tobacco poisoning, without any other demonstrable change in the heart. Similar attacks may occur after strong mental excitement, but these have more or less connection with certain forms of hysterical and neurasthenic attacks, bearing a merely superficial resemblance to angina pectoris. The true angina pectoris is most often seen as a symptom in association with certain diseases of the heart and aorta, particularly sclerosis of the coronary arteries, insufficiency of the valves, aneurism of the root of the aorta; less often, idiopathic hypertrophy of the heart, and hypertrophy secondary to interstitial nephritis. The way in which the other symptoms of these conditions may be conjoined with the anginous attacks is sufficiently clear from their presentation in a previous chapter.

We can not speak of the prognosis of angina pectoris as a distinct entity. In general angina pectoris is certainly of evil omen, and occasionally death may occur during the attack.

With regard to the symptomatic treatment of angina pectoris, there is no doubt that in severe cases the greatest relief is obtained from a subcutaneous injection of morphine. We confess that the employment of morphine in severe heart disease always demands the greatest caution, but yet experience shows that in numerous instances this remedy is excellently borne, and most beneficial. Among other remedies should be named chloral and chloralamide, which also are to be employed with caution, but which sometimes have a good effect; nitroglycerine to the amount of $\frac{1}{60}$ to $\frac{1}{12}$ of a grain (gramme 0.001–0.005), which is highly praised by many physicians; also nitrite of sodium, and inhalations of amyl nitrite, ether, or chloroform. If there are decided signs of cardiac weakness, camphor, strophanthus, and alcohol are to be employed as stimulants. An ice-bag may be placed over the heart, and sometimes, also, hot applications are of benefit; mustard-poultices, hot foot-baths, and the like are also frequently prescribed in practice.

We must always bear in mind the general treatment of the underlying condition, as well as the employment of purely symptomatic remedies. Special attention should be directed to any ætiological factors, such as muscular strain, mental excitement, alcoholism, or excessive smoking. If we have to do with the "purely nervous" form of angina pectoris, without any physical signs of change in the heart, we must of course avoid the use of the stronger narcotic remedies so far as possible, and lay the main emphasis upon the general treatment of the nervous system by means of electricity, cold-water cures, and psychical influences.

[In well-marked angina sudden death during a paroxysm is not very rare. In Dr. Arnold, of Rugby, the first attack proved fatal. The patient may, however, live for years—cases of survival for upwards of twenty, and one even of thirty, years being recorded. Flint has known recovery to occur. The prognosis depends somewhat on the condition of the cardiac valves and walls: but changes, especially in the latter, may escape detection by any save a very skillful observer. Walshe states that in every one of twenty-four cases he examined during life he found physical signs of changes either in the heart, the aorta, or both. The experience of Balfour and Latham is similar.]

2. **Nervous Palpitation.**—By "palpitation" we understand the subjective perception of the movements of the heart. It is usually excited by increased action of the heart, but there is no constant relation between the intensity of the cardiac pulsations and the subjective feeling of them. We sometimes observe that patients with aortic insufficiency do not perceive the very strong action of their hypertrophied hearts, while in other cases a patient complains of a troublesome feeling of palpitation, although the action of the heart does not appear objectively to be especially increased.

We term cases "nervous palpitation" if the patient complains of palpitation when a physical examination of the heart shows no anatomical change in it.

As a rule, in these cases we really have to do with a heart whose action is increased by abnormal nervous influences. In many cases the palpitation arises from slight external causes, which may give rise to little or no palpitation in a healthy person, as, for example, after the slightest mental excitement, after any slight physical exertion, after taking food, after indulging in certain drinks, such as tea, coffee, wine, or beer, or after assuming certain positions, as in lying on the left side. Here, then, we have to do with an abnormal sensitiveness of the heart to external irritation. Usually the increase in vigor of the heart's action is associated with a decided increase in its frequency. We have often seen patients with nervous palpitation who had a pulse-rate of 140 to 160 beats a minute after comparatively slight exertion; but in other cases there is apparently a sort of hyperæsthesia of the patient with regard to the motions of the heart, so that contractions of normal strength and rapidity give rise to disagreeable sensations.

The patient rarely complains of continuous palpitation; it usually occurs in more or less sharply defined paroxysms. Very commonly in pure nervous palpitation we have to do with persons who, in general, suffer from nervous, hysterical, and neurasthenic symptoms, or they are anæmic persons, chlorotic girls, etc.; but, on the other hand, nervous palpitation may occur in very full-blooded, "plethoric" subjects.

Often hypochondriasis plays an important part, and dread of heart disease and the thought of its possible consequences excite a palpitation which confirms the patient in his delusion.

The diagnosis of nervous palpitation can be made only when repeated careful examination shows no objective abnormality in the heart. In many cases, as when there are anæmic murmurs, the decision may be quite difficult. We must always pay particular attention to the whole constitution and the general impression which the patient makes.

It is often especially difficult to distinguish between purely nervous palpitation in a heart otherwise functionally normal, and conditions of congenital or acquired weakness of the heart (*vide supra*).

The prognosis is so far favorable in that the disease is not dangerous. In many cases improvement and final recovery may be effected, but other cases, of course, resist all therapeutic efforts very obstinately.

The treatment must first be directed to improving the patient's general constitution. The anæmic are to be given iron, quinine, and strengthening diet. We put full-blooded persons, however, on scanty fare, and prescribe for them bitter waters, or a bath cure at Marienbad or Kissingen. When there is hysteria or neurasthenia, it requires special treatment. If the patient is hypochondriacal, of course the main thing is for the physician to reassure him. We should avoid all the influences which seem to excite palpitation. As a symptomatic indication during an attack we should recommend the patient especially to keep quiet. The use of cold to the cardiac region—cold compresses and ice-bags—often acts beneficially. On the other hand, however, it is to be noted that a tendency to palpitation associated with cardiac weakness may often be allayed by methodical exercise and the invigoration of the cardiac muscle consequent thereupon (see the preceding chapter). Among internal remedies we must employ nervines, and in severer cases even narcotics. Among the former we would mention especially ethereal tincture of valerian and bromide of potassium, which have repeatedly done us good service. Digitalis is usually of little value in pure neuroses of the heart, but we may give it as an experiment, say fifteen to twenty drops of the tincture with the same amount of cherry-laurel water.

3. **Tachycardia (Paroxysmal Tachycardia).**—A peculiar and quite rare neurosis of the heart, tachycardia, consists of an enormous frequency of the pulse, coming on in paroxysms, up to 160–200 beats and more a minute.

We have already mentioned these attacks as a rare symptom in mitral and aortic valvular disease and in diseases of the myocardium, but precisely similar attacks occur as a pure neurosis without demonstrable lesion of the heart, particularly in anæmic, nervous, or obese individuals. We saw one very well-marked case in a lady with extreme neurasthenic melancholia. The attacks occurred many times a day, each lasting only a few minutes. We were often able while feeling the pulse to notice that it suddenly became extremely rapid, and then in a brief time almost with equal suddenness resumed its approximately normal rate. We have seen many similar attacks of genuine tachycardia, although not so extreme, in young and neurasthenic individuals, following muscular exertion—for example, in soldiers. Similar states develop sometimes after diphtheria (*q. v.*), and after other acute infectious diseases; but in such instances the tachycardia is usually more continuous and not so distinctly paroxysmal.

In men we must consider the possibility of the action of injurious habits, such as drinking and smoking. The individual attack usually begins quite suddenly, by day or by night, sometimes without any cause, but often it is apparently produced by certain exciting causes, especially at times by overdistention of the stomach. The patient feels that the attack is coming, he becomes anxious and restless, and looks pale; but there are not, as a rule, at least according to our experience, any symptoms like præcordial anxiety, dyspnœa, or attacks of faintness. We notice in the heart itself, during the attack, chiefly a great acceleration of the heart-sounds. We sometimes hear indefinite, functional murmurs. The action of the heart is often quite regular, but there is not infrequently manifest arhythmia during the attack. Acute increase of the heart's dullness has been observed during the attack. We have repeatedly been able to verify a marked temporary dilatation of this sort, particularly in patients with heart disease, and also in one case of paroxysmal tachycardia affecting a sufferer from cirrhosis of the liver. In other instances and especially when the tachycardia is a mere neurosis, no such dilatation of the heart can be made out.

We know little that is definite as to the nature of the attacks. The affection is usually regarded as a temporary paralysis of the vagus.

We may also state here that paroxysmal and constant tachycardia have also been repeatedly observed in anatomical lesions of the cardiac nerves and their centers, in tumors and other affections in the vicinity of the medulla oblongata, and in compression of the vagus in the neck from new growths, and aneurisms.

The prognosis of tachycardia depends first upon the nature of the underlying disease. We do not know whether a permanent recovery is possible in idiopathic cases, but we can always succeed in improving the condition. The treatment during the attack consists in enjoining complete bodily rest, and in applying ice to the heart. Such sedatives as bromide of potassium, water of bitter almonds, and tincture of valerian are particularly beneficial in the "purely nervous" cases. Sometimes the physician may even be constrained to the cautious injection of small amounts of morphine. In other respects our treatment is determined mainly by the underlying condition, if known. [If there is reason to think that the stomach is overloaded, gentle measures should be taken to empty it.—V.] The best way to guard against the return of the attacks is to give precise hygienic directions, suited to the patient's constitution and manner of life. The continued use of iodide of potassium has sometimes seemed to us to be of service.

SECTION II

DISEASES OF THE PERICARDIUM

CHAPTER I

PERICARDITIS

(Inflammation of the Pericardium)

Ætiology.—Pericarditis seldom appears as a primary idiopathic disease. It is usually merely a sequel or a complication of other diseases. Thus, it is observed with particular frequency in the course of acute articular rheumatism, where it appears sometimes alone, sometimes in combination with acute endocarditis. It is not impossible that some few cases of apparently primary acute pericarditis belong, from an ætiological standpoint, to acute articular rheumatism—that is, they are excited by the same pathogenic factors which exceptionally attack the pericardium alone, without simultaneous participation of the joints in the disease. This supposition is rendered probable from the later course of many such cases. For instance, arthritis may ensue. Cases of secondary pericarditis, unassociated with articular rheumatism, occur, although much less often, in other acute infectious diseases, among which scarlet fever, measles, and septico-pyæmic processes, as well as scurvy and purpura hæmorrhagica, deserve especial mention. In sepsis and pyæmia the pericarditis is purulent, and in purpura, hæmorrhagic. Among the chronic diseases in the course of which pericarditis sometimes appears we must mention especially chronic nephritis. We have seen hæmorrhagic pericarditis in association with leukæmia. Pericarditis sometimes occurs also in the victims of carcinoma, but here it is probably dependent upon a secondary septic infection. Finally, it should be mentioned that severe hæmorrhagic pericarditis is seen comparatively often in alcoholic subjects, and apparently as a primary disease. Often it is really tubercular, but by no means always; and if not, the author is inclined to regard it as a primary hæmorrhagic inflammation, analogous to hæmorrhagic pachymeningitis or hæmatoma of the dura mater.

A large number of cases arise from an extension of the inflammation from the vicinity. Thus we not infrequently see pericarditis as a result of pleurisy, especially on the left side, and in pneumonia complicated with pleurisy. New growths and ulcerative processes in the œsophagus, in the vertebræ, in the bronchial glands, or in the lungs, also lead at times to perforation into the pericardium and a consequent inflammation. It is not settled whether the pericarditis, which appears in the course of chronic valvular disease, is also to be regarded as arising from extension by contiguity. We have thought of this possibility, because we have been struck with the fact that secondary pericarditis is especially frequent in valvular disease of the aorta, suggesting a direct propagation of the inflammatory germs through the aortic wall to the pericardium. Still, it must of course be allowed that this form of pericarditis may have an independent origin, particularly in cases of mitral disease. Pericarditis may also develop as a result of myocarditis, abscess of the heart, etc.

Tuberculosis plays a very important part in the ætiology of pericarditis. No small number of apparently primary cases of pericarditis turn out at the autopsy to be tubercular. This seems to come on in quite an isolated way, or as one symptom of a special localized form of tuberculosis, which we term tuberculosis of the serous membranes. In many cases we can discover the origin of a tubercular pericarditis in the direct extension of a tubercular pleurisy. In apparently primary cases the occurrence of the infection may sometimes be explained by the discovery of a tubercular lymph-gland, which has broken through into the pericardium.

Pericarditis is usually a disease of youth and middle life, but it may also occur in advanced age.

Pathological Anatomy.—Ordinary pericarditis involves the internal surface of the pericardium in either a circumscribed or diffuse manner. Inflammation of the outer surface of the pericardial sac is distinguished as external pericarditis (*vide infra*). The anatomical processes in pericarditis are precisely analogous to those in inflammations of the serous membranes in general, especially of the pleura.

We usually divide pericarditis into fibrinous, sero-fibrinous, hæmorrhagic, and purulent (or ichorous) forms, according to the character of the exudation. The fibrinous and sero-fibrinous forms, with an abundant fluid effusion into the pericardial cavity, are the most frequent, occurring in articular rheumatism, in valvular disease of the heart, etc. Both layers of the pericardium are covered with masses of fibrine, which often show a reticular or villous arrangement (*cor villosum*). Besides this, we find more or less of a fluid effusion which distends the pericardium. The fluid is of a serous nature, contains more or less numerous flakes of fibrine, and is turbid from the admixture of cells—pus-corpuscles, and, in part, desquamated endothelium. A purulent pericarditis is always the expression of a specific infection of the pericardium. It is seen in pyæmic diseases, as a result of empyema, and in perforation of abscesses, cancers of the œsophagus, etc., into the pericardium. A hæmorrhagic effusion is seen chiefly in tubercular pericarditis. In this we find miliary tubercles, and little cheesy nodules in the inflammatory new growths, besides all the signs of inflammation. The specific tubercular changes are sometimes recognizable with the naked eye, but at other times we have to use the microscope to find them. Hæmorrhagic pericarditis also occurs in general hæmorrhagic diseases, such as scurvy, and in weak and debilitated persons, especially drunkards (*vide supra*).

In long-continued pericarditis the cardiac muscle almost invariably undergoes changes. The heart is usually flabby and dilated, and the muscle often shows fatty degeneration. After the pericarditis has lasted a long time there is often considerable atrophy of the cardiac muscle, which is partly replaced by fat tissue. We have already mentioned the occurrence of pericarditis in connection with valvular disease and degenerations of the myocardium.

In favorable cases of pericarditis we may have a perfect recovery. The so-called *maculæ tendineæ* sometimes remain in the pericardium as residua of a past circumscribed pericarditis. In some cases the pericarditis leads to an adhesion of the two layers of the pericardium to each other, and obliteration of the pericardial cavity (*vide infra*). In many cases a chronic pericarditis finally develops from the acute form, or the whole affection takes a more chronic course from the outset. In this way chronic adhesions of connective tissue arise, and great thickening of the pericardium, but the amount of fluid is usually small. Sometimes the chronic pericarditis is interrupted by an acute exacerbation of the disease.

Clinical Symptoms.—1. *Subjective Symptoms, General Symptoms, and Fever.*—Mild forms of pericarditis may develop, as in the course of an acute articular rheumatism, without causing any subjective symptoms. They are discovered only by a careful physical examination of the heart. In severe cases, however, the pericarditis causes violent subjective symptoms, which of course have in themselves little that is characteristic.

On careful questioning we often find that there is pain in the cardiac region, or not infrequently in the epigastrium. This pain is of decided diagnostic importance, but it does not always exist.

A general feeling of constraint and distress is almost constant in all acute cases of any severity, and so is a feeling of dyspnœa, which may increase to the highest degree of orthopnœa. The patients often complain of headache. In severe cases they become stupid and comatose.

It is easy to understand that extensive pericarditis must impede the motions of the heart. Probably the most important way in which it does this is by increasing the tension in the pericardium, and thus embarrassing the diastole of the ventricles, so that the cavities of the heart are not normally filled and general disturbance of the circulation ensues. Furthermore, the pericarditic deposits and adhesions, and such changes in the myocardium as may occur, interfere with the cardiac systole. Thus are explained the early signs of diminution in the amount of blood received by the arteries, as shown by pallor and cerebral anæmia; the distention of the systemic veins, as shown by cyanosis; and the disturbance of the pulmonary circulation, causing dyspnœa.

The dyspnœa is also increased in large pericardial effusions by the mechanical pressure of the distended pericardium on the left lung.

Acute pericarditis is usually associated with fever. This has no special type, and usually keeps at a moderate height—102° to 103.5° (39°-39.8° C.)—but it often exhibits considerable variations. In cases of recovery the fever declines by lysis. Chronic pericarditis may run its whole course without fever.

2. *Physical Signs—Inspection.*—The general hue of a patient with severe pericarditis is pale, but also more or less cyanotic. He has an anxious expression. He lies with the upper part of the body raised, or he sits up in bed. The breathing is usually rapid, labored, and somewhat irregular. The veins in the neck are swollen and prominent. We very often see marked undulating or pulsating movements in the jugular veins, as a result of stasis. The cardiac region seems unusually prominent in all cases with much effusion, and the intercostal spaces there are flattened out. We sometimes detect a slight œdematous swelling of the chest-wall itself.

If the action of the heart is powerful and the exudation small, the heart's movements may be distinctly visible. In other cases they are seen only faintly, and they may be noticeably diffuse. If the exudation is abundant, or if adhesions occur (*vide infra*), they may disappear altogether.

Palpation in the milder cases shows the apex-beat in its normal position and of about normal strength; but if the amount of the pericardial effusion increases, the heart is pushed away from the chest-wall by it, and hence the heart-beat grows weaker until it disappears entirely. In such cases it is sometimes to be felt again if the patient bends forward or lies on his left side. In the rest of the cardiac region we sometimes feel the movements feebly, but they entirely disappear as the effusion increases. There is diagnostic importance in the contrast between the great extent of cardiac dullness (*vide infra*) and the faintly distinguishable motions of the heart, with absence of a distinct apex-beat and any marked epigastric pulsation. In some cases, by laying the hand flat on the chest, we can feel the rub of the rough pericardial surfaces against each other.

The pulse is usually accelerated, and in severe cases it becomes irregular. In every large effusion, as we have already said, the tension and height of the pulse are diminished. In severe cases the pulse sometimes becomes very small and weak, but, when the heart is otherwise normal and strong, it may also remain quite strong—and indeed this condition of the pulse, in contrast to the great weakening of the heart-beat, is sometimes of diagnostic significance. In some cases with a large pericardial effusion we have seen a manifest *pulsus paradoxus*—that is, a diminution or a complete disappearance of the radial pulse on every inspiration.

Changes in percussion-resonance are evident as soon as the pericardial sac is distended with exudation. The so-called triangular shape of cardiac dullness is regarded as especially characteristic of large pericardial exudations. The blunt apex of the triangle is found above in the second or third left intercostal space, near the edge of the sternum. The lateral boundaries run obliquely to the right and downward to about the right parasternal line, and to the left and downward to the left mammillary line, or beyond. The broad base of the triangle which lies below is usually not to be defined by percussion, on account of the adjacent left lobe of the liver. On the border of the dullness we often find a tympanitic resonance due to the retraction of the adjacent lung.

In general, the author's personal experience forces him to say that we must not be too dogmatic in the establishment of special shapes of cardiac dullness, as peculiar to pericarditis. The fact of enlargement of the area of cardiac dullness, and the marked sense of resistance upon percussion, are important signs, but there is a considerable variety in the shapes of the dull area, although extension of the dullness upward and to the right may be regarded as particularly frequent. According to Ebstein's observations, the first change in percussion in incipient exudation is usually an extension to the right of the heart's apex, in a triangle between the heart and the liver.

The area of the dullness depends, of course, in the first place, upon the amount of the effusion, but we must take special notice that in regard to this the relation is not constant. In old cases of pericarditis especially we sometimes find the cardiac dullness very extensive, while the autopsy detects only a little fluid in the pericardium. This is explained partly by a secondary dilatation of the heart, and partly by a persistent retraction of the lung.

It is an often-mentioned but seldom available diagnostic sign of pericarditis that in many cases the still perceptible apex-beat lies within the cardiac dullness, since the pericardial effusion extends farther to the left than the heart itself. It is also worthy of note that the dullness in pericarditis often shows very great changes when the patient changes his position. The dullness is more extensive when the body is erect than when lying down, and when the patient lies on his side it sometimes shows a lateral displacement of several centimetres. The same changes, however, though rarely so marked, also occur in a hypertrophied heart.

The characteristic pathognomonic auscultatory sign of pericarditis is the pericardial friction-rub. This arises during the movements of the heart from the rubbing of the rough and inflamed pericardial surfaces against each other. The friction-rub is absent in pericarditis if the rough surfaces of the two layers of the pericardium are separated from each other by a considerable fluid effusion, or if they can no longer rub against each other from an adhesion of the layers of the pericardium. We usually hear the friction-rub loudest in the neighborhood of the base of the heart, but it may also be heard at other parts of the heart. In general the adventitious sounds of pericarditis are not transmitted to any great distance. The quality of the sound is described as rubbing, grating, or scratching. The friction-rub may be heard chiefly either during the systole or during the diastole of the heart, but it is in general not often closely associated with the phases of the heart's action. We sometimes find it intermitting frequently, and jerky. The intensity of the friction-rub sometimes varies with the phases of the respiration. It is usually louder on inspiration, but sometimes on expiration. If the patient changes his position, the intensity of the sound is sometimes altered. It is louder when sitting up than lying down, etc. The friction-rub often sounds louder if the stethoscope is pressed firmly against the chest, since in this way the layers of the pericardium are approximated to each other.

The heart-sounds, when the valves are intact, may sometimes be heard as well as the friction-rub, or they may be completely drowned by the loud rub, at least in some parts of the heart. In general, they are faint in every case of pericardial effusion, since their conduction to the ear is impaired. In large effusions where no friction-rub is to be heard, we hear the heart-sounds, especially the first, but only very faintly and obscurely. This condition, in connection with the increase of the cardiac dullness, is of diagnostic importance. If there is also valvular disease with the pericarditis, the pericardial and endocardial murmurs are sometimes hard to distinguish from each other, but usually the former greatly preponderate.

3. *Sequelæ of Pericarditis.*—A large pericardial effusion may excite special symptoms from pressure on the neighboring organs. Thus we have already said that compression of the left lung must increase the dyspnœa. In many cases we also notice a moderate dullness over the left lower back, from compression of the left lower lobe. Not infrequently there is a combination of pericarditis and left-sided pleurisy with effusion. In rare cases difficulty in deglutition has been observed as a result of pressure on the œsophagus, and paralysis of one vocal cord from pressure on the recurrent nerve.

In cases of long-continued pericarditis the same sequelæ may develop as in any chronic disease of the heart. The amount of urine diminishes as a result of the low arterial pressure. The venous stasis finally leads to general dropsy and to symptoms of passive congestion in the liver, spleen, and kidneys. We would also state that we have repeatedly met with large effusions in the cavities of the body, especially hydrothorax, without any œdema of the skin. All the symptoms of stasis mentioned, however, are often due much less to the pericarditis itself than to the atrophy and dilatation of the heart which frequently follow it (*vide supra*).

SPECIAL FORMS OF PERICARDITIS

1. **Pericarditis externa and Mediastino-pericarditis** (*Pleuro-pericarditis*).—By pericarditis externa we mean an inflammation of the external surface of the pericardial sac, which is usually combined with an inflammation of the mediastinal connective tissue and the neighboring pleura, especially over the lingula of the left lung. This form of pericarditis may exist by itself, or it may be combined with internal pericarditis. It is a rare disease, and is most frequently seen as a result of tubercular pleurisy.

The physical signs must differ so much, according to the localization and extent of the process, that we can give few general data in regard to them. There are only a few peculiar signs, which must be noted as characteristic of many cases. In the vicinity of the apex-beat, or at the left border of the cardiac dullness, we sometimes hear a so-called extra-pericardial (pleuro-pericardial) friction-rub. This depends both upon the cardiac movements and upon the respiratory movements. The friction sound often becomes louder during inspiration, or, again, it may be audible only when the lungs are fully inflated. On holding the breath we hear merely the sound due to the pulsations of the heart, while on deep breathing the pleuritic friction-sound is also to be heard. In individual cases there are many modifications, which can not all be mentioned. Another interesting sign, first found by Griesinger and Kussmaul in a cicatricial mediastino-pericarditis, is the so-called *pulsus paradoxus*. This consists of a diminution of the pulse at each inspiration. This condition arises, in part of the cases at least, from the fact that the bands and adhesions of connective tissue at the origin of the aorta mechanically nick into and contract its lumen at every inspiratory movement of the thorax. This explanation, of course, does not suffice for all cases, since the *pulsus paradoxus* also occurs under other conditions, as with large pericardial effusions. In some cases there may be seen a marked swelling of the jugular veins in the neck at each inspiration, at the same time with the *pulsus paradoxus*, since the large venous trunks also undergo a mechanical nicking and constriction at each inspiration. We have ourselves seen a very pronounced slowing of the pulse at every inspiration, in a complicated case of extra-pericardial adhesions (vagus irritation?). We must also mention that Riegel observed a disappearance of the apex-beat on expiration in some cases where there were bands of connective tissue between the lungs and the outer surface of the heart. At every expiration the bands were stretched more tightly, and hence checked the movements of the heart.

2. **Obliteration of the Pericardial Cavity** (*Adhesive Pericarditis; Adhesions of the Layers of the Pericardium; Concretio seu Synechia pericardii*).—We may have a more or less complete adhesion of the two layers of the pericardium with each other as a result of pericarditis. We can sometimes observe the occurrence of this condition during the course of a pericarditis. Quite frequently, however, we meet with extensive adhesions of the two layers of the pericardium at autopsies, without being able to gather any history of a previous acute pericarditis. The pericarditis must have occurred here in a chronic way, and without symptoms from the outset.

Even extensive adhesions of the pericardial surfaces may develop and remain entirely without symptoms, and be met with accidentally at the autopsy. In other cases, however, the obliteration of the pericardial sac causes special physical signs and severe clinical sequelæ. Of the physical signs, one of the best-known and most discussed is the systolic retraction of the chest, either limited to the apex or involving a larger area. This is most comprehensible if there is an adhesion of the pericardium with the heart, and also with the chest-wall (Skoda); but we certainly find this retraction at the systole without co-existing extra-pericardial adhesions. It is not, however, an absolutely certain sign of an intra-pericardial adhesion, especially if we have to do with a systolic retraction at the apex alone, or limited to the third or fourth intercostal space, since systolic retractions may sometimes occur in other disturbances of the heart's motions, or even under normal conditions; but systolic retractions of the whole cardiac region are, in the majority of cases, a certain sign of pericardial adhesion. The amount of this retraction is often dependent upon the respiration, it being usually more marked on inspiration.

On the other hand, it must be noted that very often there is no retraction of the præcordia during systole, in many cases of undoubted obliterative pericarditis. In brief, the symptom under consideration is extremely ambiguous, and it must always be viewed with considerable reserve in forming a diagnosis. Of at least equal importance, in our opinion, is the obscurity or absence of any cardiac motion either to sight or touch. We can lay great diagnostic value upon the absence of a normal cardiac impulse, when the cardiac dullness is increased, although the heart-sounds are normal; and when the adjacent borders of the lung do not move with respiration, because of the adhesions due to the associated pleurisy (*vide infra*); and finally, when the history of the case and the general course of the illness are confirmatory, for instance, if there are indications of a previous acute pericarditis.

The other symptoms of obliteration of the pericardial cavity are more rare and in their diagnostic significance still more uncertain. Friedreich observed a sudden collapse of the jugular veins at each diastole—the "diastolic collapse"—while they became well filled again at the next systole. He explained this phenomenon by supposing that the conditions for emptying the veins, at the moment of the diastole of the ventricle, were especially favorable, since the chest-wall, which had previously been drawn in by the systole, went back again quickly. But we have ourselves seen distinct diastolic collapse of the veins in a case of adhesive pericarditis, the diagnosis of which was confirmed by autopsy, in which there was no systolic retraction of the præcordia. Riess described some cases of pericardial adhesions in which the heart-sounds had a metallic character from the resonance of the stomach, which had been drawn up. All things considered, we must say that although the diagnosis of pericardial adhesions can be correctly made in many cases, yet the signs given for it are more or less uncertain, since they may be absent in obliteration of the pericardial sac, and they may also be caused by other conditions without such an obliteration. In a word, it is no easy matter to establish the diagnosis of obliterative pericarditis, and in cases of this sort the autopsy not infrequently brings surprises, as our own experience has shown.

The general constitutional symptoms of obliterative pericarditis are also subject to great variations. In many cases the affection, as we have said, has no symptoms at all or at least no grave results. In other cases, however, there is distinct evidence of circulatory disturbance. This disturbance is sometimes due directly to the mechanical embarrassment of the heart, but more often to secondary changes in the myocardium. We find atrophy, fatty infiltration, and fatty degeneration, with consequent dilatation. In such cases the pulse becomes small and frequent, and the ordinary picture of ruptured compensation is displayed, characterized by œdema, dyspnœa, and congestion of the liver and kidneys. In these cases the diagnosis is often very difficult. It is scarcely possible to distinguish between this form of pericarditis and chronic myocarditis, or the "idiopathic" enlargement of the heart.

There is another symptom-complex, somewhat different from the above, which deserves special mention. We have been acquainted with it for years, but it has only lately received general attention. Sometimes, when there is obliteration of the pericardial sac, there is developed a condition so like that of hepatic cirrhosis (*q. v.*), that the name of "pericarditic pseudo-cirrhosis of the liver" has been applied to it. There are increasing dyspnœa and great ascites, with subsequent œdema of the lower extremities, so that we have dropsy of the lower half of the body, while the upper half remains free from œdema (see Fig. 43). There may be, however, a serous effusion demonstrable in one or both pleural cavities. At the autopsy we do not find the hepatic cirrhosis which has in many cases been supposed to exist, but instead, a total obliteration of the pericardial sac, with which is often associated a nutmeg liver (sometimes atrophic, *vide supra*, page 330), and often, also, chronic peritonitis and chronic pleurisy. The peritonitis causes a marked fibrous thickening of the peritoneum, apt to be especially marked on the surface of the liver, in the form of what is called "frosted liver." This condition, therefore, in many cases, should be classed as one of "chronic inflammation of the serous membranes" (*vide supra*, page 289). We may have a similar group of symptoms not associated with inflammation of the peritoneum, if the circulatory disturbance resulting from the obliteration of the pericardium affects chiefly the portal circulation. We have also observed a combination of obliterative pericarditis with genuine hepatic cirrhosis. In a word, the cases are not all after one pattern. The diagnosis can not be correctly made unless one is acquainted with the symptom-complex, and pays special attention not only to the history of the case but to the cardiac signs, such as increase in the area of dullness, absence of apex-beat, or presence of systolic retraction.

3. **Tubercular Pericarditis.**—Tubercular pericarditis is an important disease clinically, since in many cases it is apparently primary. It may be either quite acute or chronic. The patient falls ill suddenly, or more gradually, with indefinite thoracic symptoms, dyspnœa, general weakness, moderate fever, etc. If the disease is of long duration, there is more or less œdema. When we find on physical examination, in such cases, the signs of pericarditis, the diagnosis of tubercular pericarditis is probable, if we discover a general "phthisical habit," hereditary predisposition, and also co-existing disease of other serous membranes, especially pleurisy, or more rarely chronic peritonitis. In the latter case the tubercular pericarditis forms one symptom of the so-called tuberculosis of the serous membranes, but, as has been said before, apparently isolated primary tubercular peri-

carditis does occur (*vide supra*). We have seen such cases repeatedly, especially in old people. In these cases the disease is not easy to diagnosticate. The patient gives one the impression of having heart disease, but the physical signs in the heart are sometimes of a very indefinite nature. Friction-rubs may be entirely absent, on account of adhesions or of large effusions. This leads to confusion with myocarditis or mitral stenosis. In other cases, of course, all the physical signs of pericarditis mentioned above may be manifest, and a correct diagnosis can be made.

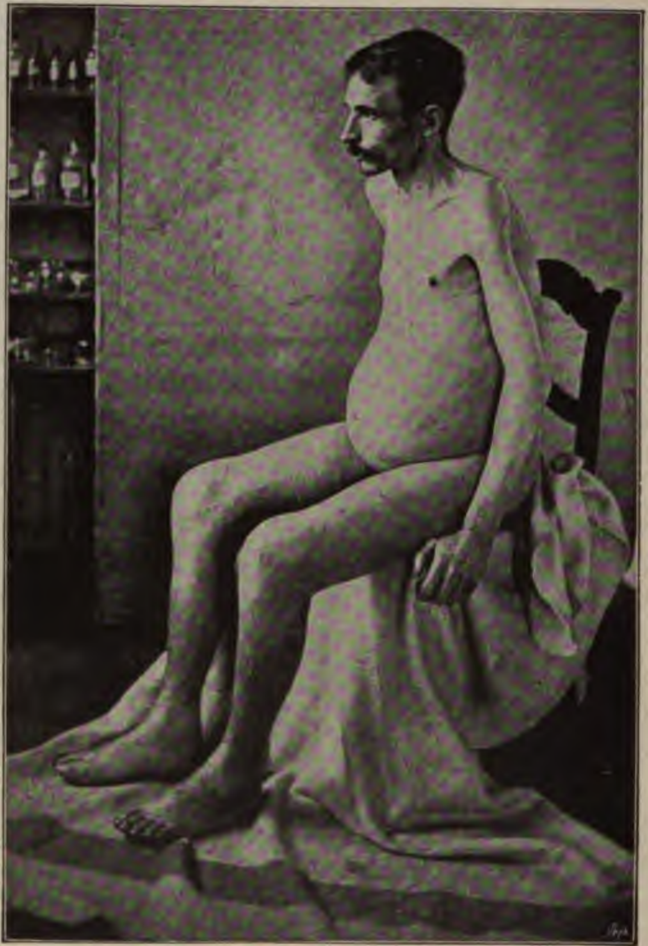

FIG. 43.—Pseudo-hepatic cirrhosis due to pericarditis. (Personal observation.)

Diagnosis.—From what precedes, it follows that the diagnosis of pericarditis is very easy in many cases, but is very difficult or impossible in others. The most unequivocal sign is the characteristic friction-rub. The practiced ear can often distinguish it from an endocardial sound by its quality. The pericardial sound is a rubbing, grating noise, near the ear; the endocardial is blowing, distant from the ear. The following features may serve as marks of distinction in doubtful cases: 1. We hear the pericardial sounds at first, and also later, over the base of the heart in the vicinity of the pulmonary valve; the endocardial are often loudest at the apex. 2. The pericardial murmurs are not so closely associated with the phases of the heart's action, with systole and diastole, as the endocardial. 3. We find that the pericardial sounds are not transmitted far. A loud rub may be audible at one spot which can not be heard a few centimetres away. Loud endocardial murmurs, however, are audible over almost the whole heart. 4. Sometimes the peculiarity of the pericardial murmur—that it becomes louder when the patient sits up, on pressure with the stethoscope, etc.—may be of diagnostic value. In many cases the loud, functional, so-called anæmic murmurs over the base of the heart may give rise to confusion with pericarditis.

If no pericarditic friction has been heard during any part of the illness, it is seldom possible to make an absolute diagnosis of pericarditis. At least, we must confess that we have made many errors in the diagnosis of such cases. Important factors to be considered are the general course of the disease, including the acute onset and præcordial pain, and also the shape of the cardiac dullness (triangular), and the signs furnished by the apex-beat, the pulse, and the heart-sounds. We have already pointed out the possibility of confusion between pericarditis and myopathic disease of the heart, and mitral stenosis if unattended by murmur. No rules of universal application in these cases can be laid down. It is very often advisable, and absolutely devoid of danger, to make an exploratory puncture in doubtful cases with a hypodermic syringe. This is done at the left edge of the sternum, or a little way in from the left limit of cardiac dullness.

Course and Prognosis.—Many cases of pericarditis in articular rheumatism, pneumonia, or heart disease, and also many of the rare and apparently primary forms, may recover completely. The disease lasts, in the mild cases, only about a week, in severe cases much longer.

Many cases of pericarditis, however, terminate fatally. The unfavorable issue depends either upon the severity of the primary disease, or upon the intensity of the pericarditis itself. In extensive croupous pneumonia, in valvular disease of the heart, or in severe chronic nephritis, an attack of pericarditis is often the terminal affection—the immediate cause of death. In otherwise healthy persons, however, a severe pericarditis with a large effusion may be the direct cause of death, as a result of the impairment of the movements of the heart. Yet one need not wholly despair, particularly in rheumatic pericarditis, even in cases which seem very bad. The prognosis of tubercular pericarditis is always very unfavorable. This form may, indeed, run quite a chronic course, but it is hardly ever capable of definite recovery. The prognosis of pyæmic pericarditis is also unfavorable.

In one class of cases pericarditis takes a chronic course from the start, or chronic pericarditis develops from an acute attack. The ultimate prognosis of these cases is usually unfavorable, since the secondary atrophy and dilatation of the heart gradually lead to severe disturbances of the circulation. We have spoken above of the termination of pericarditis in obliteration of the pericardial sac.

Treatment.—Since pericarditis is a severe affection under all circumstances, we must see especially that the patient has perfect rest and care. Extreme caution must be enjoined upon him, especially in the cases in which at first the subjective symptoms are slight. We must keep the patient strictly confined to the bed, and not let him leave it even temporarily.

The remedies which are used against pericarditis aim partly at keeping the inflammation in check, and partly at aiding the action of the heart. For the first, the continued application of ice to the cardiac region deserves especially to be recommended. Local blood-letting, ten or twelve leeches to the cardiac region —formerly very often but now more rarely used—may, in otherwise strong and healthy persons, afford great relief in cases with marked subjective symptoms.

Painting with tincture of iodine and blistering, however, deserve little confidence. Digitalis is our chief means to bring down an accelerated pulse, and to strengthen the heart's action. It is a drug which is most active and most frequently used in pericarditis, and is always indicated when the pulse is rapid and of diminished tension. Of course, the action of the remedy must be carefully watched, as in all cases where digitalis is prescribed. Tincture of strophanthus is also useful. As a palliative, morphine often does indispensable service when the subjective symptoms are marked and the patient is very restless. Diuretics are also employed, as in pleurisy with effusion (*q. v.*).

If the symptoms are threatening, the question arises whether a large fluid pericardial effusion is the cause of the severe symptoms. In this case the evacuation of the exudation is of course imperatively indicated. The difficulty of forming a correct opinion, however, is very great, because in any individual case it is rarely possible to determine the amount of fluid that may be present. In the first place, we must consider the size of the cardiac dullness and the weakening of the movements of the heart, but both factors may give rise to deception. Hence we always first make an exploratory puncture with a Pravaz's hypodermic syringe. The best point for insertion is, in general, the sternal end of the fourth or fifth [left] intercostal space with the patient on his back, partially raised in bed. If the exploratory puncture gives a positive result, we make a puncture with Billroth's, Fraentzel's, or some similar trocar. With regard to the details, we will refer to the description of puncture of the pleura. Puncture of the pericardium is always performed by the aid of aspiration. It is less dangerous than might be feared. Even injuries to the heart during the operation have scarcely ever had grave results. The temporary relief to the patient, in cases of successful puncture, is usually very striking, but the permanent results of pericardial puncture are, of course, much less favorable than those of puncture of the pleura, which is chiefly due to the character of the underlying disease. In some cases of purulent pericarditis, incision of the pericardium has also been practiced after the analogy of the treatment of empyema. In other forms of pericarditis with an abundant fibrino-serous exudation, it might often be preferable to make an incision rather than to aspirate, but there has not yet been much experience with the former method.

[The experiments of Rotch show that pericardial effusion causes dullness in the fifth right interspace, a sign which he thinks is not produced in cardiac enlargement. So far as is known to the editor, he is the only person who has acted on this observation, and punctured on the right of the sternum for pericardial effusion. In the case referred to, the signs pointed to a very large acute effusion of rheumatic origin. The first puncture was made in the fifth left interspace, but only about an ounce and a half of bloody serum was obtained. The needle was then withdrawn and immediately inserted in the fourth right space near the sternal border with absolutely negative result. The gravity of the symptoms led to two more punctures on the left some days later, one with small though positive result, the other with negative. Absorption and recovery ultimately took place.

The recommendation of the author to make a preliminary puncture with a hypodermic syringe for diagnostic purposes seems unnecessary, just as with pleural effusions.]

If there is a condition of cardiac weakness, stimulants are indicated—strong wine, subcutaneous injections of ether or camphor, or wine of musk. We try to keep up the patient's strength by the best of nourishment.

The resulting conditions of disturbance of the circulation, such as œdema, in chronic pericarditis, are treated in the same way as in valvular disease (*vide supra*). Digitalis and diuretics are the chief remedies.

CHAPTER II

HYDRO-PERICARDIUM, HÆMO-PERICARDIUM, AND PNEUMO-PERICARDIUM

1. Hydro-Pericardium

(Dropsy of the Pericardium)

The collection of a serous transudation in the pericardial sac, without any inflammatory symptoms in the serous membrane itself, we term hydro-pericardium, or dropsy of the pericardium. Dropsy of the pericardium, which formerly played quite a great rôle in pathology, is never a disease of itself, but is always a secondary condition. It may occur in anæmic and cachectic persons as a result of hydræmia, but it usually depends upon a local or general venous stasis in the pericardium. In the latter case the hydro-pericardium is one symptom of general dropsy, and hence is found chiefly in heart disease, renal disease, or pulmonary emphysema.

The clinical symptoms of hydro-pericardium are only exceptionally distinct, being obscured by the underlying affection. Large amounts of fluid in the pericardial sac, which may amount to a quart (a litre) or more, must of course impair the action of the heart, weaken the heart-beat objectively, and cause an increase in the cardiac dullness. The distinction from pericarditis is rendered possible by the absence of a friction-rub, but especially by attention to the existence of an underlying disease. In other respects the distinction between a pericardial transudation and an effusion during life is not always easy.

The prognosis and treatment depend wholly upon the nature of the underlying disease. Only exceptionally do we need to puncture, when the exudation is very large.

2. Hæmo-Pericardium

(Blood in the Pericardial Sac)

In rare cases hæmorrhages occur into the pericardial sac. The source of the hæmorrhage is most frequently an aneurism of the aorta, which perforates into the pericardium. Other causes of hæmorrhage are the bursting of aneurisms of the coronary arteries and rupture of the heart. The latter has been seen after injuries, and also as a result of cardiac aneurism and the cicatricial formations in myocarditis (see myocarditis). Finally, direct injuries to the heart, especially bullet-wounds, may also cause hæmorrhages into the pericardial sac.

In most cases death occurs in a few moments from compression of the heart, when a hæmo-pericardium comes on. Hence the amount of blood poured out into the pericardial sac is usually not very considerable. Only in the cases in which the blood oozes out more slowly can a great distention of the pericardial sac be reached. The diagnosis is rarely possible. With regard to treatment we can merely note that, in some traumatic cases, the aspiration of the blood has been performed with success.

3. Pneumo-Pericardium

(Air in the Pericardial Sac)

The entrance of air or gas into the pericardial sac has been observed in rare cases, apart from external wounds, as a result of the perforation of a pyo-pneumothorax, or of some other suppurating process in organs that contain air. Thus cases are known in which the rupture into the pericardial sac comes from the œsophagus, as in cancer; from the stomach, in cancer or ulcer; or from the lungs, in tubercular or gangrenous cavities. Since the agents of inflammation enter the pericardium along with the air, a purulent pericarditis almost always develops, besides the pneumo-pericardium, or it may rarely be simply a sero-fibrinous pericarditis.

The most characteristic and striking sign of pneumo-pericardium is the presence of a metallic sound, due to the movements of the heart. Either the heart-sounds themselves, or some existing friction-rub, may acquire a metallic timbre from the increased resonance, or splashing metallic sounds may be produced in the pericardial sac from the movements of the air and the fluid, which may often be heard at a distance from the patient. In regard to diagnosis, however, it is important to know that signs similar to those of metallic resonance in the heart may arise from the stomach, when it is drawn or pushed upward.

In true pneumo-pericardium percussion gives a more or less complete absence of the cardiac dullness. On rod-percussion (see page 297) a metallic sound is sometimes heard, whose pitch may vary somewhat with the phase of the heart's action. If fluid is also present in the pericardial sac besides the air, the dullness caused by this will rise on raising up the patient.

The other symptoms of the disease and the treatment are the same as in a severe pericarditis. The prognosis, however, corresponding to the primary disease, is wholly unfavorable.

SECTION III

DISEASES OF THE VESSELS

CHAPTER I

ARTERIO-SCLEROSIS

(*Endarteritis chronica deformans. Atheroma of the Vessels*)

Ætiology.—Atheromatous degeneration is probably in most cases an expression of the wearing out of the arteries, because of the many ways in which the intima of the arteries is damaged during life, both chemically and mechanically. Atheroma of the arteries is then mainly a change incident to age, and as such is often regarded as not a true disease.

On the other hand, if arterio-sclerosis appear in earlier life, say before the fortieth year, we must look for such circumstances as would tend to make the ordinarily efficient causes unusually active. We must not forget that there is an apparent predisposition to the condition, which often seems to be hereditary; but without doubt there are more appreciable causes. The reason that arterio-sclerosis is so common and occurs comparatively so early in hard laborers, is the great mechanical strain which the arteries undergo during persistent, severe, bodily exertion. Again, in diseases which are associated with a persistent elevation of arterial tension, such as hypertrophy of the left ventricle in interstitial nephritis, arterio-sclerosis develops probably as a result of the constant high tension. It must be confessed that here a decision is often very difficult, inasmuch as the arterial disease may be simultaneous with the nephritis, or even the actual cause of it. Again, excessive chemical irritation must be considered, as experience shows that a continued excess in eating and drinking, particularly in the use of alcohol, is apt to occasion arterio-sclerosis; and in the same connection are to be named such constitutional diseases as syphilis, gout, and chronic nephritis, and perhaps also definite poisons such as lead. Nor can we disregard the possibility that the disease may follow persistent mental strain and excitement. All these circumstances explain why well-marked atheroma is far more frequent in men than in women.

Pathological Anatomy.—Atheroma is almost exclusively confined to the arteries; only exceptionally do like processes occur in the veins. Among the arteries the aorta is almost always the most intensely and extensively diseased; we also find disease in the iliac and femoral arteries, the brachial, radial, and ulnar, the coronary arteries of the heart, and the arteries of the brain. In some of the other arteries, however, like the gastric artery, the hepatic, and the mesenteric, we very rarely find atheromatous changes.

The atheromatous process is easy to recognize macroscopically. Instead of the normal smooth internal surface, we find more or less numerous irregularities and thickenings on the intima, which appear either more or less gelatinous and translucent, or dense and fibrous, or ossified as a result of calcification, in which case they also feel perfectly hard. In many cases we find the surface of the thickenings destroyed—atheromatous ulcers—and covered with masses of fibrine.

The wall of the arteries is usually increased in thickness, making the vessel feel stiff and hard. Very frequently the calcareous plates may be felt by the finger. The frequently associated increase in arterial tension gradually stretches the walls of the arteries, rendering them tortuous. This is best seen in those of medium size, such as the brachial and radial.

Microscopic examination shows that the chief changes are situated in the intima of the arteries. This appears three or four times as thick as normal, partly from the swelling of its elements and partly from the new growth of connective tissue and the deposit of round cells. In the connective-tissue cells of the intima, and in the endothelial cells of its surface, we usually find a marked fatty and hyaline degeneration, to which the yellowish, translucent appearance of the surface is due. Finally, in the deeper layers there is a complete breaking down of the tissue into a mixture of fat, detritus, and cholesterine crystals, which has given the whole process the name of atheroma [=pulp]. If this destruction extends to the surface, an atheromatous ulcer is formed. In other places, however, it does not reach ulceration, but the superficial layers of the intima become sclerosed, and are finally changed to lamellæ of bony hardness from the deposition of lime-salts. The atheromatous spots on the intima of the vessels often give rise to the formation of parietal thrombi.

The media and adventitia of the arteries also show changes, in the later stages of the process. Here, too, we may finally get fatty degeneration and calcification. In other cases, however, there is a marked atrophy of the media.

The immediate result of the atheromatous changes is a loss of elasticity in the walls of the vessels. The ability to resist the blood-pressure is reduced, and this is why diffuse or circumscribed aneurismal dilatations of the vessels so often arise as a result of arterio-sclerosis (see the following chapters).

Another result of extensive atheromatous degeneration of the vessels is an increase of the resistance to the blood-current, and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor for the propulsion of the blood. The left ventricle, in consequence of these additions to its task, becomes almost invariably hypertrophied in cases of extensive arterio-sclerosis, provided the general nutrition of the patient is still well maintained.

We see, therefore, how close the mutual relations are between increased arterial tension, hypertrophy of the left side of the heart, and arterio-sclerosis.

The thickening of the intima in the smaller vessels often causes so marked a diminution of the blood-supply that secondary disturbances of nutrition are not wanting in the various organs. The lumina of the vessels may be still further narrowed, or even completely closed, by the formation of thrombi on such portions of the wall of the vessels as have undergone atheromatous changes. We have already in part learned to recognize the sequelæ which necessarily arise in the various organs, such as indurations in the heart as a result of atheroma of the coronary arteries, and we will return later on to the analogous changes in some other organs, such as cerebral softening and certain forms of contracted kidney.

Clinical Symptoms.—In order to decide whether an arterio-sclerosis is present in the living subject, we are of course restricted exclusively to the examination of those peripheral arteries that are accessible to palpation. We must examine, first of all, the radial, brachial, femoral, and temporal arteries. If there is atheroma, we feel the hard and partly calcified vascular tube. In marked cases we have a feeling, especially in the radial, as if we had hold of a goose's neck. We sometimes notice a diffuse dilatation of the femoral arteries. In many cases the marked spiral form of the vessels is very striking, and it is, as we have said, a direct result of the loss of elasticity of their walls and of the increased blood-pressure. The spiral form is most frequently observed in the temporal, brachial, and radial arteries.

Although we can often directly demonstrate atheroma in the vessels mentioned, we must always be cautious in deciding from this that there is also an atheroma of the internal arteries, for the radial arteries often feel very rigid, while the autopsy later on shows little or absolutely no atheroma of the internal arteries. In other cases, however, we find at the autopsy marked atheromatous changes in the arteries of the brain, the kidneys, the heart, etc., although the external arteries during life felt perfectly normal. We see from this how hard it is to make an absolute diagnosis of general arterio-sclerosis.

It is impossible to give a uniform picture of arterio-sclerosis, since its results appear now chiefly in this organ and now chiefly in that, whereby entirely distinct types of disease arise. Hence we must confine ourselves here only to mentioning briefly the most important sequelæ. For the most part, they are described separately in other portions of this work.

In the heart we find a hypertrophy of the left ventricle as a result of the increased resistance to the arterial circulation. This is often apparent during life from the strength of the apex-beat and its displacement to the left, and also from the extension of the area of cardiac dullness to the left. On auscultation, the increased tension in the aortic system is made manifest by the strength of the aortic second sound. The examination of the heart, however, is often rendered difficult by the presence of pulmonary emphysema. On the other hand, we sometimes can not decide how far a manifest hypertrophy of the left ventricle is due to an arterio-sclerosis, and not to other co-existing processes, such as contracted kidney. We often find other anatomical changes in the heart besides hypertrophy of the left ventricle. We have already spoken of the important and interesting results of atheroma of the coronary arteries, the formation of the so-called indurations of myocarditis in the heart (see page 340 *et seq.*). Sometimes, from an invasion of the aortic valves by the atheromatous process, we get an insufficiency, or much more rarely a stenosis of the aortic orifice. Finally, we may also mention here that atheroma, especially of the ascending aorta or the arch, is the commonest cause of the formation of aneurism of the aorta.

We have already described the character of the peripheral arteries. The radial pulse is hard and tense, and the wave is either quite large, or, when the tube is very narrow, small. Since the wall of the vessel contracts only slowly, in consequence of its loss of elasticity, the radial pulse is usually sluggish—*pulsus tardus.* This condition is also pronounced in the sphygmographic tracing, which shows a slow ascent, and a still slower descent, of the pulse-curve, and an absence of the elevation in the descending limb of the curve, due to the normal elasticity. The frequency of the pulse is quite different in different cases; it is often rather slow as a result of sclerosis of the coronary arteries. The pulse is very often irregular as a consequence of changes in the heart. We sometimes find an abnormal delay in the radial pulse, or in the pulse in other arteries, in comparison with the heart-beat, from the lessened rapidity of transmission of the pulse-wave.

Besides the heart, the brain is the chief place in which we observe definite results of arterial sclerosis. The increased tendency to rupture which the atheromatous vessel-walls show, and the co-existing heightened blood-pressure, explain the comparatively frequent occurrence of cerebral hæmorrhages. Cerebral hæmorrhages often result from little miliary aneurisms, which have formed in the atheromatous cerebral arteries. Atheroma is also the most frequent cause for the formation of foci of softening in the brain, since the arterial changes may give rise to a closure of the cerebral arteries from thrombosis or embolism. Finally, so-called senile dementia, or, at any rate, many cases of that condition, are referable to cerebral atrophy, resulting from the faulty nutrition of the brain through the sclerosed arteries.

In the kidneys, too, atrophic processes often develop from the diminution of the blood-supply owing to the narrowed lumina of the vessels, and they lead to a special form of contracted kidney. The origin of the granulated "senile kidney" is in large part atheroma of the renal arteries.

The lower extremities display symptoms of arterio-sclerosis less often than the parts already mentioned, but if present the symptoms may be very striking. Here belongs a peculiar group of symptoms, which has been specially studied by Charcot, Erb, and others. It is termed intermittent claudication or lameness, or *dysbasia intermittens angiosclerotica* (Erb). It is sometimes associated with lesions of one or both femoral or iliac arteries, more often with obliterative arteritis of the distal arteries of the lower leg and foot. It usually begins with disagreeable paræsthesiæ of crawling, tickling, or coldness in the feet and calves. If the patient walks even for a short time the symptoms increase, the feet grow cold and blue, there is actual pain, and the patient becomes completely incapable of walking farther. After a short pause he can resume his walk, but soon he has to stop again. Upon examination we find the feet and legs cold and bluish-red. If we palpate the arteria dorsalis pedis, or the tibialis postica, we can detect no pulsation whatever. This is the diagnostic sign. The course of the trouble is chronic, but decided improvement may ensue under correct treatment (*vide infra*). From a prognostic point of view there is an unfavorable tendency to the development of gangrene of the toes, as the arteritis increases. With regard to causation, an important part in the development of intermittent lameness is played by syphilis, excess in tobacco, glycosuria, damage from thermic influences, and other causes.

Sometimes gangrene of the lower extremities occurs as a result of marked atheroma without any previous appearance of intermittent lameness. So-called "spontaneous gangrene," senile gangrene, and diabetic gangrene are probably, without exception, due to primary lesions from arteritis and consequent impairment in the arterial blood-supply.

All this shows how different the symptoms of arterio-sclerosis may be in different cases. The symptoms in the vascular apparatus often predominate over all others. The heart, which is simply hypertrophied, or which has undergone in part cicatricial degeneration, is finally paralyzed, and then all the symptoms of a chronic heart disease develop—dyspnœa, œdema, etc. If there is also albuminuria, a type of disease is produced which resembles that of contracted kidney. In other cases, however, the symptoms in the brain are especially manifest, either alone or in combination with the other symptoms mentioned, and, finally, in rare cases we have the above-described symptoms relating to the lower extremities.

We must remark, however, in conclusion, that all the results of arterio-sclerosis mentioned may be absent for a long time or altogether. Many persons have practically no symptoms at all from their arterio-sclerosis, and reach an advanced age, but we must always consider the possibility of the sudden occurrence of severe symptoms, and make our prognosis accordingly.

Treatment.—If we recall the above-mentioned causes of arterial sclerosis, we see at once the possibility and importance of prophylaxis, and of course if the disease already exists we must endeavor to avert these injurious influences, so as to limit the progress of the disease as far as possible. There is little direct influence upon the arterio-sclerotic process to be expected from drugs. Iodide of potassium has won the highest reputation in this regard, and it is employed extensively in all cases of arterio-sclerosis. Probably it is most efficient in those cases which are associated with a syphilitic taint. In general, treatment must be symptomatic, according to the indications in each instance. In the symptom-complex known as intermittent lameness, Erb recommends iodide of potassium, or iodide of sodium; local applications of warmth (for instance, by means of the warm pack); galvanic foot-baths; such cardiac tonics as strophanthus; and careful regulation of bodily exercise. If gangrene has set in, prompt amputation of the necrotic part is necessary, for we have no means of influencing the process itself. In each case treatment must be adapted to the sequelæ which ensue. Prophylaxis is important. It may hinder, or at least delay, the development of the process if the patient avoids the causes already enumerated.

CHAPTER II

ANEURISM OF THE THORACIC AORTA

Ætiology and Pathological Anatomy.—The circumscribed dilatation of an artery is termed an aneurism. The cause of its formation is almost always to be sought in a primary disease of the vessel-wall, which weakens its resistance to the blood-pressure. As we have already said in the previous chapter, it is chiefly arterio-sclerosis which lies at the foundation of aneurisms in many cases. The same factors, therefore, which favor the origin of arterio-sclerosis belong to the ætiology of aneurism. This explains why the root of the aorta, where arterio-sclerosis is most marked, is also the most common seat of aneurism, and further, why aneurisms are especially seen in advanced life and in men. Still, the development of aneurism is a rare occurrence in comparison with the great frequency of arterio-sclerosis; so that there are probably other circumstances of importance which we shall some day know more about. Possibly there is a congenital weakness of certain portions of the arterial wall. Traumatic influences, also, are not wholly out of the question. Lately many authors have laid special weight on syphilis affecting the intima of the aorta. Indeed, syphilis is said by some to be by far the most frequent factor in the ætiology of aneurism of the aorta. This seems to us an exaggerated view, but certainly the point deserves consideration.

[The occurrence of aneurism in early middle rather than in advanced life shows that too much stress can be laid on atheroma as a cause. That sudden strain often plays an important part in the ætiology can scarcely be doubted, though the cases in which a perfectly healthy aorta yields locally to internal pressure must be very rare. It is highly probable that violent exertion tends to produce changes in the walls of the aorta. The far greater frequency of aneurism in the male sex is notable. Syphilis, gout, alcoholic excess, and lead-poisoning appear to be factors in some cases.]

The size of aneurisms of the aorta varies very much, of course, in different cases. They most frequently are about the size of an apple or the fist; but in rare cases much larger aneurisms are observed. According to their shape we distinguish the more diffuse or spindle-shaped dilatations from the saccular aneurisms (*aneurisma diffusum seu cylindricum, aneurisma fusiforme, et aneurisma sacciforme*). Intermediate forms and combinations of the different forms occur in manifold ways.

As we should expect from its origin, we never find the wall of the aneurism formed of a normal vessel wall. The intima almost always shows the same changes that are characteristic of arterio-sclerosis, only in a much higher degree. The media, too, is usually changed, and its muscular structure often shows fatty degeneration. The adventitia is usually thickened by chronic inflammatory processes. The media, and sometimes the intima, are in many cases so much atrophied that the wall of the aneurism, at least in part, is formed only of the adventitia.

In the cavity of the aneurism the blood is only partly fluid. We usually find it more or less full of new and old masses of thrombi. The oldest thrombi, which lie upon the wall of the aneurism, are firm, yellowish, adherent to the wall, and sometimes calcified. At other points the thrombi are softened and broken down. The most marked coagulation is usually found in the saccular aneurisms with a narrow entrance, because in this form of aneurism the blood is almost completely stagnant in the aneurismal sac.

Aneurisms of the aorta usually have their seat in the ascending aorta, or in the arch. Aneurisms of the descending thoracic and of the abdominal aorta are far more rare. The following description refers principally to aneurisms at the beginning of the aorta.

We do not give separate consideration to aneurism of the ascending aorta and of the arch of the aorta, respectively, for it is impossible clinically to draw a sharp distinction between them. Other aneurisms will be considered further on.

Clinical Symptoms.—The subjective sensations of the patient, relating directly to the aneurism, are of a very uncertain nature, and are often entirely absent. In other cases there is pain in the region of the aneurism, either only a slight sense of pressure, or very severe and subject to paroxysmal increase, sometimes rather localized, sometimes radiating into the back, the shoulders, and the extremities. Sometimes, too, the patient feels the beating and pulsation of the aneurism. The remaining symptoms of aneurism may be divided into two groups. The first group embraces those symptoms which are directly related to the aneurism itself, and most prominent in this group are the physical signs. The second group of symptoms includes the resultant phenomena which the aneurism occasions in the circulatory apparatus and by pressure upon the neighboring parts.

1. *Physical Signs.*—It depends entirely upon the position of an aneurism of the aorta whether it causes physical signs or not. Deep aneurisms, which nowhere approach the chest-wall, may, of course, be quite inaccessible to direct examination.

Aneurisms of the ascending aorta, however, and of the arch, often extend so near to the anterior wall of the chest that they cause an abnormal pulsation. We feel this most frequently at the sternal end of the second right intercostal space, or over the upper part of the sternum. The pulsation of an aneurism of the arch of the aorta may sometimes be felt in the root of the neck. It often occurs a moment later than the systole of the heart. In many cases the pulsation is clearly double, analogous to the normal dicrotism of the pulse. We sometimes feel a slight systolic thrill with the flat of the hand. In the rare aneurism of the descending thoracic aorta the pulsating swelling may make its appearance in the back, between the vertebral column and the left scapula. If the aneurism has reached a certain size, the pulsating part protrudes as a tumor. The protrusion is either merely slight, or in many cases it forms a large prominent swelling. It then shows usually a marked pulsation, not only from below upward, but also in a lateral direction, which is of diagnostic significance. In large aneurisms, however, the pulsation sometimes is only very weak, and scarcely perceptible, from the formation of many coagula.

[The Roentgen ray furnishes a most valuable means of searching for thoracic aneurism, as the latter casts a shadow which dilates with each cardiac systole.—V.]

The marked prominence of large aneurisms is possible only because the covering parts, not only the muscles and skin, but also the cartilages and bones, the ribs and sternum, are brought to a gradual atrophy and wasting by the persistent pressure. The skin over large aneurisms gradually becomes thinner and thinner, until finally it may even become necrotic.

There is of necessity more or less dullness on percussion over every aneurism of any size. The dullness is usually evident in the upper right intercostal spaces, or the adjacent parts of the sternum. Sometimes it even precedes the palpable pulsation, although then its significance is usually still very uncertain. In rare cases of aneurisms of the ascending aorta and of the arch, dullness and abnormal pulsation have been observed to the left of the sternum.

Auscultation gives varying results. In some cases (probably chiefly when many coagula form) we hear nothing at all over the aneurism. In other cases we hear one or two sounds, which are usually the audible heart-sounds transmitted. Perhaps a systolic sound may also arise from vibration of the wall of the aneurism. In other cases, we hear a murmur over the aneurism. A dull and usually not very loud systolic murmur often arises from the formation of eddies in the aneurismal sac. If we also hear a diastolic murmur, it is almost always due to a co-existing insufficiency of the semi-lunar valves of the aorta (*vide supra*).

2. *Sequelæ.*—An aneurism of the aorta by itself probably never causes such an increased resistance to the blood-current as to give rise to the development of a hypertrophy of the left ventricle. In the quite frequent cases in which hypertrophy of the left side of the heart exists, it may almost always be referred to a co-existing insufficiency of the aortic valves, and sometimes to very extensive atheroma of the arteries. During life a hypertrophy of the heart may be simulated, because the heart is pushed to the left by the aneurism.

In many cases the signs in the peripheral arteries are important. Marked inequality of the pulse in symmetrical arteries is often an especially valuable diagnostic sign. Either the trunk of an efferent vessel is compressed by the aneurism, or the lumen of the exit of the vessel is itself involved in the aneurism, and hence the opening of the vessel is distorted or contracted, or partly stopped by a coagulum. This readily explains why, in aneurism of the ascending aorta, the radial, and sometimes the carotid pulse, are plainly weaker on the right than on the left, as a result of implication of the trunk of the innominate, while in aneurism of the arch or of the beginning of the descending aorta the opposite condition may obtain. Abnormal differences, too, in the intensity of the pulse in the upper and lower halves of the body may arise under some circumstances.

[W. S. Oliver has described a valuable sign to which he gives the name of "tracheal tugging." In aneurism of the transverse arch with pressure on the left primary bronchus, a distinct downward pull, synchronous with the heartbeat, can be felt by the thumb and forefinger on either side of the inferior border of the cricoid cartilage, when the patient, in the erect or sitting posture and with the mouth shut, raises the chin as far as he can. The late R. L. MacDonnell, of Montreal, has shown that non-aneurismal tumors in this region are not accompanied by this sign, and that pressure on the trachea, from whatever cause, does not produce it. It may be the sole sign of aneurism present, and may then possess the double diagnostic value of indicating both the nature and exact seat of the disease.—EDITOR. Very exceptionally, tracheal tugging is caused by other conditions than thoracic aneurism.—V.]

A marked delay of the pulse in the arteries arising below the aneurism is a symptom that is occasionally seen. Thus, we see in aneurism of the arch of the aorta that the left radial pulse is later than the right, and that in aneurism of the descending aorta the pulse in the lower extremities is later than the radial pulse.

We see very striking signs in the veins if the large venous trunks in the thorax, the superior vena cava, or an innominate vein, are compressed by the aneurism. The veins swell in the neck, in the upper extremities, or upon the surface of the thorax, according to the seat of the compression. Local œdema may also be produced in this way.

The respiratory organs are exposed to the pressure of aortic aneurisms in many ways. Compression of the lungs by large aneurisms actually contributes toward increasing the dyspnœa in many cases. This may be still more distressing if the trachea be compressed. Of the two main bronchi, the left bronchus, which lies beneath the arch of the aorta, is more apt to be compressed. This produces the symptoms of a unilateral bronchial stenosis (*vide supra*). The comparatively frequent compression of one recurrent nerve, especially the left, is also of diagnostic importance, as it results in paralysis of one vocal cord.

Pressure on the vagus may occasion changes in the rate of the pulse, and perhaps sometimes attacks of dyspnœa. The dyspnœa in most cases, however, takes the form either of angina pectoris or of cardiac asthma, and is referable to the other coincident lesions of the heart and the coronary arteries.

Very prominent symptoms sometimes arise from compression of the intercostal nerves or branches of the brachial plexus by the aneurism. As a result of this pressure, extremely severe and distressing neuralgias are felt in the nerve territories affected, and sometimes we see motor paresis in the arm.

Finally, disturbances of deglutition arise in many cases from compression of the œsophagus. If this be falsely interpreted, it may lead to a mischievous use of the œsophageal sound. Cases have repeatedly occurred in which perforation of the aneurism has been caused by passing a sound into the œsophagus. Hence we must always remember this possibility in practice.

Course and Termination of the Disease.—Aneurisms may remain latent for a long time without causing any symptoms. In such cases a sudden perforation may lead to a speedy and unexpected death.

In the cases which have shown the above symptoms to a greater or less extent for a long time, and often for years, sudden death quite frequently results from rupture of the aneurismal sac and perforation into a neighboring organ. In perforation into the pericardium death follows almost instantly from cessation of the heart's action. In perforation into the œsophagus a fatal hæmorrhage occurs. In perforation of the aneurism into the air-passages (the trachea or bronchi) or into one pleural cavity, two factors, hæmorrhage and suffocation, unite in causing death. In aneurisms which gradually erode the anterior wall of the chest, the perforation is in rare cases external; but here a sudden, immediately fatal hæmorrhage seldom ensues; much more commonly a slowly increasing anæmia develops as a result of repeated slight hæmorrhages which may sometimes go on for weeks. Death then is due to the gradually increasing weakness, or to a final severe hæmorrhage. Perforation of an aneurism into the right side of the heart, into the pulmonary arteries, or the vena cava, is a rare termination. Here death does not follow at once, but severe general disturbances of the circulation, such as dropsy, soon arise. In many of these rare cases peculiar physical signs also appear —a venous pulse, a loud systolic murmur over the point of perforation, etc.

If, in patients with aneurism of the aorta, death does not ensue from a sudden perforation, the general type of the disease takes a form similar to chronic heart disease. The aneurism, as we have said, is often also combined with aortic insufficiency. The left ventricle gradually becomes paralyzed, and the well-known disturbances of compensation set in—increasing dyspnœa, œdema, etc. In other cases the patient gradually becomes duller and weaker from the distressing pain, the sleeplessness, and the other symptoms, and dies with the signs of increasing general weakness.

Recovery from aneurism of the aorta scarcely ever occurs.

Diagnosis.—The diagnosis of aneurism of the aorta can in many cases be made with great ease and certainty, but in other cases it is very difficult and even impossible. If the direct physical signs are plain, especially if we feel an abnormal pulsation, we shall not be apt to commit an error; but the diagnosis presents great difficulties in those cases in which the aneurism is not accessible at all, or accessible only with great difficulty, where it merely causes indefinite symptoms, pain in the chest, occasional oppression, symptoms of pressure on neighboring organs, etc. A very stubborn intercostal neuralgia, which no remedy can relieve, may be for a long time the only symptom, often misinterpreted, of a latent aneurism. The disease is often not recognized, however, because in such cases we do not generally think of the possibility of an aneurism, and hence we neglect a careful examination of the heart and the arteries, and also the search for other symptoms of compression, such as paralysis of the vocal cords; but sometimes, even with the most careful examination, the diagnosis can not amount to more than a suspicion.

If there is a loud systolic murmur in the second intercostal space on the right without other decisive signs, it may be difficult to make a differential diagnosis between aneurism and aortic stenosis.

The distinction between aneurism and other tumors in and about the thorax sometimes presents difficulties in diagnosis. Mediastinal sarcomata and abscesses, circumscribed empyemas, tumors arising from the sternum, or new growths in the lungs and bronchial glands, may all give rise to confusion. We can scarcely lay down any general rules for diagnosis, since the conditions differ in almost every case. If we feel a swelling, its pulsation is the symptom which points most to an aneurism, but we must be certain that the pulsation is not merely transmitted, but that it really takes place in all directions within the swelling itself. We must also consider the auscultatory symptoms, the condition of the heart and the arteries, and any symptoms of compression; yet in such cases we can not always make a definite diagnosis.

Treatment.—Many attempts have been made to bring about an obliteration of the aneurism, and thus a recovery. Although the methods of treatment aiming at this have obtained decisive results in the aneurisms of peripheral arteries, their results in aneurism of the aorta are still of a very doubtful character; yet we are always justified in any given case in trying one of the methods recommended.

Persistent compression by a pad can of course be employed only in those cases where the aneurism projects at one part of the chest-wall. The pressure, however, usually causes great pain, and hence is ill borne.

Tying the carotid, the subclavian, or both vessels, has also been repeatedly performed in aneurism of the arch of the aorta, sometimes with apparent success, but oftener without any benefit.

"Acupuncture" of the aneurism (Velpeau) consists in inserting a needle or an iron wire into the aneurismal sac in order to excite coagulation in it. The results obtained by it in aneurism of the aorta are not very encouraging.

Better results are reported from galvano-puncture. Two needles inserted into the aneurism are connected with the poles of a galvanic battery, by which a weak current is passed through the aneurism. Here we must regard the chemical and electrolytic action of the current as well as the mechanical action of the needles.

Injections of chemical substances into the aneurismal sac, in order to produce coagulation, are dangerous, since the coagula caused by them may give rise to emboli. Hence we have abandoned making trial of liquor ferri sesquichloridi and similar substances. We can better recommend injections of ergotine into the vicinity of the sac, two to five grains (gramme 0.1–0.3) of the aqueous extract of ergot dissolved in water or glycerine, injected every day or two. This method was first employed with success by Langenbeck in peripheral aneurisms. Its action depends upon a contraction of the smooth muscles in the wall of the aneurism, caused by the ergotine.

We can expect little action on an aneurism from the use of internal remedies, although favorable results have been reported. Especially recommended is the long-continued use of iodide of potassium or iodide of sodium, drugs which have obtained a great reputation in all arterio-sclerotic lesions and, in particular, in syphilitic disease of the arteries (*vide supra*, ætiology of aneurism). Yet it is easy to see why one should not expect too much from the iodides. Acetate of lead, 5 to 10 grains (gramme 0.3–0.6) daily has also been employed. Its value, however, is questionable.

The symptomatic treatment of aneurism, which tries to relieve the patient's sufferings, and the dietetic measures prescribed, follow the generally customary principles. In a rupture of the aneurism externally we try to avert the fatal catastrophe by absolute rest, ice-bags, styptic cotton, etc. Treatment is powerless against internal perforations.

[Tufnell's method, so-called, which has given good results in abdominal and peripheral aneurisms, proves sometimes useful in palliating the symptoms and lengthening the course of aortic aneurism. The aim of this method is to diminish the force and rapidity of the circulation, and, if possible, to increase the fibrinous deposit. It is carried out by enforcing absolute rest in the recumbent position, and by limiting the amount of food, especially of liquids. About ten ounces of solid food and eight of liquid are allowed daily, divided into three meals.]

CHAPTER III

ANEURISMS OF THE OTHER VESSELS

Aneurism of the Abdominal Aorta.—Its favorite seat is the vicinity of the cœliac axis. In many cases it may be felt through the abdominal wall as a pulsating tumor, over which a systolic sound or a whirring murmur can be heard. The possible symptoms of compression are very numerous. The stomach, intestine, and liver (jaundice) may be implicated. Pressure of the aneurism upon the nerve trunks, or even pressure on the spinal cord after gradual erosion of the vertebræ, with consequent severe neuralgia, paralysis, etc., has been repeatedly observed. Death usually ensues from rupture of the aneurismal sac and internal hemorrhage.

Aneurism of the trunk of the innominate is rare. Its symptoms are very much like those of an aneurism of the arch of the aorta. If we feel a pulsating tumor, it is usually situated somewhat higher up than the aneurism of the aorta, in the first right intercostal space, or the tumor even extends into the supra-clavicular fossa. In rare cases aneurisms of the subclavian and of the carotid have been observed. We have ourselves seen an aneurism of the internal carotid the size of a cherry pressing on the Gasserian ganglion, which caused an extremely severe trigeminal neuralgia lasting for years.

Aneurism of the pulmonary artery may appear as a pulsating tumor in the second left intercostal space. It is usually impossible to distinguish it with certainty from an aneurism of the aorta.

We have already mentioned, in the description of pulmonary tuberculosis, the great importance of small aneurisms of the branches of the pulmonary artery in pulmonary cavities, as a frequent cause of hæmorrhage.

Aneurisms of the arteries of the brain, which are relatively most frequent in the basilar artery and the artery of the fissure of Sylvius (the middle cerebral artery), may cause severe cerebral and bulbar symptoms (see page 1052). As has already been mentioned, miliary aneurisms of the cerebral arteries play an important part in the ætiology of cerebral hæmorrhage (*q. v.*).

The symptomatology and treatment of aneurisms of the peripheral arteries belong to the domain of surgery.

CHAPTER IV

RUPTURE OF THE AORTA

A RUPTURE of a previously healthy aorta, with fatal hæmorrhage, after violent traumatic influences, has been seen only in a very few cases. In the majority of the very rare cases of rupture of the aorta we have to do with a vessel that is already atheromatous. In some cases a special exciting cause is present, and in others it is absent. We once saw sudden death caused by rupture of the ascending aorta in a young man about twenty-five, who before that seemed perfectly healthy. No trace of atheroma was found; but at the point of rupture there was a slight protrusion and a decided thinning of the wall, which was probably congenital. The formation of a so-called dissecting aneurism, which has often been seen in the aorta, is of anatomical interest. Here only the intima and media are torn. The blood burrows between them and the adventitia or between the layers of the media. Most of the cases of dissecting aneurism of the aorta also result, like rupture of the aorta, in sudden death. In many cases death results from the secondary perforation of the aneurism into the pericardium. On the other hand, a sort of recovery from dissecting aneurism may occur. A secondary perforation takes place into another part of the aorta itself (Boström). Cases of this sort were formerly more than once mistaken for double aorta. If the capsule containing the blood is preserved for a considerable length of time, the symptoms may assume the character presented in ordinary aortic aneurism.

CHAPTER V

NARROWING OF THE AORTA

CONGENITAL narrowness of the aorta and its branches is a condition to which Rokitansky first, and later Virchow, have directed attention. We find this anomaly especially in those, mostly women, who during life have shown the signs of persistent chlorosis. Sometimes such persons are backward in their whole development; they retain a puerile habit, and show a defective development of the genitals. They often suffer from palpitation, faintness, and a tendency to hæmorrhages. In many cases the heart is also small, but in others it is dilated and hypertrophied. We have already pointed out (page 350) that congenital hypoplasia of the aorta may be regarded as the cause of certain cases of "idiopathic hypertrophy of the heart." Valvular disease of the heart has been repeatedly found combined with general narrowness of the arterial system. During life this anomaly of the vascular system may sometimes be suspected, but it can never be recognized with certainty.

Narrowing of the aorta at the point of insertion of the ductus arteriosus is a lesion observed in rare cases, whose origin probably always falls in the period directly after birth, and is associated with obliteration of the fœtal ductus arteriosus. Other congenital anomalies of the heart are usually present at the same time. If the narrowing of the aorta is not very marked, it may be properly equalized by a secondary hypertrophy of the left ventricle and the development of a collateral circulation. The latter is brought about by dilatation of the anastomoses between the first intercostal artery, the dorsalis scapulæ, the subscapularis, and the transversalis colli on one side, and the lower intercostal arteries, which come off from the descending aorta below the narrowing, on the other. Anastomoses are also formed between the mammary and the superior epigastric on one side and the lumbar and femoral arteries on the other. During life the dilated arteries are prominent, in part abnormally tortuous, and perceptibly pulsating, especially the dorsales scapulæ, the subscapulars, the mammaries, and the epigastrics. In some cases a systolic murmur has been heard over some of these vessels. The pulse in the arteries of the lower extremities, the femoral and popliteal, is very weak and scarcely perceptible.

In many cases the collateral circulation is so complete that the person affected may feel no subjective disturbance at all, and may attain an advanced age, but in other cases disturbances of the circulation appear sooner or later, and the patient finally succumbs to dropsy. Sudden death from rupture of the heart or of the aorta has also been observed.

DISEASES OF THE DIGESTIVE ORGANS

SECTION I

Diseases of the Mouth, Tongue, and Salivary Glands

CHAPTER I

CATARRHAL STOMATITIS

(*Catarrhal Inflammation of the Mouth*)

Ætiology.—Inflammation of the buccal mucous membrane is not infrequently the direct result of mechanical or chemical causes. As mechanical causes we may mention particularly the sharp edges of broken or carious teeth. It should be said that often infectious organisms (*vide infra*) are active at the same time, because they gain a much easier foothold on account of the presence of bad teeth. Chemical irritation may come from highly spiced food, or from tobacco-chewing or excessive smoking. Chemical and mechanical irritation provokes stomatitis in many trades which expose the artisan to lime, copper, coal, or other varieties of dust. In drunkards we find not infrequently a chronic stomatitis, which is characterized by a thickly coated, moist, and shiny tongue, which is somewhat swollen and exhibits indentations on its side due to its pressure against the teeth. Inflammation of the mucous membrane of the mouth from poisoning with corrosive acids or alkalies is also the result of direct chemical irritation. It may take the form of severe ulcerative stomatitis (see the following chapter). The mercurial stomatitis, which results from acute or chronic poisoning with mercury, is caused indirectly by the particles of mercury deposited from the blood in the mucous membrane. This mercurial stomatitis when severe always takes the form of ulceration or necrosis. The stomatitis attendant upon the cutting of teeth in children will be discussed below.

In many instances stomatitis comes from a direct propagation of inflammation from neighboring parts. It thus forms a frequent complication of pharyngeal catarrh, and less often of rhinitis.

Infection plays an important part in the ætiology of stomatitis. The local inflammation may be merely part of a constitutional infectious disease, as in measles, variola, and syphilis. Stomatitis is still more frequently a complication of some severe and protracted illness, when the mouth is not properly attended to and cleansed. The bits of food and the mucus quickly begin to decay. Great numbers of fungi and bacteria invade the buccal cavity, and excite inflammation in its mucous membrane. Even the cases of primary stomatitis which sometimes occur are probably all of an infectious character. They usually soon assume the ulcerative form. Scorbutic stomatitis will be considered under scurvy.

Clinical History.—The usual symptoms of an inflammation of mucous membrane—namely, redness, swelling, and increased secretion—are exhibited in stomatitis. The redness is usually most intense on the inside of the cheeks and on the gums. Indeed, we have the special name—gingivitis—for inflammation of the latter. The swelling is best shown by the indentations made by the teeth in the cheeks and the edges of the tongue. The tongue and gums are smeared with mucus. There is often considerable salivation. If the inflammation is more active, we find a muco-purulent coating on a greater or less portion of the membrane. The tongue is almost always thickly coated. If we scrape off a little of the coating and put it under the microscope, we find a great abundance of pavement epithelium, in part fatty-degenerated, pus, micrococci, and remains of food. White spots made up of epithelium may also be seen on various parts of the oral mucous membrane. Here and there little vesicles appear which burst and leave superficial ulcers.

The local discomfort of severe stomatitis is by no means trifling. There is burning pain, which interferes with taking food, and usually the processes of decomposition occasion a constant bitter or disgusting taste in the mouth, as well as a foul and offensive breath.

The duration of the disease depends on the nature of the immediate cause or the character of the primary disorder. Usually a stomatitis which gets well in one or two weeks is called acute, and a more tedious attack, chronic. The chronic form is seen in topers, inveterate smokers, and persons with bad teeth. It may last for years, with the symptoms described above, only milder. (For lingual psoriasis, *vide infra.*)

Treatment.—If the inflammation is considerable, the diet must be liquid. Sometimes cold drinks are most agreeable, but usually lukewarm are preferred. Often the pain is relieved by taking from time to time a sip of iced water or a bit of ice; but in most cases the patient will prefer to rinse the mouth with lukewarm water. The important indication, to keep the mouth as clean and pure as possible, is best met by having the mouth frequently rinsed out with a one- or two-per-cent. solution of carbolic acid, a two-per-cent. solution of chlorate of potash, or one or two teaspoonfuls of a one-per-cent. solution of permanganate of potash in a glass of water. It is also advisable to rinse the mouth with a two-per-cent. solution of peroxide of hydrogen, and with thymol, which latter is the chief constituent of Miller's mouth wash, the ingredients of which are: Thymol, ¼ part; benzoic acid, 30 parts; tincture of eucalyptus, 4 parts; distilled water, 750 parts. In children who can not do this, the mouth is to be carefully washed or sprayed. If the gums are spongy, they should be painted with a mixture containing equal parts of tincture of myrrh and tincture of rhatany. If there are superficial ulcers scattered about, it is sometimes an excellent plan to touch them lightly with lunar caustic, to hasten their healing.

Chronic stomatitis is often very obstinate, resisting all sorts of treatment for a long time. The first thing is to remove any such injurious agencies as tobacco or bad teeth. It is recommended, besides the above-mentioned remedies, to swab out the mouth with a solution of corrosive sublimate (1 to 5,000) or of lunar caustic (1 to 30–50).

CHAPTER II

ULCERATIVE STOMATITIS

(Stomacace)

Ætiology.—By ulcerative stomatitis is meant a severe disease of the buccal mucous membrane, with superficial necrosis and the consequent formation of ulcers. The abnormal processes are not in all cases identical, and their cause may vary. Still it is probable that infection is the important factor, at least in the primary cases. The disease has repeatedly been epidemic, chiefly among soldiers in barracks or on a campaign, and among the inmates of jails. We have repeatedly seen sporadic cases of primary ulcerative stomatitis with fever. In children the disease is especially common at the time of the second dentition. Here, too, endemic and contagious influences are frequently in evidence.

Mercurial stomatitis is, from a practical point of view, the most important of those forms of ulcerative stomatitis which are referable to definite chemical action. It is due to the inhalation of the fumes of mercury in those whose trade or scientific occupation exposes them to this danger, and very frequently also to the therapeutic employment of mercury in the form of calomel or mercurial ointments, and the like. (For the scorbutic form, see page 741.)

Symptoms.—The disease usually attacks the gums of the lower jaw first, gradually spreading thence to neighboring portions of the lips and cheeks. The tongue and palate are generally not very much affected, though often the seat of a simple catarrhal inflammation. In severe mercurial stomatitis the gums are especially apt to be affected. The ulcers also show a preference for the angles of the lower jaw, and those portions of the mucous membrane of the cheeks which rest upon the teeth.

Inspection shows that the mucous membrane in the places mentioned has a pasty, purulent coating. The gums are swollen, spongy, and red, and bleed easily. The mucous membrane on the edges of the gums becomes necrotic, giving rise to ulcers. The incisor teeth are so loosened by gingivitis that they may even fall out. There is usually profuse salivation. The lymph-glands at the angle of the lower jaw and on the chin are generally swollen. The breath is very offensive, poisoning the air of the whole room.

The local discomfort of the patient is the same as in simple stomatitis, only much worse. It is very difficult to take nourishment. In many cases there are marked constitutional symptoms. The patient feels very weak and languid. The primary, and sometimes also the toxic, cases of ulcerative stomatitis are associated with moderately high fever—100.5°–102° (38°–39° C.). Now and then severe symptoms of constitutional sepsis have followed the disease.

The course of ulcerative stomatitis is favorable in the great majority of cases. With good treatment and nursing, the ulcers gradually clean up, and at the end of one or two weeks recovery is complete. Exceptionally, the disease may be more chronic. The most frequent way in which recovery is delayed is that the disease extends to the periosteum of the lower jaw, causing necrosis of small portions of the bone, which must be expelled before the patient gets well.

The **treatment** does not differ essentially from that of the milder forms of stomatitis. The mouth must be still more carefully and more frequently cleansed and disinfected. A solution of potassic chlorate (1 to 30) is the favorite mouthwash. Good results are also obtained from permanganate of potash, boric acid, peroxide of hydrogen, and similar remedies. Some authors strongly recommend the simultaneous internal administration of potassic chlorate; but we must employ it cautiously in children, as it has repeatedly caused poisoning. For children two or three years old we ought not to give over fifteen to thirty grains (grammes 1–2) in a day.

As to prophylaxis, we should mention that all patients who are using mercury should employ a gargle of potassic chlorate faithfully from the beginning of treatment, in order to prevent the occurrence of mercurial stomatitis. If salivation occurs, the mercury must be stopped.

CHAPTER III

APHTHOUS STOMATITIS

(*Aphthæ. Disseminated Fibrinous Stomatitis*)

Aphthæ is a name given by physicians to several entirely distinct diseases. Many doctors call every disease aphthæ in which there are white spots upon the buccal mucous membrane. It is thus frequently confounded with thrush. The same name (*Schwämmchen*, or fungus) is often applied indifferently to thrush and to aphthæ.

There is a special form, known as Bednar's aphthæ. In new-born children white patches are not infrequently found lying symmetrically on both halves of the hard palate near the alveolar processes, and persisting till about the third month. These plaques are not syphilitic, although often thought to be. They are probably due merely to the tongue pressing upon the thin mucous membrane during nursing. Generally they do no harm; but in marantic, neglected children, they may develop into quite deep ulcers. In that case, repeated cauterization with a five-per-cent. solution of argentic nitrate is required.

The genuine aphthæ are roundish spots upon the mucous membrane, grayish white, and of small size, unless made larger by the confluence of several into one another. They usually have a narrow, red areola. They are most numerous on the edges and dorsum of the tongue and on the frænum, but they also occur on the lips and cheeks. The attempt to remove the white spot with forceps never succeeds, but it causes bleeding. In addition to the genuine aphthæ there are almost always the signs of a common stomatitis, which may be mild or severe. The white spots are due in part to a thickening and opacity of the epithelium, and in part are said to be caused by the formation of a fibrinous exudation, which penetrates the most superficial layers of the mucous membrane. If the necrotic epithelium is cast off, the aphthous spot develops into a little ulcer, which often heals promptly, but which may be very obstinate.

The disease occurs chiefly in children, and at the time of the first dentition. The child is usually restless, often somewhat feverish, and evidently suffers pain when nursing. Generally there is considerable salivation. The lymph-glands may be a little enlarged. On the outer skin in the neighborhood of the mouth there may also appear a few blisters and pustules. These, as a rule, have no connection with herpes, although now and then genuine herpes may appear in association with aphthous stomatitis. The disease is not rare in adults. Many individuals seem especially liable to it, and very frequently have little, white, and often very painful spots here and there on the tongue or elsewhere in the mouth. These have a tendency to develop into ulcers which are usually superficial, but sometimes deep. They may prove very troublesome, from their frequent recurrence and the hindrance they cause to speaking and mastication. If there are fairly deep ulcerations on the tongue and mucous membrane, the patient's condition for a time is very painful and distressing.

With the exception of the form just described, which is constantly relapsing, aphthæ almost always run a favorable course. There is usually complete recovery in a week or two. The treatment of children consists in carefully washing out the mouth with cold water, and in administering potassic chlorate. Of a mixture consisting of three parts of potassic chlorate, twenty of syrup, and a hundred of water, we may give a dessertspoonful every two hours. If the spots do not disappear, we can paint them with a solution of permanganate of potash (1 to 150), a five-per-cent. solution of sulphate of zinc, or a solution of borax (1 to 30). If some of the places are especially painful, particularly in adults, we may touch them with lunar caustic, when they usually are soon cured. In the constantly recurring form, treatment sometimes proves almost unavailing. Local cauterization, which unfortunately in such cases is often of no benefit whatever, and the use of a mouth-wash containing chlorate of potash or carbolic acid in weak solution, may be employed, and, furthermore, some physicians strongly recommend the internal administration of iodide of potassium. Calomel also appears sometimes to have a beneficial influence.

It is often a very difficult matter to treat chronic, recurrent, aphthous stomatitis, and the ulcerations which are occasioned by it in adults. Cauterization with lunar caustic, chromic acid, and similar agents usually does more harm than good. None of the ordinary mouth-washes has any great effect. A much praised remedy is the chewing of bilberries, either fresh or boiled, for several minutes several times a day, and we have ourselves employed it in some cases with good results. Some physicians also recommend the internal use of iodide of potassium, or small doses of calomel. It is remarkable that habitual aphthous stomatitis sometimes ceases completely for months—for example, upon change of residence or during a visit to the country—but then appears afresh without special cause.

In conclusion, as to ætiology, infection is a not unlikely cause, if we consider that small epidemics or endemics have repeatedly occurred. Lately attention has been called to the possibility that the milk of cows suffering from hoof-and-mouth disease may be a source of infection. That infection of this sort may occur seems to us indubitable, from the experience of others, as well as our own. In such cases there is not only a severe aphthous stomatitis, but a development of vesicles, pustules, or even extensive purulent inflammation affecting the fingers and nails, and occasionally other parts of the body. Most cases of this kind also pursue a favorable course, but a few epidemics have been described in which the disease became dangerous to life because of constitutional sepsis. Probably the various forms of stomatitis aphthosa are connected with various infectious germs.

CHAPTER IV

THRUSH

(*Soor. Muguet*)

Ætiology.—Weak and artificially nourished children are particularly liable to this disease; but it also attacks adults who are suffering from phthisis, carcinoma, and severe typhoid or typhus fever. In it, grayish-white deposits are developed upon the buccal and pharyngeal mucous membrane. The microscope shows these collections to be fungi; there are a multitude of oval spores, or conidia, and a tangled mass of long mycelium threads. Until lately the fungus of thrush was called *oïdium albicans*, and was held to be identical with the *oïdium lactis* found in sour milk. This view is now abandoned, although no other has as yet been established with regard to the botanical position of the thrush fungus. Grawitz maintained that the thrush fungus is a sprouting fungus, and nearly related to the mould fungi (mycoderma vini,* or saccharomyces albicans—Rees). It seems to be a sort of transitional form between the sprouting fungi and the thread fungi, inasmuch as it appears both in the yeast form and also in the form of threadlike mycelia. Plaut, according to his investigations, makes the thrush fungus identical with the monilia candida, which belong to the Torulaceæ. At any rate, the thrush fungus is widely distributed, for the development of thrush upon the mucous membrane of the mouth and throat is a frequent phenomenon, especially in ill-nourished children. Nursing-bottles and the nipples used upon them are probably not infrequently the agents by which the disease is conveyed.

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* Mycoderma vini is that fungus which is found in the development of vinegar from alcohol, when alcoholic beverages sour.

Symptoms.—The mucous membrane of the tongue, cheeks, and soft palate is usually somewhat red and swollen. Upon it we see at first small white spots, which may gradually spread. Microscopic investigations have shown that the fungus develops first in the middle layers of the epithelium. From this starting-point it grows not only upward, but also downward into the mucous membrane. If the growth is abundant, it is easy to scrape off the upper layers, and make a diagnosis by aid of the microscope. In exaggerated cases the growth may even extend from the pharynx into the upper part of the œsophagus and the entrance of the larynx; but we never find thrush in the larynx itself, or the nostrils, or the stomach—briefly, in no place where there is cylindrical epithelium. Yet in exceptional cases the thrush fungus may extend its growth through the wall of a blood-vessel, and so reach the circulation. Usually thrombosis of the affected vessel takes place, but the metastasis of thrush to the brain and the kidneys has been observed. If abscesses are associated with these processes they depend upon the simultaneous intrusion of pyogenic cocci.

As a rule, thrush is accompanied by a more or less severe stomatitis. The fluids of the mouth have an acid reaction. Nursing, or chewing, and swallowing, are painful. Still, it is a question whether the stomatitis is due to the fungus, or whether it prepares the territory for the fungus to settle in. Nursing infants, who suffer from thrush, often have diarrhœa or marasmus at the same time, which latter affections are more probably the cause than the result of the thrush. If vigorous and healthy sucklings are attacked by thrush, the disease is usually quite harmless, quickly vanishing if proper cleanliness is maintained. In sickly children, particularly if bottle-fed, the appearance of the disease is very ominous. If there is a large amount of thrush in the œsophagus, the ingestion of food may be seriously interfered with. In adults, as we have said, thrush is with few exceptions confined to persons greatly prostrated by such diseases as severe typhoid fever or tuberculosis, and from this point of view it is an unfavorable symptom.

Treatment.—To prevent the development of thrush in children, the mouth must, if possible, be wiped out, each time they drink, with a cloth wet in cold water; and if adults are very ill, they require equal attention in this regard. As soon as we see the first traces of the disease, we should touch the parts attacked with a brush wet in an aqueous solution of borax (1 to 20). Honey should not be added to the borax solution, as is often unwisely done. Another good solution is permanganate of potash (1 to 150). If the thrush extends into the œsophagus the most efficient remedy (Baginsky) is said to be the internal administration of resorcin, in a one-half- to one-per-cent. solution, the dose being a teaspoonful every two hours. The food should contain as little sugar as possible, as this furnishes a favorable culture medium for the thrush fungus. Mild cases can usually be cured by the remedies named. If the thrush has once got a vigorous start in the mouth of marantic children, or of adults suffering from an incurable disease, it must be confessed that we often fail to check its growth.

CHAPTER V

GLOSSITIS

(Parenchymatous Inflammation of the Tongue)

Inflammation of the true lingual parenchyma is rare, although the tongue's mucous surface is frequently involved in the various diseases of the mouth.

1. **Acute parenchymatous glossitis** is the name given to an inflammatory infiltration of the whole or a part of the tongue, usually ending in abscess. The most frequent cause is the sting of a bee or wasp, or it may follow burns or severe cauterization. In the rare instances when it is apparently spontaneous, it is probable that some little wound has afforded ingress to the inflammatory poison.

The symptoms of acute glossitis are very violent in the severer cases. The tongue is enormously swollen, so as sometimes to protrude from the mouth. It has a thick, soft, purulent coating, and often presents excoriations and ulcerations. The subjective symptoms are very disagreeable. The patient has violent pain. Talking and eating are almost impossible. There is usually catarrhal inflammation of the rest of the mouth. The cervical lymphatic glands are swollen. The salivation is profuse and very annoying. In many cases the tongue swells so much as to cause dyspnœa and more or less suffocation. There is usually fever.

Treatment consists in the employment of ice, which the patient should keep constantly in his mouth, if possible. Very great relief follows deep scarification done in a few places where the swelling is greatest. As soon as fluctuation is obtained, we must give exit to the pus. This is usually followed by a rapid abatement of the discomfort, and complete recovery. It is the exception that the increasing dyspnœa necessitates tracheotomy.

2. **Glossitis Dissecans.**—This is a chronic disease, of comparatively rare occurrence and unknown ætiology. It causes the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures.

The trouble is not intrinsically dangerous, nor does it need special treatment. We must try the same remedies which have been mentioned above in connection with the various forms of stomatitis, such as antiseptic mouth-washes, the chewing of bilberries, etc. If ulcers are present they sometimes require cauterization with lunar caustic.

3. **Lingual Psoriasis. Leucoplacia.** *(Tylosis; Ichthyosis linguæ et oris.)*—This, again, is a superficial disease, the ætiology of which is unknown. It consists in localized circular hyperplasiæ of the epithelium of the tongue, rarely conjoined with similar spots upon the cheeks and lips. The central portion of mucous membrane which is surrounded by this whitish, slightly raised, and more or less circular, wall of epithelium, is abnormally smooth and of a reddish color. Usually the tongue takes on a resemblance to a map (*lingua geographica*). The disease generally persists for years, and without symptoms except in severe cases. Often it is found by chance in individuals who have known nothing about the unusual appearance of their tongues. Still, it may cause a hypochondriac endless anxiety, especially if he takes it to be syphilitic.

This last statement applies still better to a peculiar disease allied to psoriasis. It is called leucoplacia, and affects the mucous membrane of the tongue and mouth. Usually it causes the appearance, on the lateral borders of the tongue, of dull-whitish spots, which have the look of scars, and are generally somewhat notched. As a rule, the lower surface of the tongue and the cheeks display at the same time similar white spots, which are evidently due merely to thickening of the epithelium. Certain spots may disappear, but they are sure to be replaced by others, so that, so far as has yet been observed, the disease must be regarded as incurable. Still, it is not of great importance, as a rule, for in many cases the local discomfort is very slight. If the indentations along the sides of the tongue become cracked or ulcerated, then there may be great pain. Leucoplacia is very often associated with bodily states of neurasthenia, or hypochondriasis, particularly if the leucoplacia appears in a person with a syphilitic taint, which happens with remarkable frequency. But it is certainly not to be regarded as a tertiary symptom, rather perhaps as a sort of sequel to syphilis, although many cases have no connection whatever with the latter disease. Anti-syphilitic treatment is invariably useless. It is for this very reason important that the physician should be well acquainted with leucoplacia, that he may spare his patient needless anxiety and ineffectual, or perhaps actually harmful, treatment with mercury. The disease has no relation to excessive smoking, one proof of which is that we have seen the condition in women. The only danger of leucoplacia is that sometimes lingual carcinoma may eventually develop on the spot affected with it. We have in this case to deal with a process which is analogous to the development of ventricular carcinoma in the scars of old ulcers, or of cancer of the gall-bladder subsequently to gall-stones. Treatment is, as we have said, usually unsuccessful. Still, thorough cleanliness and good care of the mouth may avert any great discomfort. We may try the effect of painting the spots with a four-per-cent. solution of borax, or a five-per-cent. solution of chromic acid. Lately the frequent chewing of boiled bilberries has been recommended for leucoplacia, and we have had good results from this procedure.

CHAPTER VI

NOMA

(*Water-cancer. Cancrum oris*)

Noma is a gangrene of the cheek, apparently of spontaneous origin, and attacking chiefly feeble and sickly children. The disease is rare. It may be primary, but it is usually a sequel of severe diseases, such as measles, scarlet fever, typhus and typhoid fevers, and pneumonia. Now and then it has been observed in adults. *A priori*, it is extremely probable that noma is due to some parasitic micro-organism; but the matter has not yet been minutely investigated. It deserves mention that noma is said to occur with much greater relative frequency in moist regions along the coast—for example, in Holland—than in Germany.

The disease begins, without any evident occasion, in an insignificant spot of gangrene on the inner surface of the cheek—that is, in the mucous membrane. It is usually situated near the corner of the mouth. Externally, the parts are soon swollen by collateral œdema, and the whole cheek gradually becomes hard and infiltrated. At first, all we see upon the mucous membrane is a dirty-greenish spot not much larger than a silver dime, but soon the whole cheek and the neighboring parts are one mass of gangrene. Bits of dead tissue come away, and foul-smelling ichor flows continuously into the mouth. The collateral œdema may finally pervade that entire half of the face. The neighboring lymph-glands are always greatly swollen.

This condition is as a rule accompanied by fever, often reaching or exceeding 104° (40° C.). The general health may indeed for a time be astonishingly little affected; but gradually prostration comes on, or even general sepsis develops, with fever, stupor, and delirium. Frequently lobular pneumonia, which may have a gangrenous character, is produced by the inhalation of sloughing bits of tissue; and often the ichor, being swallowed, excites violent and offensive diarrhœa. The local discomfort is not really very considerable in most cases, compared to the severity of the disease. There may even be no pain felt whatever.

The prognosis is almost always fatal. Death sometimes occurs suddenly from collapse. Sometimes it comes at the end of three or four weeks, from a gradual sinking of the bodily powers. Recovery has been seen in only a few cases: there is a line of demarkation formed, the sloughs come away, and a slow convalescence follows, leaving extensive and usually very disfiguring scars behind.

Treatment must have for its chief object to check further extension of the gangrene, by removing all parts that are already destroyed. Local cauterization with concentrated hydrochloric acid, or fuming nitric acid, or lunar caustic, or chloride of iron, is usually futile. It is probably the best way to remove all the gangrenous portion by means of Paquelin's thermo-cautery. At least in the early stages of noma we may hope something from this method of treatment; but if the case is far advanced, we can hardly expect to accomplish much.

We should also disinfect the mouth as thoroughly as possible. The most efficient means is to syringe it out with solutions of salicylic or carbolic acids, or permanganate of potash. We should do our best to maintain the patient's strength.

CHAPTER VII

PAROTITIS

(Mumps)

Parotitis, or inflammation of the parotid gland, appears not only as a peculiar, primary, infectious disease, usually epidemic, but also as a secondary complication of numerous other severe diseases. These two forms should be considered separately.

1. **Idiopathic, Primary Parotitis** (*Epidemic Mumps*).—**Ætiology.**—The disease occurs in epidemics that, although not very frequent, may be quite extensive. Endemics also occur, in barracks and schools; and here and there a sporadic case is seen. Children and young adults are most liable to it. Nursing infants enjoy a marked immunity, as well as elderly persons. Males are much oftener attacked than females.

There can be no doubt that mumps is a specific infectious disease, and claims have already been made of the discovery of its specific bacillus, but these lack confirmation. Still, it is natural to suppose that the infectious matter reaches the gland by way of Steno's duct. Numerous observations support the view that the disease is directly contagious; but the degree of contagiousness is not great. Perhaps the saliva of patients has something to do with the extension of epidemic mumps. The period of incubation seems to vary. On the average, it is about fourteen days.

Clinical History.—There may be a prodromal stage of one or two days, with mild feverish symptoms. The disease itself begins with swelling of one parotid gland. The swelling is directly below and in front of the lobe of the ear, which is gradually pushed upward. In the next few days the swelling rapidly increases, and it and the collateral œdema of the cheek and floor of the mouth may become very considerable. The face is much distorted, but often makes a very comical impression, especially as everybody knows how harmless the disease is. In most cases the other gland also swells a few days later.

Suppuration scarcely ever occurs in genuine mumps. If it is seen as a rare exception, it is probably due to secondary infection. The swelling often becomes very hard. Generally it has a somewhat doughy consistency. The corresponding portion of skin is usually pale and shiny. The submaxillary gland not infrequently swells also in addition to the parotid, and this may occur upon one or both sides of the neck. Penzoldt has observed cases in which the submaxillary and sublingual were swollen, but not the parotid. We ourselves have seen a swelling of the submaxillary precede the parotitis. The sublingual gland also seems sometimes to be involved with the parotid.

The local discomfort is moderate in most cases. There are local pain and difficulty in chewing, but less in swallowing and talking. Often quite a severe stomatitis develops, with foul breath.

There sometimes seems to be no fever whatever. Usually there is a moderate elevation of temperature. Often, however, it reaches 102.5°–104° (39°–40° C.). Only occasionally has there been a case with grave typhoidal symptoms.

Complications.—It is not rare for men to have a swollen testicle with inflammatory serous exudation into the tunica vaginalis, which may be quite painful, but which usually subsides in a few days. The orchitis is usually ushered in with a fresh rise of fever. Resultant suppuration is a rare occurrence. Once we saw the orchitis appear several days before the swelling of the parotid gland. Double orchitis is rare. In boys this complication is much more exceptional than in adults. Some observers have mentioned analogous swellings of the female genitals (oöphoritis) and mammæ, but this is very rare. Sometimes mumps is associated with herpes labialis.

The prognosis of epidemic parotitis is, as we have said, almost always favorable. The trouble seldom lasts more than a week or ten days, when the swelling goes down, and the patient completely recovers. Possible but exceptional sequelæ are: ptyalism; cessation of the salivary secretion; chronic swelling of the parotid; deafness; and atrophy of the testicle.

The diagnosis is easy. The only thing to exclude is swelling of the lymph-glands, and they never have exactly the same location as the parotid.

Special treatment is hardly necessary. Children should be kept in bed. Usually some salve, or vaseline, is applied to lessen the feeling of tension. If resolution is tedious, we may paint the swelling with iodoform collodion (1–15) or with tincture of iodine; or we may prescribe iodoform ointment (1–15). If there is orchitis, the testicle must be elevated, as by a suspensory bandage. If the pain and swelling are marked, an ice-bag should be applied.

2. **Secondary Suppurative Parotitis.**—This secondary form may be a complication of any grave disease. In most cases it is due to inflammatory agents, probably staphylococci in most cases, generated by decomposition of the contents of the mouth, which agents reach the gland through Steno's duct. It was formerly the universal belief that the infection was metastatic, being conveyed through the blood-vessels; but it is not certain whether this occurs. It is probable that the pyæmic form is in many instances thus produced. Secondary parotitis is most frequently observed in typhus and typhoid fevers. It is also seen occasionally in all other severe acute diseases, and in phthisis and carcinoma.

The parotid gland swells, just as in the primary disease. It is, however, much oftener of excessive size, and in the majority of cases suppurates. If one has an opportunity to make an autopsy on such a case of secondary parotitis in its early stages, the cross-section of the swollen gland presents a large number of rather small discrete abscesses. These finally unite to form one larger abscess, which usually discharges outward through the skin or into the external auditory meatus.

Sometimes the parotid suffers from gangrenous inflammation, and there is extensive sloughing. If such a case finally gets well, still, as a rule, some permanent injuries have been inflicted: there is facial paralysis, due to destruction of the facial nerve, or deafness, caused by an extension of the inflammation to the middle ear.

The treatment of secondary parotitis is that of any suppurative inflammation. We may at first try to scatter the swelling by ice or iodoform ointment, but this usually fails. As soon as fluctuation is detected, the spot must be incised, and a drainage-tube inserted. The prognosis depends chiefly on the nature and course of the original disease.

CHAPTER VIII

ANGINA LUDOVICI

The name *angina Ludovici* is applied to a rather rare phlegmonous inflammation of the floor of the mouth. Probably this is not occasioned by a specific cause, but is only a special localization of the ordinary germs which occasion suppuration. Its starting place seems to be the submaxillary gland, at least in most cases. It may be primary, or a complication of other severe acute diseases.

Angina Ludovici usually begins with swelling in the neighborhood of the submaxillary gland. The swelling rapidly increases, and comes to involve the whole floor of the mouth and the anterior surface of the throat. It causes great discomfort. Talking, chewing, and swallowing are almost impossible. There is usually fever, and in many cases we even find the symptoms of general sepsis. There may be great dyspnœa, due either to compression of the larynx or to œdema of the glottis. The final result in some cases is an extensive sloughing of the soft parts. This has the special name of *cynanche gangrænosa*. In other cases an abscess forms, and points outward or into the oral cavity. The swelling is sometimes, though seldom, reabsorbed.

The prognosis should always be guarded, for severe constitutional symptoms and a fatal ending are not infrequently seen, particularly if the patient has a weakly constitution. There may also be repeated exacerbations and relapses.

Treatment.—At the commencement of the disease we may make the attempt, in suitable cases, to check the process by local depletion and by ice; but, as soon as suppuration or gangrene begins, the case becomes a surgical one. Now and then the threatening asphyxia demands tracheotomy.

CHAPTER IX

ANOMALIES OF DENTITION

(*Difficult Dentition*)

The processes of dentition play so important a *rôle* in the disorders of childhood that we feel obliged to discuss the subject, at least briefly.

The first appearance of any of the milk-teeth usually takes place when the child is seven to nine months old; it may, however, occur either earlier or later than this period. As a rule, the two lower central incisors are cut first; then the upper central incisors appear, a few weeks later, and next the lateral incisors of the upper jaw. In the beginning of the second year come the lower lateral incisors, and almost simultaneously the four anterior molars. The four canine, or "eye" and "stomach" teeth, are cut in the second half of the second year; and last of all comes the eruption of the four posterior molars. The first dentition is therefore completed by the end of the second or in the beginning of the third year, with the development of all the twenty milk-teeth. The accompanying diagram (Fig. 44), after Vogel, represents the order in which the separate teeth appear. In the fifth or sixth year the milk-teeth begin to be replaced by the permanent teeth of the second dentition. "Trouble with teething," however, almost invariably refers to anomalies of the first dentition.

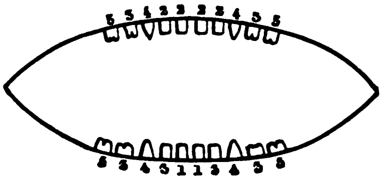

FIG. 44.

Noticeable delay in teething is frequent in weakly, and particularly in rachitic children, in whom the teeth may develop not in pairs and in symmetrical groups, as in healthy children, but in irregular succession. In such cases, sometimes, all the teeth are not cut till the end of the third year.

On the other hand, it sometimes happens that certain teeth appear very early, or even are present at birth. If an abnormally early tooth is only loosely inserted in the gums, it should be removed with the forceps; for it interferes with nursing, and injures the opposing surface of the mouth; but if the tooth is firm in its place, we let it be.

During the eruption of the teeth there is in every child considerable redness of the mucous membrane and an increased flow of saliva. The child evidently feels an itching in the mouth, and therefore a constant desire to bite something. This simple catarrh is sometimes accompanied by a slight rise in temperature. Occasionally there is a severe stomatitis, with which fever and thrush may be associated. These troubles should be treated as already described.

In consequence of the salivation, and the large amount of saliva which is swallowed, in which the various processes of decomposition are apt to develop, we often see gastro-intestinal diseases in teething children. In most children a temporary and mild diarrhœa occurs. We should be particularly careful at this period about the child's nourishment, and in treating any marked gastro-intestinal symptoms. Experience shows also that teething children are unusually liable to simple or even capillary bronchitis, and catarrhal pneumonia.

Nervous disturbances are often referred to dentition. The most important symptom of this kind is eclampsia. The attacks are sometimes called "teething-convulsions." Although the laity go too far in ascribing all sorts of nervous disorders to teething, still experienced specialists do recognize the possibility of such an origin for many cases. Some of the convulsions may in fact be regarded as reflex (*vide infra* the chapter on the convulsions of children, page 1151).

When the upper canines, or "eye-teeth," are being cut, there is sometimes a unilateral purulent conjunctivitis, which is perhaps to be explained as an extension of the inflammation by way of the antrum of Highmore and the nostrils. Eczema and other cutaneous eruptions have been often ascribed to dentition, whether justly or not is doubtful. Yet it is remarkable that little children sometimes have an eruption of facial eczema with the appearance of every tooth.

There is, of course, no special treatment for difficult dentition; and the various disturbances which it may indirectly produce are to be treated on general principles.

SECTION II

DISEASES OF THE SOFT PALATE, TONSILS, PHARYNX, AND NASO-PHARYNX

CHAPTER I

SORE THROAT

(Inflammation of the Soft Palate and of the Tonsils)

Ætiology.—Acute inflammation of the soft palate and tonsils, in its various forms, is one of the commonest of diseases. Almost everybody has had personal experience with it. It is chiefly a disease of early life, being infrequent after the thirty-fifth year. Individual predisposition to it varies greatly. There are persons who have one or more attacks almost every year, while with others attacks are rare and insignificant. In many instances exciting causes have evidently been potent. Chief among these is catching cold; the sufferer has had wet feet, or has been talking in a damp, cold atmosphere. Most cases, therefore, occur in cool weather, although now and then attacks may occur in the hottest days of summer. Again, direct injuries of the pharynx may produce the disease, e. g., the smoky atmosphere of inns, combined with loud talking or shouting; the inspiration of poisonous vapors; cauterization of the mucous membrane with concentrated acids, alkalies, and other chemical agents; and burns.

Apart, however, from these last-named external irritants we have many cases of primary angina, for which we must assume an infectious origin. This supposition is suggested by the severity of the constitutional disturbance, with fever, the nature of the local process (giving rise to suppuration), and the occasional appearance of the disease in an epidemic or endemic form. At the same time, exposure to cold and the other exciting causes above mentioned, may often favor its development. [Especially when tonsillitis recurs in an individual, or attacks several members of the same household about the same time, it is well to have the drainage carefully examined. The precise connection between bad drainage and sore throats we do not know, but that they are often connected there seems to be no doubt.] With regard to the nature of the infectious organism, there is as yet great uncertainty. When the tonsillitis is of the suppurative parenchymatous variety, it is almost certain that we have to deal not with specific germs, but with staphylococci and other ordinary pus-organisms. The same is probably true of follicular tonsillitis also, although several different kinds of pathogenic organisms may be concerned; thus we may find not only staphylococci, streptococci, and particularly diplococci, but probably, in many cases, even the genuine diphtheria bacillus.

Frequently the inflammation is due to extension from neighboring parts, as in coryza, laryngitis, and stomatitis. In many cases both affections are simultaneous results of one common cause. Finally, sore throat may be a symptom of many acute infectious diseases, such as scarlet fever, measles, small-pox, and erysipelas.

To distinguish between an inflammation of the soft palate by itself and an affection of the tonsils is not practicable. In most cases the tonsils are the stronghold of the disease; less often we find the inflammation limited to the soft palate.

Clinical History.—The most important subjective symptom of sore throat, and that by which it is usually first recognized, is the difficult and painful deglutition. The pain is sometimes manifest before any objective changes are to be seen. It may in a severe case be very violent and distressing. The pain has a "darting" character, or sometimes is "burning"; and it is most acute whenever the patient swallows, although in well-marked cases it seldom entirely intermits. Swallowing is not only painful, but it is laborious; it requires more than usual effort and time. The patient feels constantly as if he had to swallow a big lump. This sensation is worse if the tonsils are swollen. It is a matter of experience that not infrequently an "empty" swallowing hurts more than swallowing a liquid or some half-solid substance.

Talking is also difficult. Every word may be painful, so that the patient expresses his wishes as briefly as possible. Even in a mild case, speaking for any length of time will produce a burning pain in the throat. The impaired mobility of the soft palate often prevents the complete cutting off of the nasal passages in talking, so that the voice has a nasal twang; and often it sounds as if the patient were talking with his mouth full: he has the "voice of sore throat."

Further local discomfort results from the mucus and saliva collecting in the mouth. Salivation is not infrequent, probably as a result of the stomatitis usually present. In other cases the patient complains that his mouth feels dry and sticky. Frequently there is a persistent bad taste in the mouth, and the breath is disagreeable.

With these local disturbances, more or less severe constitutional symptoms are almost always conjoined. Indeed, these latter may begin a day or two earlier than the local symptoms. The patient is indisposed, languid, has anorexia and headache. The general disturbance may be surprisingly great in comparison with the slight objective changes in the tonsils.

There is fever in most of the well-marked cases; it may even be quite high. Temperatures of 103° or 104° (39.5°–40° C.), or even higher, are not rare. Sore throat can not be said to have one particular type of fever. Usually the fever appears rather abruptly, remains high for several days, with an occasional slight interruption, and then falls with equal abruptness to normal again.

The entire attack usually lasts only a few days, seldom more than a week. Even when a person is quite ill for several days, convalescence is almost always rapid and complete—that is, if the patient has a good constitution.

Special complications are very infrequent, except that the neighboring parts—the larynx, mouth, and throat—are not seldom involved. Herpes labialis is quite frequent. Beyond this there is nothing to mention.

Various Forms of Sore Throat

The symptoms thus far mentioned are much the same in all cases of sore throat, varying only in intensity and duration. But the objective changes to be observed in the soft palate and tonsils are noticeably different in different cases. Whether the ætiology differs also we have no certain information. In some instances it seems probable that it does.

We shall distinguish five chief varieties of acute sore throat. Transitional forms are, however, by no means rare. Genuine diphtheria, which is a specific, acute, infectious disease, and which has already been discussed, does not need to be brought up again here.

1. **Catarrhal Sore Throat** (*Simple Catarrhal Inflammation of the Mucous Membrane of the Soft Palate*).—There is a more or less vivid reddening of the mucous membrane, either uniform or in patches. The swelling is most marked in the pillars of the fauces and the uvula. The surface of the tonsils is likewise reddened; their size may be somewhat increased, or remain unchanged. The mucous membrane of the palate and uvula may be covered here and there with a thin layer of muco-pus, which can easily be wiped off. The tonsils may present small, superficial erosions, scattered about. These little ulcers are apt to lie at

the openings of the follicles. The small "blisters" which are often seen on the mucous membrane of the soft palate may be caused in various ways. Either they are mucous glands or solitary follicles, swollen; or, rarely, they are real vesicles filled with a clear fluid and produced by a raising up of the epithelium. The cervical lymph-glands are usually but slightly swollen, if at all.

This is the common and mildest form of sore throat; and it may be over in a day or two. In some instances, however, it causes considerable local and general discomfort; but the disease seldom lasts longer than five to eight days.

2. **Follicular Tonsillitis.**—In this form there is not only more or less catarrhal inflammation of the soft palate, but a decided swelling of one or both tonsils. On the reddened surface of these swollen bodies are whitish-yellow spots, varying in number from two or three to ten or more, and corresponding to the follicles. These spots are often seen to be plugs projecting from the openings of the follicles. It is usually easy to press out the pasty contents of the follicle, represented by the white speck, with a spatula. The microscope shows it to consist of numerous epithelial cells and pus-corpuscles, bacteria, and detritus, and sometimes there are also crystals of the fat acids and cholesterine. The pus-corpuscles may so predominate that we may have small follicular abscesses, which, on being opened, leave superficial ulcers behind. The parenchyma of the tonsil is swollen with a serous and cellular infiltration, increasing the bulk of the part. The trouble is usually bilateral, though often more marked and extensive on one side than on the other. In the severer cases the cervical lymph-glands are swollen. The local symptoms and still more the constitutional disturbance in follicular tonsillitis may be decided. The fever not infrequently reaches 104° (40° C.) or even higher. The patient feels very languid, has no appetite, and complains of headache; but there are mild as well as severe cases. The disease never lasts longer than a few days, even when the fever is high at first. The yellow spots vanish from the tonsils, and there is rapid recovery; severe complications are hardly ever seen. Often there is a herpes labialis and there may be slight albuminuria, but this very rarely amounts to a genuine acute nephritis. It should be mentioned that sometimes the contents of a few of the follicles remain in them for a considerable length of time, and become inspissated and calcified. It is not a rare thing to find such plugs in the tonsils of those who are subject to sore throat. Timid and hypochondriacal individuals are sometimes badly frightened by expectorating these old chalky plugs, which they believe to be "tubercles"!

From an ætiological standpoint, follicular tonsillitis is in most cases entirely distinct from genuine diphtheria, and so far the search for the Klebs-Löffler bacillus in the plugs contained in the follicles has almost invariably proved negative. Still it is noteworthy that precisely at the time of diphtheria epidemics follicular tonsillitis is remarkably frequent. It might, after all, be possible that the mildest degrees of diphtheria present themselves in the garb of an apparently simple follicular tonsillitis. The latter is very probably an infectious disease; but this matter has not yet been settled by pathological investigations.

3. **Tonsillar and Peritonsillar Abscess** (*Parenchymatous Sore Throat; Quinsy*).—In this form the swelling of the tonsils is the most striking symptom. They may be more than twice their natural size. The anterior pillars of the fauces are pushed forward and become convex. The swelling extends so far toward the median line that the tonsil touches the uvula; or, if the affection is bilateral, the two tonsils press against each other, grasping the uvula between them, or pushing it forward. The soft palate is very much reddened, particularly at first. Its surface is usually thickly covered with mucus. If this be wiped off, the mucous membrane is seen to have a moist, œdematous luster. The mucous

membrane of the tonsils not infrequently suffers a superficial necrosis. Follicular and parenchymatous tonsillitis are often combined.

In well-marked cases of abscess the local discomfort is usually great. With every attempt to swallow there is pain which shoots into the ear. The patient is in a pitiable condition; he can neither talk, nor swallow, nor gargle. The few words which he painfully utters have in an extreme degree the nasal quality of the "voice of sore throat."

In the milder cases the trouble seldom lasts but a few days before the swelling goes down, and the discomfort and usually rather moderate fever gradually abate. In other cases an abscess of the tonsil develops, usually on one side only, but it would be more correct to say a peritonsillar abscess, for the pus usually collects mainly in the connective tissue between the tonsil and the arch of the palate. In such cases the mucous membrane bulges out, usually at a point in the velum of the palate, and on palpation there is a sense of fluctuation, and finally the abscess ruptures. In other cases, however, a tonsillar abscess forms, as a rule, on only one side. The mucous membrane bulges out more in one spot; fluctuation is detected; and, finally, the abscess breaks. With the discharge of the pus the pain is relieved very rapidly, or it may vanish at once. The rest of the tonsil soon regains its former size, and in a few days the patient is well. Relapses are possible, but rare.

Phlegmonous sore throat, in which the soft palate and not the tonsil is chiefly affected, is infrequent. Its usual cause is some severe external injury, such as burns, and cauterization with concentrated acids or alkalies. The swelling extends deep down into the submucous tissue. The uvula may have the diameter of one's finger. There is intense hyperæmia. Sometimes there are hæmorrhages into the mucous membrane: this is called hæmorrhagic sore throat.*

4. **Necrotic Tonsillitis,** or *Necrotic Sore Throat.*—This name is applied to a disease which is very frequent in Leipsic, but seems to be rather rare in Erlangen, and in which the tonsils are the main seat of the pathological processes. The pillars of the fauces and the uvula are but slightly affected with a simple catarrhal inflammation. The tonsils are, as a rule, moderately swollen, seldom attaining great size. The mucous membrane covering them presents a whitish or grayish-white discoloration, often quite extensive, and most marked on the side toward the uvula. These spots are often erroneously said to be a white "coating"; but a more careful investigation shows that there is in reality a necrosis. The process may be superficial; sometimes it reaches quite deeply into the structure of the mucous membrane. It is not possible to pull off this white matter, as one can loosen croupous membranes, although little bits may perhaps be scratched off with a spatula or a pair of forceps. These particles are found, on microscopic examination, to be made up merely of detritus, bacteria, epithelium, and pus-corpuscles. The necrosis is almost invariably confined to the tonsils, and a sharp boundary-line separates it from the reddened and inflamed pillars of the fauces. After a few days the slough may come away, leaving behind an ulcer, which, though usually shallow, has sometimes a considerable depth. This generally cleans up rapidly. In severe cases, however, the floor of the ulcer consists for a number of days of a dirty necrotic material, which comes away only gradually. The worst cases may be properly called "gangrenous tonsillitis."

Necrotic tonsillitis is almost always attended by considerable fever and marked constitutional disturbance. Children particularly seem very ill in the first days of the attack. The cervical glands are usually swollen, but seldom as much so as in genuine diphtheria.

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* Another form with the same name occurs where there is violent tonsillitis with necrosis or gangrene. There is also a necrotic, hæmorrhagic sore throat accompanying scurvy, leukæmia, and analogous diseases.

Despite the rather ominous commencement, the disease does not last a great deal longer than the other forms of sore throat. It seldom continues more than five to eight days before a speedy convalescence begins.

The necrotic tonsillitis is distinguished from the follicular form by the greater area of the white or grayish-white spots. Still it should be particularly noted that sometimes combinations of these two varieties, or transitional forms, occur.

ÆTIOLOGY.—In our opinion, necrotic tonsillitis is in many instances an entirely different disease from genuine diphtheria. Perhaps it is often a very intense form of follicular tonsillitis, not limited, as it usually is, to the follicles. On the other hand, our experience in several epidemics of diphtheria in Leipsic and Erlangen has taught us that in all probability mild cases of genuine diphtheria of the tonsils do occur, and that these objectively resemble necrotic tonsillitis. This is the only way to explain what other physicians as well as ourselves have often observed—namely, that necrotic tonsillitis appears not infrequently in families at the same time with severe cases of genuine diphtheria. Besides, these cases of necrotic tonsillitis are sometimes followed by the characteristic "diphtheritic" paralysis. Still, more extensive pathological investigation is needed to settle the question of the ætiology of necrotic tonsillitis.

Diagnosis and Prognosis.—It is never very difficult to recognize a sore throat, and a little practice makes it easy in most cases to decide what particular variety is present, if we examine the objective changes carefully. It is very important in practice to distinguish diphtheria from the benign forms of inflammation. Follicular and necrotic tonsillitis are very frequently mistaken for diphtheria—an error which explains the success of a large number of remedies said to cure diphtheria. Many physicians call every case of sore throat, where there is anything white to be seen, "diphtheria." Certainty in diagnosis of genuine diphtheria can be gained only by practice; no description, however complete, can take the place of personal observation. It may be a help to remember that in both follicular and necrotic tonsillitis the white spots are usually limited to the tonsils, while in croupous sore throat the deposits are generally from the very first also situated upon the pillars of the fauces and the uvula. The white spots of the follicular variety can generally be recognized by their arrangement. The plugs are seen projecting from the follicles. In necrotic sore throat there is never a separable croupous membrane with its characteristic histological structure, but there is simply a superficial necrosis of the mucous membrane and parenchyma. In doubtful cases the condition of the cervical lymph-glands is not unimportant; as a rule, they are much more affected in diphtheria than in the benign cases. Inasmuch as we have seen that the milder forms of tonsillitis may be ætiologically related to diphtheria, it is well in practice to make a guarded prognosis, even in cases of follicular and necrotic angina, particularly in children; and we should always isolate the patient from other children.

Treatment.—These troubles run so favorable a course that active treatment is very seldom needed. The gargle usually prescribed generally gives the patient more discomfort than relief. The most common prescriptions are: Solutions of potassic chlorate (5–10 : 300), of alum (5–10 : 500), or of borax (10 : 300); salt and water; and weak solutions of carbolic acid or of permanganate of potash. To paint the parts is a useless and now almost obsolete proceeding. Inhalations of spray are better, with alkaline solutions, diluted lime-water, alum, or tannin. It is beneficial to put a cold wet compress around the throat. Children must be kept in bed, and adults are generally forced to go to bed, if the constitutional symptoms are well marked.

In parenchymatous tonsillitis with a tendency to suppuration ice will sometimes alleviate the pain. Often, however, the patient can not bear it. If there is evident fluctuation, we can make an incision with a spear-pointed bistoury, after guarding a portion of the blade with sticking-plaster. The best place is almost always in the velum palati, corresponding to the outer boundary of the tonsil. Great relief follows; and even if there is no distinct abscess an incision will usually give relief, if there is excessive swelling, and if there is a deeply-seated abscess will facilitate its spontaneous discharge. Punctures of this sort cause scarcely any pain.

We may add that if a person is subject to sore throat, he can lessen his liability to attacks by hardening his skin through the use of cold baths. [Hoffman has pointed out that in some cases permanent recovery can be attained by breaking down the partitions between the crypts of the tonsils, thus establishing thorough discharge of their secretions.—V.]

CHAPTER II

CHRONIC HYPERTROPHY OF THE TONSILS

Chronic hypertrophy of the tonsils occurs not only in those who have had repeated attacks of tonsillitis, but also in cases in which no occasion for it can be found. Even in childhood there may be well-marked hypertrophy, which must be due to a congenital predisposition.

The condition is at once revealed by inspection. There may be no signs whatever of any acute or chronic inflammation, or there may be an accompanying chronic pharyngitis. The tonsils bulge out in two great lumps. They may be so large as to touch the uvula on each side. Histologically, there is a genuine hypertrophy of the organ—that is, an increase of all its component tissues.

In many cases, where the swelling is moderate, there is no discomfort. The possessor of the tonsils is not aware that they are enlarged. In other cases the hypertrophy proves of clinical importance, inasmuch as all forms of sore throat are found to occur more frequently if the tonsils are enlarged, and to cause more trouble when they do appear. The hypertrophied organs may also be the seat of a chronic catarrh, which by extension gives rise to chronic nasal catarrh, catarrh of the Eustachian tubes, or hoarseness.

If the hypertrophy is considerable, the local discomfort may be quite marked. Swallowing is rendered difficult, if not painful. Frequently there is evident dyspnœa. The patient has to breathe through his mouth, and sometimes when asleep snores and snorts in a way to frighten one. Children are particularly apt to suffer in this manner. Many instances of *pavor nocturnus*, or "night terrors," in children are referable to this cause. We have already mentioned that cases of bronchial asthma sometimes seem to be connected with hypertrophy of the tonsils (see page 182).

Treatment.—The attempt to reduce the enlargement by applying lunar caustic, tincture of iodine, etc., usually fails. If there is much distress, if the patient is subject to frequent sore throats, or if the hypertrophy of the tonsils keeps up a chronic nasal or pharyngeal catarrh, then the simplest remedy is to remove the tonsils. The operation is free from any danger. The extirpation of hypertrophied tonsils can be accomplished either with the tonsillotome, the galvano-cautery, or with scissors and forceps. The latter way is the simpler, and is equally easy. [In adults, excision of the tonsils sometimes causes troublesome bleeding, and for them the galvano-caustic snare is the best means to employ.—V.]

CHAPTER III

CHRONIC PHARYNGITIS

Ætiology.—It is not practicable to distinguish between chronic catarrh of the soft palate and of the pharynx, for, as a rule, the two are combined. Sometimes the condition is the result of repeated acute attacks; sometimes—and probably oftener—it is due to persistent, injurious, local influences. A large number of cases originate in bad habits, or in abuse incident to certain vocations. Examples are seen in smokers, drunkards, singers, preachers, teachers, and men who work out-doors. In talking and singing, the soft palate is strained; or the disease is excited by breathing cold or impure air, or by such chemical irritants as alcohol or tobacco. In many cases, chronic pharyngitis follows chronic rhinitis or chronic laryngitis. The general passive congestion due to cardiac disease or pulmonary emphysema may sometimes promote the development, or prolong the existence, of a chronic pharyngitis.

Symptoms.—The local discomfort is often slight. The patient gets used to it, and does not mind it except when there is some exacerbation. It becomes a more important matter if the calling of the patient is interfered with, as in a preacher, singer, or teacher.

Deglutition is seldom impaired. There is often, however, a constant feeling of dryness, burning, or scratching in the throat. The patient has to clear his throat frequently, and often acquires an habitual, short, sudden cough, which may or may not be dry. The expectoration, if there is any, is almost pure mucus. When there are violent efforts to clear the throat, there may be a slight hæmorrhage from the dilated blood-vessels of the posterior wall of the pharynx, explaining the presence of small amounts of blood in the expectoration. This blood often has a dried-up look, as if not fresh. It sometimes leads over-anxious patients and physicians to suspect pulmonary hæmorrhage. The uvula becomes so long that its tip rests on the tongue or the posterior wall of the pharynx; and this gives rise to a peculiar and disagreeable sensation of tickling. All these uncomfortable feelings are temporarily increased if anything affects the throat unfavorably; and they are generally at their worst on rising in the morning, apparently because the mucous membrane has become dry, or a collection of tough mucus has formed during the night. Every one knows how drunkards have to hawk and cough in the morning, so that often they almost strangle or vomit.

On inspection, we generally find the mucous membrane reddened. Very often a number of dilated and tortuous veins are visible both on the soft palate and in the back of the throat. Of equal frequency is the appearance of numerous small gray projections, corresponding usually to swollen follicles or else to hypertrophied mucous glands. This is called granular pharyngitis. Small follicular ulcers are not infrequent. Exceptionally there are more extensive catarrhal ulcers. The mucous membrane of the posterior wall of the pharynx may present patches of opaque or thickened epithelium, giving the surface a grayish-white appearance.

Frequently chronic pharyngitis is combined with chronic laryngitis, evidenced by hoarseness; or with posterior nasal catarrh, or catarrh of the Eustachian tube, producing deafness and ringing in the ears.

VARIETIES OF CHRONIC PHARYNGITIS

1. **Chronic Catarrh of the Naso-pharynx, or Chronic Posterior Nasal Catarrh.**—This has the same ætiology as the ordinary form. It is practically important because the nose and ear are frequently involved.

The anatomical changes are essentially those already depicted under chronic pharyngitis. The region affected can not be seen by direct inspection, so that accuracy in diagnosis requires the use of a nasal speculum (see particulars in the works mentioned on page 135). The ordinary examination of the throat may reveal a condition which is quite characteristic of posterior nasal catarrh: a collection of muco-pus, or of firmly adherent dry crusts, rests upon the posterior wall of the pharynx, and can be seen to extend upward toward the naso-pharynx.

The local discomfort is somewhat similar to that experienced in chronic pharyngitis. There is a scratchy feeling, or a feeling as if there were a foreign body in the back of the throat, accompanied by a constant desire to blow the nose, hawk or cough. Dried and decomposing secretion often causes extremely foul breath. There is often also vertigo, and occipital headache.

In many cases the nostrils are stopped up. The posterior opening of the nostrils is closed in part by the swelling and hypertrophy of the mucous membrane, and in part by the accumulated secretions. The patient, therefore, usually has to breathe through the mouth. The ear is frequently involved. The catarrh extends into the Eustachian tubes and the tympanic cavity, or the opening of the tubes is occluded with the secretions. For a detailed consideration of the deafness, tinnitus, etc., thus produced, consult works on otology.

2. **Pharyngitis Sicca**, or "*Dry Atrophic Catarrh of the Throat and Naso-pharynx.*"—This name is applied to an atrophic disease of the mucous membrane, which sometimes is spontaneous and sometimes is a sequel of chronic pharyngitis. The whole mucous membrane of the pharynx and the naso-pharynx (seen with the rhinoscope) seems pale, smooth, and perfectly dry, and has a peculiar luster, as if varnished. Here and there tortuous veins project from the general anæmic surface.

If an opportunity is afforded to examine the mucous membrane microscopically, it will be found that the atrophy involves all the elements of the tissue, though the follicles and mucous glands suffer most.

This condition may not cause any symptoms, but, in many cases, the patient suffers constantly and considerably. The chief trouble is a feeling of dryness in the throat, rendering deglutition difficult or even painful. There is also a constant desire to clear the throat. The secretion hawked up may be scanty and tough or more abundant, and it is often tinged with blood. Actual coughing may also be due to pharyngeal trouble ("throat-cough"). Talking is often rendered difficult, the voice grows weak and it becomes easily tired. In severe cases there is considerable general debility. Not infrequently pharyngitis sicca is associated with atrophic rhinitis (*q. v.*), but it occurs also where there is no nasal disease.

The disease is most frequently seen in the elderly, but it also occurs in children and young persons. It is especially common in ill-nourished individuals or in those who are suffering from such diseases as tuberculosis or chronic nephritis.

3. **Hypertrophic Catarrh in the Pharynx and Naso-pharynx.**—An opposite condition, of hypertrophy, sometimes results from chronic catarrh. The changes consist mainly in hyperplasia of the lymphatic tissue, and they are usually termed "adenoid growths." The choanæ and the posterior extremity of the nasal septum may be almost completely hidden by these growths, as they extend down from the roof of the pharynx in grayish-red, uneven masses. In many cases the hypertrophy seems to originate chiefly in Kölliker's "pharyngeal tonsil."

The adenoid growths are especially common in childhood. The symptoms consist in a change of the voice (which loses its reverberating quality and becomes nasal), frequent snuffling and hawking, and a tough mucous secretion, often tinged with blood. Not infrequently there is headache. Of greater importance is ear-trouble, which is often occasioned by the growths.

An accurate diagnosis requires rhinoscopy. Positive results are also often obtained by palpation. The index-finger, being pressed backward and bent upward, can touch the protuberances and the enlarged pharyngeal tonsil in the naso-pharynx.

Prognosis.—In all varieties of chronic pharyngeal catarrh, the prognosis as to recovery is doubtful, for in all severe cases the process is very obstinate, and permanent restoration to health exceptional. It is essential for the best results that all injurious influences should be removed. Even if decided improvement occurs there is a tendency for the trouble to grow worse again, and for acute exacerbations to occur.

Treatment.—Many of the milder cases never apply to a physician. The patient uses some domestic remedy or gargle, or becomes so accustomed to the disagreeable sensations that he does not consider it necessary to do anything in particular about them.

The treatment of a well-developed case requires great patience and persistence on the part of all concerned. If there is some underlying disease, such as pulmonary or cardiac disease, that must be treated. All exciting causes must be avoided. Energetic local treatment is also indispensable. This has been greatly elaborated by specialists, and for the many details we must refer to their writings; but the following remarks will meet the requirements of ordinary practice:

Gargles are seldom satisfactory, for they never reach farther than the soft palate. Inhalations are better; we may use solutions of alum or tannin, or, in mild cases, of common salt. Still more efficient is the painting of the entire surface of the pharynx with some concentrated solution. The physician usually has to perform this, although some patients learn to do it for themselves. Proper solutions are: Argentic nitrate, five or ten per cent.; tannin, eight to twenty per cent.; tincture of iodine, either pure or diluted; or iodized glycerine, composed of pure iodine, parts 0.5; iodide of potassium, 2; glycerine, 20, with the addition, if desired, of two drops of oil of peppermint. These applications must reach all the diseased surface. If the naso-pharynx is involved, the brush must accordingly be bent upward, to reach that region. For this a mirror may be needed. It is very important to make the applications to the mucous membrane itself, freed from any interposing secretions.

In the treatment of chronic posterior nasal catarrh the nasal douche (*vide* diseases of the nose) plays an important part. It should be used two or three times a day. It not only removes the collected secretions, but it is a means of making local applications. The instrument is merely a fountain-syringe. The nozzle must be of a size to fill the nostril completely. The force of the current should always be moderate, and the patient's head should be sharply flexed forward. The fluid used—the best is a one-per-cent. solution of sodic chloride or bicarbonate —must have about the temperature of the body. Other medicated solutions must be very weak, such as sulphate of zinc, 1 to 1,000.

The insufflation of powders into the throat can be made through any small glass tube, three to six times a week. Alum or tannin may be used, either pure or mixed with equal parts of pulvis gummosus [P. G., made of gum-arabic, three parts; licorice-root, two parts; and sugar, one part], or 3–5 parts of nitrate of silver to 100 of starch. For the naso-pharynx, a bent tube of glass or hard rubber is to be introduced through the mouth. There are numerous "insufflators" to be had at the instrument-makers.

Many baths enjoy a great reputation for the cure of chronic pharyngitis. Besides Ems, there are Reichenhall, Kreuznach, Salzungen, the cold sulphur springs, such as Weilbach, and many others. Good results are also achieved in Kissingen and Marienbad, if these places are favorable to the patient's general constitution.

In pharyngitis sicca, the nasal douche with a one-per-cent. salt solution is to be recommended. It is sometimes also beneficial to paint the parts with solution of argentic nitrate, iodized glycerine, etc. Many irritating influences which do harm in common pharyngitis seem sometimes actually to benefit this form—such as smoking and taking snuff.

In treating the hypertrophic forms of pharyngitis and the adenoid growths, cauterization with nitrate of silver suffices for the milder cases alone. A thorough and permanent cure can be achieved only by removing the growths with the galvano-cautery.

[To Hooper, of Boston, belongs the credit of showing that a radical operation for the removal of adenoid growths of the naso-pharynx can safely be done under anæsthesia. The blood does not, as was feared, run into the air-passages, but either into the stomach or outwardly. Anæsthesia thus renders it possible to scrape away with the finger or tear away with forceps the hypertrophied tissue at one sitting, and to do it in the child before secondary changes have taken place in the facial expression, the jaw, the ears, or the form of the chest.]

CHAPTER IV

RETROPHARYNGEAL ABSCESS

Retropharyngeal abscess is formed by a suppurative inflammation of the connective tissue lying between the posterior wall of the pharynx and the spinal column. It is a serious disease, although a rare one. If unrecognized, it proves fatal in many instances; while, if a correct and timely diagnosis is made, the patient can usually be easily cured. It is commonest in childhood, and before the second year. It almost always appears as a primary, acute disease, without any special cause being evident. Probably the agents which excite the inflammation penetrate into the tissue from the pharynx. The idea that the inflammation originates in the small lymphatic glands which lie in front of the vertebræ lacks proof as yet.

The disease attacks not only weakly children, but those who have been perfectly healthy and vigorous. The child grows restless and fretful, and does not nurse well. Apparently, deglutition soon becomes painful, but one can not be certain about this except in older children. Generally, the respiration quickly takes on a peculiar stertorous character, particularly during sleep. Mucus collects in the mouth and throat. Upon swallowing, there is often regurgitation through the mouth or nose, or some of the food gets into the windpipe and causes violent coughing. The lymph-glands of the jaws are usually somewhat swollen, and the neighboring parts may seem slightly œdematous. After a week or two the dyspnœa gradually increases. Respiration becomes more and more laborious, with loud rattling, and signs of stenosis. The jugular veins become distended, the lips cyanotic, and portions of the thorax are retracted during inspiration. The voice is feeble, and may be hoarse and indistinct.

The correct interpretation of these symptoms, which are common to various disorders, requires a careful examination of the throat. It must be confessed that this has its difficulties in an infant. Still, we can sometimes see distinctly a swelling in the posterior wall of the pharynx. This may be either in the median line or on one side. All doubt is removed by digital examination, in making which, however, we must insert a wedge between the teeth, to avoid being bitten. The finger detects fluctuation.

The diagnosis once established, the abscess must be opened at once. We should not delay, even if the dyspnœa has not yet become extreme. To use the finger-nail for the purpose, as has been recommended, is permissible only in an emergency. As a rule, incision is made with a bistoury, of which all but the point is guarded with sticking-plaster. The left index-finger is placed upon the abscess, and used as a guide. Meanwhile, the child's head is kept upright, and, as soon as the cut is made, bent over forward. The pus pours out in abundance. It is advisable to syringe out the mouth repeatedly with lukewarm water. The threatening symptoms vanish almost instantly upon the escape of the pus. Exceptionally, the abscess refills and requires a second incision.

If the trouble is not correctly diagnosticated, or if the abscess is not opened promptly, the patient may suffocate. The abscess, however, may burst spontaneously; then there is either speedy recovery, or asphyxia from the pus filling the larynx. In some instances, when a retropharyngeal abscess has not been properly treated, the pus has gravitated far down into the neck and posterior mediastinum. The recognition and incision of the abscess may prove very difficult if from the start it is situated lower down in the throat than usual.

Analogous to this acute idiopathic abscess of which we have been speaking is the chronic abscess due to caries of the cervical vertebræ. This occurs in children as well as adults. Incision of such abscesses is indicated only when there is danger of suffocation. Because of their tuberculous character, the prognosis is, of course, much more unfavorable than in ordinary suppurative inflammation.

Retropharyngeal abscesses sometimes occur in pyæmia or other severe acute infectious diseases, but they have hardly any interest except to the pathologist.*

SECTION III

DISEASES OF THE ŒSOPHAGUS

CHAPTER I

INFLAMMATION AND ULCER OF THE ŒSOPHAGUS

Ætiology and Pathology.—The various forms of œsophageal inflammation and ulceration are not of very great clinical importance. The processes are seldom of a severe grade, or, if so, they are generally a part of some complicated disease, to which they seldom contribute prominent symptoms. Very likely the milder forms of inflammation occur frequently, but the symptoms are hardly ever characteristic.

A simple catarrhal inflammation of the œsophageal mucous membrane may be caused by swallowing substances which are injurious mechanically, chemically, or from their temperature. It may also occur in the general infectious diseases, such as typhoid and typhus fevers, and the acute exanthemata. Any inflammation of neighboring tissues may extend into the œsophagus. Chronic catarrh is seen in heart disease, from the passive congestion. It is also found in the vicinity of other chronic œsophageal diseases, particularly cancers and diverticula (*vide infra*).

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* Tuberculosis of the pharynx is spoken of in the chapter on pulmonary tuberculosis, p. 248. New growths in the mouth or pharynx belong to the domain of surgery.

The acute catarrh is distinguished by not having the usual increase of secretion. The epithelium grows spongy, as a rule, and is cast off more rapidly than usual, so as to suggest the name of a desquamative catarrh. It is in only a few cases that the scanty mucous glands become swollen and look like papules upon the surface of the membrane; this form is called follicular catarrh. In limited areas the desquamation may be complete, giving rise to small catarrhal erosions. Likewise, the swollen follicles may break down into small follicular ulcers.

In chronic catarrh there is a moderate increase in the secretion of mucus, and a marked thickening of the epithelium. In very protracted cases actual papillomata may finally be formed. In some cases ulcers are seen.

Croupous and diphtheritic inflammations of the œsophagus are very rare. We have already said that the specific pharyngeal diphtheria frequently extends into the larynx, but only exceptionally into the gullet. Still, we have ourselves seen in a child a stricture in the upper third of the œsophagus, which was said to have been a result of a severe attack of diphtheria. Isolated cases of diphtheritic œsophagitis have also been seen in connection with severe infectious diseases, such as typhus, typhoid, small-pox, cholera, pyæmia, and pulmonary tuberculosis, as well as in the course of Bright's disease and cancer. In variola it is not unusual for pocks to appear upon the œsophageal mucous membrane.

A purulent, phlegmonous œsophagitis now and then attacks the submucous layer. It may be either diffuse or circumscribed. The mucous membrane is dissected up from the muscular layer by the pus, and pushed inward, so as to diminish the lumen of the œsophagus more or less. Most of the cases end by the discharge of matter into the tube, when complete recovery may ensue. If the mucous membrane, however, has been extensively undermined, Zenker states that a fissure-like cavity may be left, even after healing has taken place. Its walls grow smooth, and finally acquire a layer of fresh epithelium.

Purulent œsophagitis is caused either by the presence of foreign bodies in the œsophagus, or by purulent inflammation in neighboring parts, as in glandular abscess, vertebral abscess, or laryngeal perichondritis. It has now and then resulted from the action of concentrated acids and the like upon the mucous membrane.

The action of corrosive poisons (corrosive œsophagitis) is to cause necrosis of the tissues, which in its turn produces inflammation. The inner surface of the œsophagus is converted into a rotten, hæmorrhagic, sloughing mass, of a dirty gray or almost black color. The muscular layer itself may be partly destroyed. If death does not occur speedily, the necrosed portions come away, leaving extensive purulent ulcers behind. These, if they heal at all, cause large cicatrices and stenosis.

Symptoms.—The milder cases, as we have stated, produce almost no distinctive symptoms. Possibly there may be pain along the œsophagus, or at some one point in it, during deglutition. In a more severe case the pain may be great; but the other symptoms are usually too grave for this to excite special attention. Laborious deglutition, and the feeling as if the food were inclined to stick in the throat, result from implication of the muscular layer. A diagnosis of the particular form of œsophagitis is attainable only when the ætiology guides us to it.

Treatment must be purely symptomatic. No solid food should be taken. The pain is to be allayed by bits of ice, or by morphine.

CHAPTER II

DILATATION OF THE ŒSOPHAGUS

1. Diffuse Dilatation

Diffuse, spindle-shaped dilatation of the œsophagus is observed as a result of stricture of the cardiac orifice. At first the muscular coat hypertrophies as the orifice contracts, and is able to overcome the obstruction, so that there is no dilatation; but as soon as the muscles are paralyzed, and food collects behind the stricture, the dilatation begins and keeps on increasing. The ectasis is greatest at the lower end of the tube, as is natural from its mode of origin, and gradually diminishes upward.

There have been a very few well-substantiated instances of this diffuse spindle-shaped dilatation, without any demonstrable stenosis of the cardiac orifice. Their cause is unknown. Sometimes the walls of the œsophagus may have been rendered more yielding and less contractile by a precedent inflammation or other disorder. In other cases, some chance bend or distortion of the lower end of the œsophagus may have produced a mechanical obstruction. In some cases the exciting cause is said to be a blow on the chest, or the lifting of a heavy weight. Perhaps in many cases there is a congenital anomaly.

The symptom of this condition, when well developed, is a chronic difficulty in deglutition, lasting perhaps for years. The patient himself feels that most of the food he eats does not reach the stomach, but lodges higher up. Usually the food is soon afterward vomited, or rather gulped up. When there is stenosis of the cardiac orifice, the explanation of these symptoms is easy. It is much harder to explain the almost equal dysphagia when there is dilatation without stenosis. Sometimes a localized bulging of the wall causes the food to collect in that spot, and thus to obstruct the lumen. As is to be expected, the partial or complete hindrance to the ingestion of food results in marasmus.

If there is stenosis, it can easily be detected with the œsophageal sound, and all the symptoms can thus be explained. In the rare cases, however, of diffuse dilatation without stenosis, the use of the sound does not give us so much information. If the instrument passes readily into the stomach, we may safely exclude stricture; but, in one case of our own, we made an erroneous diagnosis of a diverticulum, because the sound sometimes glided readily into the stomach, and sometimes could not be passed. A pocket must have been formed at the lower end of the dilated tube, in which the sound caught.

The treatment is directed chiefly to the satisfactory nourishment of the patient; for the œsophageal trouble itself is dangerous only as it prevents the taking of food and leads to starvation. We do not speak of the initial lesion, if there be one, which causes the dilatation. If nourishment can be given through a stomach-tube, the patient almost invariably shows a rapid improvement, which lasts as long as the artificial feeding can be kept up. If, however, any cause prevents the introduction of the tube, we must resort either to nutrient enemata (*vide infra*)—and these will not support the system indefinitely—or we must make a gastric fistula. In the latter case the prognosis depends on the success of the operation and the nature of the original lesion.

2. Diverticula

Ætiology and Pathology.—Circumscribed pouches in the wall of the œsophagus are termed diverticula. They are divided into two essentially distinct varieties according to their mode of origin. Zenker has given them the names of pressure and traction diverticula.

The diverticulum due to pressure is extremely rare. It is caused by pressure upon the mucous membrane from within, by which some abnormally weak spot is forced outward. All cases that have been carefully examined thus far have shown that, histologically, the wall of the diverticulum is not the distended but otherwise unchanged wall of the œsophagus, but is composed exclusively of the mucous membrane and the thickened submucous coat. We are therefore obliged to suppose that the mucous membrane is pushed out like a hernia through some gap in the muscular coat. It is only about the neck of the diverticulum that any muscular fibers are found.

The original factor, therefore, in the occurrence of a pressure diverticulum is apparently to be sought in some circumscribed lesion of the muscular coat. As a result of several observations, it is established that a foreign body, sticking in the throat, may separate some of the muscular fibers and push the mucous membrane through the gap thus formed. Or a severe injury leads to a trifling rupture of the muscular coat, and then the food, as it is being swallowed, presses out the mucous membrane at this weakened spot. There are still many other cases in which the true origin of the diverticulum remains obscure.

As soon, however, as the formation of the pouch has once begun, there are many influences to make it grow larger. Each successive bit of food, as it glides by, presses upon this yielding and inelastic spot. Gradually a little sac is formed, in which bits of food lodge. These exercise a constant pressure upon the walls of the pouch, and by their weight drag it bodily downward. The larger the pouch, the more it holds, and consequently the more it grows. Thus a pressure diverticulum of the smallest size originally may gradually attain to a diameter of four inches or more. The general shape of the diverticulum may approach the hemispherical, or it may be more cylindrical or pear-shaped.

It is remarkable that, with very rare exceptions, these pressure diverticula are always situated at the beginning of the œsophagus, or rather between it and the pharynx, and almost invariably affect the posterior wall. The pouch hangs, therefore, in front of the spinal column. It pushes out through the lowest fibers of the inferior constrictor of the pharynx; and the feebleness of this muscle is apparently a potent factor in determining the precise point of origin.

The cases thus far seen have been almost all in men, and at a rather advanced age. A few cases have occurred in children.

Traction diverticula are much more common, but in most instances have little interest except for the pathologist. They are not infrequently found unexpectedly at the autopsy. Rokitansky, and later Zenker, have given explanations of their occurrence: some tissue, which has formed adhesions to the œsophagus, contracts and gradually pulls out the œsophageal wall in the shape of a funnel. Bronchial glands are apt to be the seat of the contractile change. These glands are situated near the bifurcation of the trachea, and accordingly the traction diverticula occur oftenest at this level. There may be two or three in one subject. They are rarely over a third of an inch in depth. From within, the mucous membrane, much wrinkled transversely, is seen to be drawn toward the apex of the diverticulum. The wall of the latter consists either of the mucous membrane alone, bulging out like a hernia, or of the mucous membrane covered by the muscular layer. Inasmuch as children quite often suffer from suppuration and caseation of bronchial glands, with subsequent shrinkage, we see why traction diverticula are frequent in children.

Clinical History.—The large pressure diverticula always cause grave symptoms, for they obstruct more and more each day the passage of food. At first there is scarcely any disturbance. Gradually, however, deglutition is impeded.

A portion of the food lodges in the pouch, and is either wholly or in part regurgitated, though perhaps not immediately. Decomposition is apt to take place in the contents of the diverticulum, giving rise to foulness of the breath and to nausea. The danger reaches its climax when the distended sac presses sidewise upon the œsophagus and closes its lumen, so that no food reaches the stomach. After protracted strangling and vomiting, the material may be in part ejected, and the patient enabled once more to swallow.

Of course the symptoms in individual cases depend upon the mechanical conditions present, and they may vary greatly. Patients contrive all sorts of manipulations, by which they manage to get at least some portion of their food down. Such individuals may maintain a tolerable degree of nutrition for years, although they scarcely ever are in a normal condition. But at last some cause or other renders the amount of food ingested inadequate; whereupon a rapidly progressive marasmus sets in, and the patient will inevitably starve to death unless some relief is afforded.

The most valuable objective evidence in these cases is gained by the use of the œsophageal sound. If the sound enters the sac, its passage is impeded. If it happens to slip by the mouth of the diverticulum, it glides readily into the stomach. This varying result may sometimes be obtained at one sitting by repeated trials, and is of the greatest importance in making the diagnosis.

In some instances where the sac was large, a tumor in the neck has been observed at one side of the trachea, appearing after eating and disappearing when the sac emptied itself. Symptoms due to compression of the recurrent and phrenic nerves and of the blood-vessels have been noticed in some cases.

Auscultation of the œsophagus during the act of swallowing has been practiced, and, of late, attempts have been made to examine it with a speculum. Whether these methods of investigation will prove valuable for diagnosticating diverticula, experience must determine.

The traction diverticula are usually of no clinical importance. They do not affect deglutition at all, and their size is too limited to permit any great accumulation of food in them. There is but one way in which they are dangerous: the apex of the funnel may undergo ulceration and perforation. A foreign body, such as some bit of food, produces necrosis of the wall, by what is probably at first a purely mechanical irritation. The tissue ulcerates; and then the inflammation may gradually progress till it causes a severe and usually fatal illness. The most frequent event is perforation into a bronchus, followed by the aspiration of food and pulmonary gangrene; or the perforation may take place into the pleural cavity, exciting an ichorous empyema. In other cases the pericardium or a large vein has been perforated. Many a case of apparently spontaneous pulmonary gangrene, or purulent inflammation of the anterior mediastinum, or empyema has been found at the autopsy to have been brought about in the manner above indicated. These occurrences are fortunately, however, exceptional.

Treatment.—The only possible way of treating the large pressure diverticula successfully would be by operation. Perhaps surgery will some day win victories in this domain. In the meanwhile our efforts are confined to sustaining the patient. If he can not swallow, we must try to feed him through a tube. As long as this is possible, starvation is averted. It is well to have the patient pass the tube himself. He will find out how best to avoid the sac and reach the stomach. If food can no longer be given in this way, there remain two alternatives: rectal feeding (*vide infra*), or making a gastric fistula. As to the latter, there has been thus far very little practical experience, because cases are so rare.

The traction diverticula admit of no special treatment. If the events above mentioned occur, we must endeavor to meet the indications of the individual case.

CHAPTER III

STENOSIS OF THE ŒSOPHAGUS

Ætiology and Pathology.—Contractions of the œsophagus occur with such relatively great frequency that they are the most important of all its disorders. They originate in various ways. By far the commonest cause is ring-shaped carcinoma of the tube. The new growth in the mucous membrane encroaches more and more upon the lumen of the œsophagus, until finally it fills it. Carcinoma will be discussed at length in the next chapter. We shall here confine our attention to its purely mechanical action in causing stenosis.

Œsophageal tumors other than cancer are very rare. Fibrous pedunculated polypi have been observed a few times. They usually originate in the lowest portion of the anterior wall of the phaynx, hanging down into the œsophagus, which they may thus obstruct.

A second cause of stenosis is the contraction of cicatrices of the œsophageal wall. The most frequent occasion for this is the extensive ulceration caused by caustic poisons, such as concentrated acids or alkalies. If the victim escapes a speedy death, he is almost certain to have extensive scars formed in the wall of the œsophagus. These scars radiate irregularly in all directions, and, contracting, may almost completely close the tube.

Ulcers from other causes, resulting in stenosis due to the scars they leave, are among the greatest rarities. Syphilis has been the well-established cause in some instances, and Quincke has described a few cases in which there were ulcers at the lower end of the œsophagus analogous to the round ulcer of the stomach, or "ulcer due to digestion" (*vide infra*). These ulcers also may eventually produce cicatricial stenosis.

A third and rare cause of stenosis of the œsophagus is compression from tumors external to it. Such swellings may originate in the thyroid gland, or in the lymph-glands of the neck or the anterior mediastinum; or the swelling may be due to a vertebral abscess or an aortic aneurism. This form of stenosis is seldom extreme, for the portion of the tube pressed upon is usually limited.

Next on the list after stenosis due to compression is usually placed what is called intermittent dysphagia (*dysphagia lusoria*). This term is applied to the difficulty in swallowing which is said to be caused by an anomaly in the course of the right subclavian artery. The artery is given off as the last branch from the arch of the aorta, and runs toward the right side just behind or just in front of the œsophagus. It seems, however, *a priori* improbable that the feeble pressure of this vessel as it pulsates should impede deglutition; nor has it yet been proved to do so. It would be more natural to believe, what was indeed the original explanation of the phenomenon, that a large morsel of food passing down the œsophagus compresses the vessel and thus excites uneasiness and palpitation; but so far as we know even this is extremely rare, if it ever occurs.

Stenosis due to foreign bodies belongs to surgery. It need not be said that the clinical symptoms differ greatly in different cases. Not only the obstruction, but also a possible laceration and consequent inflammation are to be considered. Occasionally thrush has been abundant enough to cause pronounced symptoms of stenosis.

Above the point of stenosis, no matter how the condition arose, if only it is well developed and has lasted a certain length of time, the circular fibers of the muscular coat are more or less hypertrophied. This hypertrophy is due to the increased force required to propel the ingesta downward. In many cases the tube is also diffusely dilated above the stenosis.

Symptoms.—The effect of every œsophageal stenosis is to render deglutition difficult. If the case is a mild one, the patient experiences nothing more than a moderate pressure in the œsophagus upon swallowing. He feels that the morsel is longer than usual in reaching the stomach. Very soon he notices that solid food and large morsels can be swallowed only with difficulty. Accordingly, he is gradually led to confine himself to a liquid diet, takes small mouthfuls, and always washes down any solid food with a swallow or two of liquid. The narrower the stenosis, the more he is troubled. Finally, even liquids can be taken only slowly and in sips.

It must not be thought that the dysphagia just described is due exclusively to the mechanical obstruction of the lumen. Sometimes a patient is almost entirely unable to take nourishment, and yet at the autopsy no adequate mechanical obstruction is found. The dysphagia must therefore be due to some lesion of the muscular coat of the œsophagus. The impaired contractility of the muscular coat at the affected spot is always a potent factor in impeding deglutition.

As soon as the dysphagia has become considerable there is usually regurgitation of food. At first only a portion of the food comes up, but at last all of it. If the tube has become dilated above the stenosis, food may collect for some hours, and then be regurgitated, mixed with an abundance of very tenacious mucus. We saw a case of this kind in which the patient could fill the sac above the stricture with quite a large amount of fluid without a drop reaching the stomach. If he bent his head sharply forward, the collected fluid would run out again through his mouth. It was not until the pouch was completely filled that a small amount of liquid would trickle through the stenosis into the stomach.

Although the dysphagic symptoms above described generally imply œsophageal stenosis, the diagnosis can not be really established without using a sound. Upon introducing this, it is usually easy to detect the obstacle, which may either allow the instrument to pass, with a noticeable jerk, or else prevent its further progress. By measuring the length of the portion introduced before the stenosis is reached we can learn its position. On the average, the entire distance from the teeth to the cardiac sphincter is in adults sixteen inches (forty centimetres); from the teeth to the beginning of the œsophagus, six inches (fifteen centimetres); and consequently the length of the latter is about ten inches (twenty-five centimetres). If we succeed in passing a smaller sound through the stricture, the feeling as we move it back and forth will give us some idea of the length of the stenosis, or will demonstrate the existence of several lying one below the other, etc. If the end of the sound can be moved about very freely above the stenosis, we may conclude that the tube is dilated there.

Hamburger has employed auscultation of the œsophagus for diagnostic purposes. If we listen behind, to the left of the upper dorsal vertebræ during deglutition, we hear a gurgling sound, due to the act of swallowing, extending down the tube to the stenosis, but no farther. Then come all sorts of sounds, some of them caused by the fluid trickling slowly through the narrow part, and some caused by regurgitation. In general, the results obtained by auscultation are rather variable and uncertain.

Having established the fact of the existence of a stenosis, we have next to determine its nature, which is our chief guide to prognosis and treatment. In certain instances the history of the case gives us the needed information. The diagnosis of cicatricial stricture can hardly be made unless the patient himself tells us of being burned or injured by caustic poisons. The previous history is likewise of great importance if the stenosis be due to foreign bodies or to syphilis. If no decisive ætiological factor can be elicited, we must carefully examine the neck and thorax, with regard to the possible existence of a swelling compressing the œsophagus. When an aortic aneurism has acted in this way, a rhythmical movement has sometimes been communicated to the free end of a sound introduced as far as the stenosis. If the physical examination does not reveal a compressing tumor, and particularly if the stenosis has developed gradually and in an elderly person, we are almost compelled to assume that there is cancer of the œsophagus. This is, after all, by far the most frequent cause of œsophageal stricture. If the new growth has ulcerated, a little portion of it may adhere to the end of the probe, and, on microscopic examination, render our diagnosis of carcinoma certain.

The prevailing characteristic in stenosis of the œsophagus is inanition, increasing as the dysphagia increases. The patient gets to be very much emaciated, and so feeble that he can not leave his bed. The temperature is subnormal; for weeks it keeps at 95° to 97° (35°–36° C.). The pulse grows very small and slow, being 40 to 60 per minute. The heart-sounds are soft. Respiration is superficial and slow; and toward the close of life short pauses occur after expiration, before inspiration begins. The stomach and intestines are so empty that the abdomen is very concave, while the abdominal walls usually feel tense and resistant. In all cases when the nature of the stenosis precludes the possibility of cure or improvement, death results from increasing exhaustion, the lamp of life gradually flickering out.

Prognosis and Treatment.—In prognosis the main factor is of course the nature of the stenosis. If it is due to foreign bodies or to cicatrices, it may be completely cured. In stenosis from other causes it is often possible to produce considerable improvement, at least temporarily. The final result must be confessed to be usually unfavorable, as we should expect from the nature of the original trouble.

The treatment is chiefly mechanical. We shall not speak of operations for the removal of new growths, etc. What we do refer to is a methodical and gradual dilatation of the stricture. Its results are sometimes brilliant, particularly in cicatricial stenosis. Other varieties, such as the stenosis from cancer, may sometimes undergo considerable though but temporary improvement with this treatment.

The best instrument to employ is the flexible, so-called English, œsophageal bougie. It is made in all sizes. If the stenosis is very narrow indeed, we may have to resort to catgut at first. Whalebone bougies, with olive-shaped ivory tips of various sizes to screw on the end, are also good, except that, being stiffer, there is more danger in using them. For introduction of the bougie, the patient should be seated, with the head slightly extended backward. The first two fingers of the left hand are introduced into the throat and guide the instrument, previously well oiled, over the back of the tongue and the epiglottis into the œsophagus. Of course, no violence must be used. Otherwise a perforation might occur if there were a soft, broken-down cancer, or an aortic aneurism. However, such a misfortune is very exceptional.

The use of the bougie is almost invariably beneficial if the stricture can be passed. The patient generally finds that he can swallow easier than before, and will himself request a repetition of the performance. If the patient is an intelligent person, it is advisable to have him introduce the bougie himself. Patients often acquire even greater skill with it than the physician has. The bougie should be passed regularly once a day, or, at most, twice daily; and in favorable cases we shall be able gradually to increase the size. If so, the symptoms speedily abate, and, with the increased ingestion of food, the patient gains flesh very fast.

If the stenosis is extreme, and, although it admits the bougie, does not allow of sufficient nourishment, we must pass a tube into the stomach through which to introduce liquid food. Milk is the best food to choose. Raw eggs, sugar, wine, etc., may be mixed with it. The various infant's foods and Hartenstein's "leguminose" are also excellent. Their consistence is favorable for the purpose, and they supply a considerable amount of nourishment in a small bulk. Until recently the sounds and dilators employed in stenosis of the œsophagus have been usually introduced by the mouth, but of late the practice has been constantly growing of treating stenosis through an artificial opening in the œsophagus. Mechanical dilatation can thus be accomplished with much greater rapidity and completeness. In the surgical wards here in Erlangen admirable results have been obtained in this manner in cicatricial stenosis; whereas in carcinoma of the œsophagus the improvement is of course merely temporary, although the symptoms are greatly alleviated. Further particulars with regard to œsophagotomy are to be sought in text-books on surgery, where a description of some other modes of treatment of œsophageal stenosis will be found, including the insertion of self-retaining canulæ, and the creation of an artificial gastric fistula.

We desire to append a few remarks as to the nourishment of patients by enemata, to which we are particularly apt to have recourse in cases of œsophageal disease.

Brilliant results are never to be expected from rectal feeding. It is indeed probable that life may be by this means somewhat prolonged, but not indefinitely. The moral effect, however, is very valuable, when the patient could otherwise receive no nourishment whatever. The sufferer feels that something is being done to avert absolute starvation.

The simplest materials for the nutrient enemata are milk, eggs, and wine; to which we may add pepsine and pancreatine in the hope of promoting absorption. Leube's pancreatic meat emulsion is still better, although more troublesome in its preparation. Leube's directions are as follows: About five ounces (grammes 150) of meat, cut very thin and then minced finely, and about two ounces (grammes 50) of minced pancreas (from the calf) free from fat, are to be stirred with about three ounces (grammes 100) of lukewarm water until the mixture has the consistence of gruel. Before it is injected, the rectum should be cleansed by an enema of plain water. One such enema is to be given daily. Also, when enemata of milk and eggs are given, the rectum must invariably be first rinsed out with plain water. Too large an amount ought not to be given at one time, say about two ounces (grammes 50), and the fluid must be raised to about the temperature of the body. [The limit in quantity set by the author is smaller than many patients can retain. If the fluid is injected gently and slowly, four to eight ounces may be administered every six hours, and cases are on record of patients retaining even sixteen ounces. The rectum seems capable of a certain amount of training in this regard, so that the amount given may eventually be greater than at first. Unusual irritability may be diminished by adding to the enema about five minims of laudanum.—V.]

CHAPTER IV

CANCER OF THE ŒSOPHAGUS

Ætiology and Pathology.—Cancer is the most important and most frequent affection of the œsophagus. We have already mentioned in the preceding chapter that often stenosis is the result of carcinoma in the œsophageal walls.

Little is known about the ætiology. It has been often maintained that mechanical, chemical, or thermic irritation of the mucous membrane may result in the development of cancer; but this is not certain. It receives some support from the remarkably frequent occurrence of œsophageal cancer in hard drinkers. Now and then the patient himself will allege a perfectly definite cause for his disease, such as the lodging of a foreign body, or the swallowing of a very large or very hot morsel. Still it is hardly possible in any particular case to decide how much value such statements have. It has been maintained that the carcinoma sometimes develops in the scar of an old ulcer. This is of interest when we recall the similar fact in regard to gastric carcinoma (*vide infra*).

Œsophageal cancer follows the general rule in being most frequent in elderly people—somewhere between forty and sixty years of age. The male sex is decidedly more often attacked than the female.

As we might expect from the histological character of the epithelium lining the œsophagus, primary cancer here is invariably composed of pavement cells. The new growth may be either hard, firm, and fibrous, or it may be soft, succulent, and but scantily supplied with connective tissue. The first variety corresponds to the "scirrhus" of older writers, and the second to "medullary" cancer. Usually the new formation encircles the entire tube like a ring, extending three to ten centimetres longitudinally. Exceptionally a still larger portion of the œsophagus is involved, sometimes almost all the mucous membrane. The tumor is usually seated in the lower and middle thirds of the œsophagus, being much rarer above.

Symptoms and Complications.—In the great majority of cases the symptoms are those of a gradually increasing stenosis, with its results. We may therefore refer to the preceding chapter for most of the particulars. There are, however, exceptional cases in which the carcinoma is flat and entails no dysphagia, or so little that œsophageal trouble may not be suspected. We have repeatedly seen cases of extensive secondary hepatic cancer, or of pulmonary gangrene (*vide infra*), in which the real primary disease was a flat cancer of the œsophagus, which gave no clinical signs of its existence, and was therefore not diagnosticated.

It is characteristic of the stenotic symptoms produced by œsophageal cancer that sometimes a considerable and apparently spontaneous amelioration occurs. This is because of an ulceration of the new growth. It crumbles away, as the result of superficial disintegration. The tumor is transformed into an ulcer, and one can easily understand how this may occasion a temporary improvement in deglutition.

Important clinical symptoms may result from conditions secondary to the new growth. The cancer may extend to neighboring organs. Not infrequently the cardiac extremity of the stomach is thus involved. Sometimes such a tumor may be felt in the epigastrium; but in most cases there is nothing to indicate that the stomach is attacked.

The neighboring parts of the trachea or bronchi are sometimes affected, and important symptoms result from such a complication. If perforation occurs, an almost certain result is the inhalation of food or of decaying bits of the tumor, with consequent pulmonary gangrene, and, as a rule, speedy death. The disease has also been observed to attack the pleura, and end in perforation. The same is true of the pericardium and the aorta. A few instances are known in which the vertebræ have been involved, the spinal cord compressed, and paraplegia thus induced. We have ourselves seen one such case.

Quite frequently the recurrent nerve is affected, and a paralysis of the vocal cords is produced, which can be detected by the laryngoscope. This nerve lies so close to the œsophagus that it is peculiarly exposed to injury from the new growth itself, or from any inflammatory process which may be set up around it.

Metastatic cancer in distant organs is not infrequent, and may give rise to important symptoms. It attacks most frequently the liver. The lungs, kidneys, pancreas, bones, and brain are also liable to it.

Pulmonary gangrene must be mentioned as a relatively frequent complication, and one which has serious consequences. We have already stated that it may result from perforation. A still more frequent cause is the inspiration of decaying masses vomited or regurgitated by the patient.

Clinical History, Termination, Prognosis, and Treatment.—The disease is incurable. Operative removal has never been successful. The entire duration of the disease seldom exceeds a year, or a year and a half. At the end of this period the patient dies either from lack of nourishment or as a result of some one of the complications above enumerated. Treatment is purely symptomatic. Temporary improvement may be obtained by mechanical treatment of the stenosis. The particulars about this may be found in the preceding chapter.

CHAPTER V

RUPTURE OF THE ŒSOPHAGUS

Medical literature records a small number of cases which prove that the sudden rupture of the œsophagus in persons previously perfectly well is possible, although of course very rare. The first and most famous instance was described by Boerhaave in 1724.

The symptoms, according to the observations thus far reported, usually commence with sudden nausea and vomiting, during or shortly after a hearty meal. There is simultaneously an extreme, general collapse. There is pallor of the face and extremities, cold perspiration, and an extremely feeble pulse. Sometimes the patient feels a sudden darting pain in the chest. Almost invariably an extensive emphysema overspreads the neck and thorax. Death results in a few hours, or at latest in a few days.

The autopsy reveals a tear in the œsophagus, invariably situated in its lower half. It may be five centimetres long, and it is almost always longitudinal. Food has usually escaped into the surrounding tissues, in which case a secondary purulent inflammation exists, if death was not immediate.

Zenker has attempted to explain this remarkable phenomenon by a supposition which is really very plausible, namely, that œsophagomalacia always precedes these so-called spontaneous ruptures. The cause of this softening of the œsophageal walls is probably the action of gastric juice escaping into the tube and attacking a surface which, through some temporary disturbance in the circulation, has lost its normal powers of resistance.

CHAPTER VI

NEUROSES OF THE ŒSOPHAGUS

1. **Spasm of the Œsophagus.**—In rare instances œsophageal disturbances are observed, which appear to result from spasmodic contraction of its muscular coat. Nervous and hysterical subjects are particularly apt to present temporarily the symptoms of extreme stenosis, for which there can be no anatomical basis. Such cases are termed "spastic stenosis" of the œsophagus, or "œsophagismus." It is, of course, possible that there may exceptionally be some real lesion at the founda-

tion of the trouble, and that the spasm is the reflex result of an ulcer or inflammation affecting the œsophagus. It is even affirmed that the reflex influence may sometimes originate in distant organs, such as the uterus; but the exact nature of these reflex spasms is at present exceedingly obscure. The dysphagia is usually attended by a painful sense of constriction in the throat and chest. The bougie comes upon an obstruction, which usually soon yields. This circumstance, that when the spasm relaxes it is possible to introduce the bougie without any difficulty, confirms the diagnosis. Other important factors are, the character of the symptoms as a whole and the other attendant nervous and hysterical disturbances. Some authors also explain the "*globus hystericus*"—that feeling as if a lump were passing up or down in the throat and chest—as a spasm of the œsophagus.

2. **Paralysis of the Œsophagus.**—Of this subject we have little accurate knowledge. It is not improbable that an extensive bulbar paralysis, affecting the muscles of the pharynx and larynx, may sometimes involve the œsophagus; although such a disturbance hardly ever gives rise to prominent symptoms in this disease. Ziemssen asserts that sometimes the œsophagus seems to participate in post-diphtheritic paralysis, when extensive.

SECTION IV

DISEASES OF THE STOMACH

CHAPTER I

BRIEF PRELIMINARY REMARKS ON THE EXAMINATION OF THE GASTRIC CONTENTS *

WHEN a disease of the stomach exists, we have the subjective symptoms to consider, such as anorexia, eructations, vomiting, and gastric pain, together with the external physical signs obtained by inspection and palpation, such as tenderness, distention, peristaltic motion, swelling, and tumors; and we also have for our guidance an examination of the stomach and the stomach contents. The procedure was introduced by Leube, followed by Ewald, Boas, Riegel, and many others. It is carried out by means of the stomach-tube, and is now universally employed among physicians, for in fact its results are so important and decisive with regard to diagnosis, that an examination of this sort is at the present day indispensable in any severe case of persistent gastric trouble. The inconvenience of the investigation for the patient is comparatively slight, especially now that a soft and yielding tube (Nélaton) is employed. This should have free apertures at its lower extremity, and it is introduced into the stomach in the following manner: The tube is moistened with water, put into the patient's mouth, and passed backward over the base of the tongue, and he is told to swallow it, as it were; the physician at the same time aids by pushing the tube, but he does not introduce his finger into the patient's mouth. In this way it is almost always easy to introduce the instrument into the œsophagus, and so into the stomach. Most patients, if the tube has to be employed frequently, soon learn to guide it themselves. Formerly a wire was placed inside the tube so as to stiffen it for introduction, but this is ordinarily useless, if not a hindrance.

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* Complete particulars with regard to the methods of examining the gastric contents can not be given here, but they may be found in the following works: Ewald, Klinik der Verdauungskrankheiten; Boas, Diagnostik und Therapie der Magenkrankheiten; Leo, Diagnostik der Krankheiten der Verdauungsorgane; Riegel, Erkrankungen des Magens, etc.

If the tube has been introduced into the stomach, and we wish to obtain a portion of the contents of that organ for examination, it can, in most cases, be got by simple "expression." If the patient makes his abdominal muscles tense ("presses"), or if he makes a few slight efforts to vomit, there is usually a sufficient amount of the gastric contents expressed through the tube into a beaker held to receive it. This portion of the gastric contents is filtered, and the filtrate subjected to examination. If we wish to empty the stomach completely ("rinse it out"), the upper end of the inserted tube is connected by means of a short piece of glass tubing, with a longer rubber tube connected at its other end with a large glass funnel, to hold about a litre of water. If warm water is poured into the funnel and this is alternately raised and lowered, we shall eventually entirely empty the stomach (see Fig. 45). It is advisable to insert a short bit of glass tubing in the rubber tube (fenestra), so as to observe whether the fluid runs well in either direction. Kussmaul originally employed a "stomach-pump"

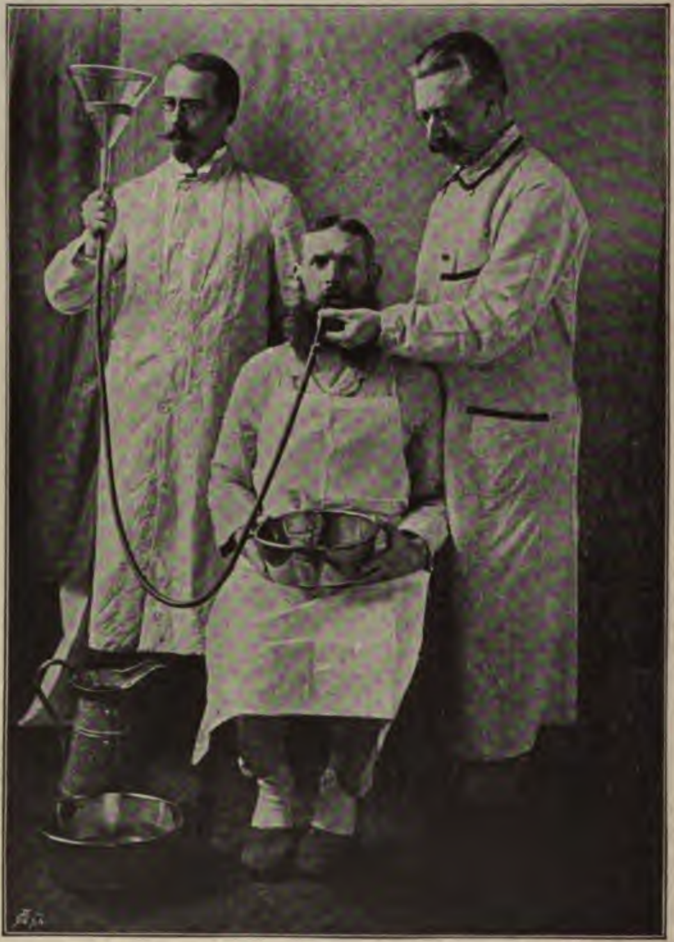

FIG. 45.—Method of washing out the stomach. (Erlangen Medical Clinique.)

for rinsing out the stomach, but this has been quite universally abandoned for the simple siphon apparatus suggested by Hegar.

By use of the stomach-tube we are readily enabled to settle the following important points: First, the chemical constituents of the gastric juice, or contents; second, the motor activity of the stomach; and third, the size and position of the organ.

1. **Constituents of the Gastric Juice, particularly Free Hydrochloric Acid, Pepsine, and Lactic Acid.**—In a healthy person the stomach is almost completely empty in the morning before breakfast—i. e., some ten or twelve hours after the preceding evening meal. If we introduce the stomach-tube we obtain, therefore, by expression either nothing at all or a very small amount of watery mucus, of neutral reaction. If this fluid contains a little hydrochloric acid, this is not necessarily anything abnormal, but if there is a large amount of acid fluid in the fasting state disease is indicated. The healthy stomach contains in the morning few vestiges of the food swallowed the day before. If we pour in some water, this is returned almost perfectly clear. If now the subject of examination eats a so-called test breakfast, consisting of an ordinary roll and a large cup of weak tea, at once the stomach begins to secrete its peculiar juice. If the stomach-tube is introduced an hour after a test breakfast has been taken, and a sufficient amount of the stomach contents is expressed and filtered, we shall obtain a fluid of acid reaction, and, under normal conditions, invariably containing free hydrochloric acid uncombined with albumen. It is the comparatively small amount of the test breakfast which makes it most suitable for the determination of the important question, whether the stomach secretes hydrochloric acid in a satisfactory manner. If we introduce into the stomach a larger amount of food ("test meal," *vide infra*) there will, indeed, in most cases, be free hydrochloric acid after one or two hours, but the amount of albuminous material present is so great that even under normal circumstances all the hydrochloric acid secreted is taken up by the albuminous substances, and consequently there is no hydrochloric acid to be discovered by our ordinary chemical tests. If, on the other hand, we do not find any free hydrochloric acid in the gastric contents, or, at any rate, merely doubtful traces of it, an hour after the test breakfast, this condition is termed anacidity, or subacidity, and it must be regarded as abnormal, particularly if repeated examination leads to the same result. The term acidity in this connection relates to hydrochloric acid. Even if this is absent it is perfectly possible that there may be other organic acids in the gastric contents.

The test for the presence of free hydrochloric acid now almost universally employed is one introduced by Günzburg, who recommended a solution of 2 parts of phloroglucine and 1 part of vanilline in 30 parts of absolute alcohol. If we mix a few drops of this phloroglucine-vanilline in a saucer with a few drops of the filtered gastric contents to be examined, and then heat the mixture cautiously so as to avoid charring, there will immediately develop on the edge of the fluid a beautiful red border, if free hydrochloric acid is present. Another good reagent for free hydrochloric acid is methyl violet. This requires an accurate comparison of colors, so that the weak watery solution of methyl violet is divided into two portions in test-tubes. If now we add to one test-tube some of the gastric contents containing free hydrochloric acid, the violet is immediately changed into a distinct blue color, which is evidently different from the original fluid contained in the other test-tube. For a third test what is called Congo paper is employed—i. e., strips of paper colored with an aqueous solution of Congo red. If the stomach contents contain free hydrochloric acid, the red color of the paper is changed to a distinct blue. There is a certain inaccuracy in this last-mentioned test with Congo red, as the red is colored blue by concentrated solutions of other acids, particularly lactic acid; but, as a matter of fact, the other acids are scarcely ever present in the stomach in sufficient amounts to invalidate the test; so that in actual practice, if the Congo paper changes to a distinct blue, we may almost always infer that free hydrochloric acid is present. Still, it is advisable in every case to carry out the two other tests also, which are almost as simple and entirely unambiguous. There is a reagent often employed which indicates the presence of free acids in general, including free lactic acid. It consists of an alcoholic (or aqueous) solution of "tropæoline OO." The yellow color of tropæoline is changed to a beautiful red by even small amounts of acid, but still more readily by hydrochloric acid than by the organic acids. Blue litmus paper, as is well known, is colored red by free acids as well as by acid salts. If the examination of the contents of the stomach an hour after the test breakfast shows the presence of free hydrochloric acid, an experienced observer will often be able to estimate from the degree of the qualitative reaction whether he is dealing with a small or considerable amount of hydrochloric acid. An accurate quantitative estimation is possible by titration of the stomach contents with a decinormal soda solution, for which a few drops of phenol-phthaleine solution serve as index.* The amount of acid determined by this process is of course the sum total of free acids, and not exclusively the free hydrochloric acid. Still, provided there is an abundant amount of hydrochloric acid in the gastric contents, the results obtained may be ascribed without great error to hydrochloric acid alone; for experience has shown that, when there is a considerable amount of hydrochloric acid in the gastric contents, there is scarcely ever any considerable amount of organic acids (*vide infra*). The degree of acidity of the gastric fluid is usually expressed in the number of cubic centimetres of soda solution which are necessary, as shown by the titration, to neutralize 100 cubic centimetres of the gastric fluid; for example, if we have required 6 cubic centimetres of soda solution to neutralize 10 cubic centimetres of the gastric contents, the acidity is reckoned at 60. In terms of hydrochloric acid we would have 60 multiplied by 3.65, equal to 19 milligrammes of hydrochloric acid—that is, 0.22 per cent. The degree of acidity which is found in the gastric contents of healthy persons after the test breakfast is usually 55–65; amounts of 70–80 indicate hyperacidity. If the gastric juice contains an abundance or an excess of hydrochloric acid, the further question, and often an important one, arises whether there is a hypersecretion, or, more correctly, a continuous secretion of gastric juice. As we have already mentioned, the normal stomach does not ordinarily secrete except when its mucous membrane is affected by food swallowed, or by other stimulating influences. The fasting stomach does not, as a rule, contain any hydrochloric acid, and when the stomach has passed the food on into the duodenum, its secretion immediately ceases. In order, then, to determine the existence of hypersecretion or continuous secretion, we must examine the contents of the fasting stomach at least six or seven hours after the last meal. If we then find fluid in the stomach, with distinct or abundant amounts of hydrochloric acid contained in it, we are justified in assuming that there is an abnormal hypersecretion on the part of the stomach.

Physiology teaches us that not only hydrochloric acid but pepsine in association with it, is necessary for the peptonizing of albuminoids. The demonstration of pepsine in the gastric contents is, therefore, essential to a determination of the peptic function of the stomach. Experience has shown, however, that pepsine is rarely absent from the gastric juice, and if there is a distinct secretion of hydrochloric acid we may almost always assume that pepsine is secreted, without making any special examination. Even in cases of anacidity pepsine often continues to be secreted. Still, there are cases when the direct demonstration of the presence or absence of pepsine in the gastric juice is desirable. The process is simple. We obtain some gastric contents in the ordinary way after a test breakfast, and put into it a square shaving of the white of a hard-boiled egg. Normal gastric juice containing hydrochloric acid and pepsine, if kept at a temperature of 98.6° (37° C.) in an incubator, will dissolve the flake of albumen completely in from thirty to sixty minutes. If the gastric juice contains no hydrochloric acid but only pepsine, the albumen will be dissolved if we add a few drops of dilute hydrochloric acid. If, however, pepsine also is absent from the gastric juice, the bit of albumen will swell up but will not be actually dissolved, unless we add artificial pepsine.

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* We scarcely need to describe this simple process more fully.

Besides hydrochloric acid there are also some organic acids in the gastric juice; the most important of these is lactic acid. At present the universal belief is that the lactic acid is not a product of the gastric mucous membrane, but the result of lactic-acid fermentation of the carbohydrates in the gastric contents, except for small amounts which may be directly introduced with the food. If we test the gastric contents a short time (fifteen to thirty minutes) after a test breakfast, we can often find some lactic acid present, but as yet no hydrochloric acid. This corresponds to the amylolytic stage of gastric digestion. Later, when the secretion of hydrochloric acid has begun, the lactic-acid fermentation promptly ceases, so that under normal conditions, as we have said, we find hydrochloric acid distinctly present an hour after the test breakfast, but usually no lactic acid. If we do find lactic acid it is always an indication of an impaired production of hydrochloric acid. There is a condition which is especially favorable for the formation and collection of large amounts of lactic acid in the stomach. This is when the absence of hydrochloric acid is associated with a stagnation of the ingesta in the stomach. This combination is especially frequent in the case of carcinoma of the pylorus (*q. v*). Under these conditions the lactic-acid fermentation may go on unchecked, and we find an abundance of that acid in the stomach at whatever time we make a test.

The qualitative test for lactic acid is by means of Uffelmann's reagent: by adding a drop of ferric-chloride solution to a three- or four-per-cent. solution of carbolic acid, we obtain a liquid of a beautiful steel-blue color. Upon adding to a portion of this blue fluid, contained in a test-tube, gastric contents in which there is lactic acid, the blue color changes to a distinct yellow, or yellowish-green. The test should not be regarded as positive unless there is a distinct yellow color. We may first agitate the gastric fluid with ether, and then make the test with the ether extract, as ether takes up the lactic acid. The quantitative estimation of lactic acid is tedious, and not very important in practice.

The fatty acids and acetic acid are not found in the gastric contents unless there is marked fermentation, and their presence therefore indicates a stagnation of the gastric contents (*vide infra*), and the absence of hydrochloric acid, which acts as an antiseptic. In practice we form an opinion as to the presence of these acids by the sense of smell, since their demonstration by direct chemical means is far from simple.

2. **Determination of the Motor Activity of the Stomach.**—The employment of the stomach-tube also enables us to form with ease a satisfactory estimate of the motor function of the stomach, and this is an extremely important matter. As physiology teaches us, the pylorus remains firmly closed during the first part of gastric digestion. It does not open until the food is sufficiently prepared, and then the chyme is discharged by successive jets into the duodenum. For practical purposes it is sufficient to know that the examination of healthy persons has shown the stomach to be completely empty again about two hours after the ingestion of a small amount of food—e. g., after the test breakfast (*vide supra*). If, then, we rinse out the stomach two hours after a test breakfast we

ought not, under ordinary circumstances, to find any large amount of bread in the wash water. But it is a comparatively easy task for the stomach to make away with the test breakfast; so it is more suitable, if we desire to test its motor powers, to administer what is called a *test meal*, consisting of a plate of broth, 150 grammes (5 ounces) of underdone broiled steak (minced), 50 grammes (2 ounces) of potato purée, and a roll. A meal of this sort disappears from the stomach often in three or four hours, but at the latest, under normal conditions, in seven hours. If we wash out the stomach seven hours after a test meal and still find any considerable amount of food, it is sure proof of an unsatisfactory discharge of the stomach contents, whether as a result of impaired motor power or, as is most frequently the case, of a mechanical stenosis of the pylorus. If we really wish to know how long portions may be retained in the stomach, we may add to the meal a few cranberries or green-colored beans, and the like. Such easily recognizable things may sometimes be found again in rinsing out the stomach several days after their ingestion.

It is of course understood that the gastric contents which we obtain a shorter or longer time after a test meal are examined with regard to the presence of hydrochloric and lactic acids, and also with regard to other characteristics, such as the presence of mucus and blood, and microscopic appearances. Many important details in regard to these matters will be brought up in later chapters.

In this connection, however, we may make brief mention of another method originated by Ewald, with this same object of determining the motor efficiency of the stomach. The patient is given, early in the morning, a gelatine capsule containing fifteen grains (gramme 1) of salol. Salol is not broken up until it reaches the intestine, when it separates into carbolic and salicylic acids. The salicylic acid is then immediately excreted in the urine, and readily detected by chloride of iron. If the stomach does not empty itself in a normal manner, the salicylic reaction is often to be detected in the urine even after twenty-four or thirty hours, at which time it would have vanished under normal conditions. This test has not been adopted very generally in practice, because it is uncertain.

3. **Estimation of the Size and Position of the Stomach.**—The position and size of the stomach are liable to great variations, and with regard to pathological conditions it is important to determine these points about the organ. An experienced eye may sometimes recognize the position of the stomach by mere observation of the abdomen, but often this is negative or misleading. By percussion we may distinguish the limits of the deep tympanitic resonance of the stomach from the usually higher tympanitic resonance of the surrounding intestines; or we may mark the lower limit of dullness when the organ is full, so that we can sometimes form a tolerably certain opinion as to the lower border of the stomach. In general, these results are deceptive and uncertain.

With the aid of the stomach-tube the matter is very simple. It is accomplished by blowing up the stomach with air, in the manner first elaborated by Runeberg. This is done by means of the ordinary double rubber bulb [such as is employed with the Paquelin cautery; or with a simple Davidson syringe]. The method used by us in our wards almost every day is as follows: The patient is either fasting or has had his stomach thoroughly rinsed out, and lies as nearly horizontal as possible upon his back. The stomach-tube, being introduced, is connected with the bulb at its upper end, and air is pumped in. As the pylorus is almost always firmly closed, the contours of the stomach become visible very shortly. We should then not only mark the position of the greater curvature but also that of the lesser curvature, if visible. If the stomach is normal in position and size, the protuberance occupies the epigastrium above the navel. If the stomach is dilated, the greater curvature reaches below the navel; while if the stomach as a whole is displaced downward (*vide infra*, gastroptosis), the smaller curvature also falls to the neighborhood of the navel or below it.

As soon as we disconnect the bulb the air escapes, and the distended stomach collapses. We can, if a certain degree of caution is used, repeat the procedure more than once, so as to make sure of our results. Of course, inflation should be interrupted immediately if the distention of the stomach becomes painful; and we should abstain from the procedure altogether whenever there is suspicion of an ulcer, because the artificial distention of the stomach in this case might do harm. In some cases, direct inflation does not render the contours of the stomach distinctly prominent, probably because of insufficiency of the pylorus. In such cases, and in such only, we should employ a rubber bag (condom) fastened on the lower end of the tube, having previously tested its distensibility. When this is introduced into the empty stomach and blown up, we can usually discern it through the abdominal walls and determine the position, but of course not the size, of the stomach. For ordinary cases the first-described method is not only simpler but more satisfactory, and, so far as the patient is concerned, less disagreeable. If the abdominal walls are thick with fat the method fails, but such a condition is very rare when there is severe gastric disease.

The simple method just described seems to us to have rendered entirely superfluous most of the other more or less complicated procedures for the determination of the size and position of the stomach, especially as no better results are obtained by them. This last statement applies especially to electric transillumination of the stomach and anterior abdominal walls by means of an incandescent light introduced with a tube. A practiced specialist can obtain valuable results in this way, but it is often very misleading.

In ordinary practice where the introduction of the stomach-tube is not always practicable, the old method recommended by Frerich may be employed. For carrying this out it is best to have the patient fasting, and in a horizontal position. Seventy-five to one hundred and fifty grains (grammes 5–10) of tartaric acid are given, and immediately after an equal amount of bicarbonate of soda, each drug being dissolved in half a glass of water. At once there is an abundant production of carbonic dioxide. The stomach is blown up and its outlines become sometimes very distinct, both for observation and for percussion. The great advantage of the previously-described method of pumping in air consists, however, in the fact that we have the degree of distention under far better control, and that we can repeat the process several times in succession.

To sum up briefly all that has been said, a careful examination of the stomach should proceed in the following manner: First, rinsing out of the empty stomach early in the morning, to determine whether there may be stagnation (as indicated by lactic acid) or hypersecretion. We should also notice whether there is any collection of mucus in the stomach when fasting; and we determine the position and size of the stomach by inflation. Secondly, the stomach being empty we administer a test breakfast; an hour later we examine the contents for hydrochloric or lactic acid. Thirdly, at noon we give a test meal, and seven hours later wash out the stomach, to determine its motor efficiency. If there is stagnation of the gastric contents, we test again for hydrochloric and lactic acids.

4. **Testing the Absorptive Powers of the Stomach.**—Brief mention should be made of Penzoldt's method of determining the absorptive powers of the stomach. A gelatine capsule filled with iodide of potassium, if swallowed by a healthy person fasting, will give rise at the end of ten or fifteen minutes to a reaction for iodine in the saliva and in the urine (by adding sulphuric acid and shaking with sulphide of carbon). In severe diseases of the stomach the time required is often much longer. In practice, however, the method has not been highly approved,

because we can not draw any certain conclusions from it as to the absorption of food—a matter about which, in general, we possess little knowledge.

5. **Demonstration of Blood in the Gastric Contents.**—Any admixture of blood with the contents of the stomach is of great importance in the diagnosis of certain gastric diseases, particularly ulcer and carcinoma. If there is fresh blood mixed with the vomitus or with the gastric contents obtained by washing out the stomach, it is often directly recognizable from its characteristic appearance, but if the blood is already decomposed or intimately mixed with the food, or present in very small amounts, we need special methods for its certain recognition. Microscopic examination alone is insufficient, for the red corpuscles are soon destroyed in the stomach. The method of spectrum analysis requires a special spectroscope. We search for the characteristic bands of hæmatine in an ethereal extract, made by shaking up a portion of the gastric contents, to which a few drops of glacial-acetic acid have been added, with ether. For medical practice the most useful and simple method is Van Deen's test, with fresh tincture of guaiac and turpentine. A mixture of equal portions of these two liquids is poured into a test-tube upon a portion of the gastric contents. If blood is present there at once appears an intense blue color, but certain other matters contained in the food may cause this same color reaction, so that it is more satisfactory to decompose the gastric contents with a few drops of glacial-acetic acid, then extract with ether and perform the guaiac-turpentine test with the ether extract. This test is especially distinct if there are organic acids present, as in case of carcinoma, in which disease the stomach contains blood in association with lactic acid but no hydrochloric acid. On the other hand, in our experience, the test is often uncertain in the case of ulcer, because the presence of hydrochloric acid is a hindrance. Another common method is to see if we can produce Teichmann's hæmine crystals from the gastric contents. The test is not always successful, but if successful it indicates the presence of blood. We put a small portion of the suspected gastric contents upon an object-glass with a trace of common salt, and a few drops of glacial-acetic acid. If we now evaporate slowly to dryness there are formed small, brown, rhombic hæmine crystals (see Fig. 46), which can be seen by the microscope. Of course, whenever we examine the contents of the stomach for blood, we should bear in mind that, if found, the blood may be due to the ingestion of underdone meat, preparations of hæmoglobin, or similar substances. Finally, we scarcely need to repeat that in all important cases one ought not to be satisfied with a single examination of the stomach, but he should make certain of his results by repeated tests.

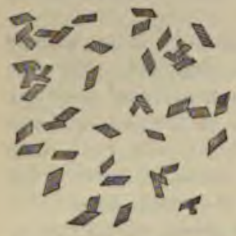
FIG. 46.—Hæmine crystals.

CHAPTER II

ACUTE GASTRIC CATARRH

(Acute Gastritis)

Ætiology and Pathological Anatomy.—The mucous membrane of the stomach not being open to direct examination,* as is that of the mouth and throat, and acute gastric disease being seldom fatal so as to render an autopsy possible, the existence of acute gastritis is mainly a matter of inference from our observations of other mucous membranes. When some harmful agency acts in a direct manner upon the mucous membrane of the stomach, we shall usually be right in supposing that there is a greater or less degree of genuine inflammation of the lining of the stomach, unless the organ in a simply functional way at once rids itself of the irritant—for example, by vomiting when the stomach has been "overloaded." If some noxious material acts long and continuously upon the mucous membrane, there will be abnormal changes not only in the blood-vessels and the interstitial tissue, but also in the cellular elements of the mucous membrane and its glands. As yet, we know little about the minute degenerative changes in the specific secreting cells. The microscopic inflammatory lesions, which we may with great certainty assume to be present, are hyperæmia and swelling of the mucous membrane, perhaps associated with an increase in the secretion of mucus, and with small hæmorrhages here and there. If the irritant is insignificant the lesions will be mild and superficial (mild catarrhal gastritis). If the noxious agent is more powerful (for example, corrosive poison) it will give rise to a deeper parenchymatous inflammation, with peeling off of the mucous membrane and similar effects (severe toxic gastritis).

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* The examination of the mucous membrane of the stomach by means of the stomach mirror and the electric light involves such technical difficulties that it has not as yet been adopted in practice.

The mild catarrhal forms of gastritis are caused by simple chemical and mechanical influences, and perhaps by thermic irritation. They are most often the result of errors in diet, such as the ingestion of too large an amount of food, or of food that is difficult to digest, unsuitable, highly spiced, or very acid. In the same class belong the acute indigestion following excess in alcohol, and the frequent derangement of the stomach from taking medicines; and also cases due to the accidental or willful ingestion of all sorts of injurious and poisonous substances. The severe cases of toxic gastritis are most often caused by the action of concentrated mineral acids and alkalies. In this class comes poisoning from sulphuric acid, nitric acid, hydrochloric acid, caustic potash, and caustic soda.

A special importance attaches to the ingestion of decaying substances. The incautious use of tainted meat or fish may be followed by relatively severe forms of acute gastric catarrh. The products of decomposition act as chemical irritants upon the mucous membrane; and the ferments and putrefactive agents likewise continue in activity after reaching the stomach, and thus contribute to produce the inflammation. The reason that this sort of gastric catarrh is not much more frequent than it is, is undoubtedly the presence of hydrochloric acid as a constituent of the gastric juice, because it has an antiseptic action.

It is universally assumed that a chill of the outer surface of the body may excite gastric catarrh, but there seems to be actual proof of this in but few cases. On the other hand, there can be no doubt that many cases of acute gastric catarrh, of apparently primary origin, are referable to infection. Infectious catarrh of the stomach may occur at times, particularly in summer, with especial frequency. As to the precise nature of the pathogenic germ we do not as yet possess any certain knowledge.

The predisposition of certain individuals to gastric catarrh differs greatly. Some persons always have a "weak" stomach, and the disease is prone to attack feeble children, anæmic persons, fever patients, and convalescents from severe diseases, as well as chronic invalids who are ill-nourished. Enfeebled persons of this sort sometimes fall sick when vigorous and healthy individuals would entirely escape. In many of these cases of unusual predisposition to gastric disease, we may surmise that the secretion of hydrochloric acid is scanty, and that there may be a diminution in the motor power of the stomach. Each of these factors would of course render ingested irritants more harmful.

Symptoms.—The most constant subjective symptom is anorexia. In many cases the very thought of food excites disgust. What the patient does eat tastes flat, and he is therefore very eager for piquant dishes, highly spiced or sour. Thirst is often present, and a feeling of dryness in the mouth.

The subjective gastric sensations are seldom those of marked pain; but sometimes there are attacks of pain which are in all probability due to cramplike contractions of the muscular coat. The usual complaint is of constant pressure and fullness. Sometimes the patient is conscious of the peristaltic movements of the stomach. He has "rumbling" of the bowels.

There is nausea, and often vomiting. In severe cases everything swallowed is at once rejected. The vomitus consists for the most part of undigested food (often ill-smelling), with which mucus, and sometimes bile, is mingled. Eructations of gas or liquid are frequent.

Physical external examination reveals little. The epigastrium may be somewhat prominent as a whole, and may be tender on pressure. The tongue is almost always thickly coated and dry. The breath is usually disagreeable, and there is a persistent flat or bitter taste in the mouth. In ordinary cases of acute gastritis examination of the gastric contents is hardly necessary, but in severe cases we find a marked diminution in, or entire absence of, the hydrochloric-acid reaction, and distinct delay in the motor activity of the stomach. Sometimes lactic acid and the fatty acids are present in large amounts.

In severe cases there is always considerable constitutional disturbance. The patient feels languid, and disinclined for any exertion; the pulse is moderately rapid, although it may exceptionally be slower than normal; the urine is usually somewhat concentrated, often it gives a marked indican reaction. There may be no fever, but not infrequently there is a moderate elevation of temperature with sensations of chilliness or heat. In rare cases we may observe an approach to the typhoid condition, with such nervous symptoms as headache, vertigo, and dullness. These cases were formerly termed "gastric fever," and they are probably most of them infectious, but yet the constitutional symptoms are probably only in part referable to coincident constitutional infection. It is more probable that toxic influences are exerted by the abnormal products of the fermentation which takes place in the stomach. For example, Senator mentions sulphuretted hydrogen as thus generated; and Litten has described several cases in which at first there were such dyspeptic symptoms as nausea, vomiting, flatulence, and a coated tongue, but which soon gave evidence, by restlessness, headache, great muscular weakness, and a gradual lapse into somnolence, of rather severe nervous disturbance in addition. The breath had a marked "fruity" odor; and, on adding chloride of iron to the urine, a strong reddish color was developed, as in the so-called acetone-reaction; so that it seems probable that an auto-intoxication had occurred, somewhat resembling diabetic coma.

Chief among complications are the intestinal symptoms, which are frequently coincident with the gastric disorder. Constipation is the rule. There may be diarrhœa. The gastric catarrh may by extension involve the duodenum, and give rise to jaundice. Sometimes herpes appears upon the skin. This fact argues for the infectious nature of many cases of gastric catarrh.

It is evident from what has been said that acute gastric catarrh is by no means uniform in its ætiology, and the general course of the disease is also subject to great variations. Sometimes there are mild dyspeptic symptoms which vanish in the brief time of one or two days, while in other cases we have a rather severe disease associated with considerable constitutional disturbance, and lasting three to ten days. There is also great variation in the intensity of the various symptoms, particularly the vomiting. The progress of the case may be irregular and there may be relapses, but still, the prognosis of primary acute gastritis is entirely favorable. The diagnosis is usually easy, although we should never omit to make an unprejudiced and careful examination into the general condition.

If there is fever we should bear in mind the possibility of a mild typhoid (see typhoid fever).

Treatment.—If, at the beginning of the disease, there is reason to suppose that the stomach is loaded with undigested food, an emetic is indicated. If it is desired to avoid the irritative action of an emetic upon the gastric mucous membrane, a subcutaneous injection of one sixth of a grain (gramme 0.01) of apomorphine may be given. Still more efficient, although disagreeable for the patient, is a washing out of the stomach by means of the stomach-tube, particularly in severe cases of toxic or infectious origin; but of course, when the gastric walls may have been weakened by corrosive poisons, the stomach-tube is contraindicated.

In most cases, however, emetics and lavage may be dispensed with. The treatment in such cases consists mainly in a strict regulation of the diet, allowing for a time nothing whatever, and then such food as milk porridge, iced milk, and toast. Of internal remedies, ten to fifteen drops of dilute hydrochloric acid in half a wine-glass of water sometimes seems beneficial; while in other cases, in which there are sour eructations and vomiting, alkalies are to be given. We may order as much bicarbonate of soda or as much Carlsbad salts as will rest on the point of a pen-knife, or Ems water. The so-called stomachics and bitters are also often prescribed, for example, compound tincture of gentian or tincture of rhubarb. Another favorite remedy is resorcine, of which four grains dissolved in some aromatic water may be given every two hours. If the vomiting is obstinate, relief may be got from bits of ice or sips of cold Seltzer water. In severer cases we must resort to narcotics, such as opium, cocaine, and chloroform, administered internally.

If there is decided constipation, an enema must be employed, or such laxatives as calomel, Epsom salts, or rhubarb.

CHAPTER III

CHRONIC GASTRITIS—CHRONIC CATARRH OF THE STOMACH

Ætiology.—The same causes which excite acute gastric catarrh, if often repeated, lead at last to chronic catarrh of the stomach; but, at least in adults, infectious influences are seldom prominent in the causation of the disease. In many cases it is due chiefly to the chemical and mechanical irritation of an unsuitable diet long continued. By far the most frequent—indeed, to speak more correctly, the only frequent—form of chronic gastric catarrh in adults, is due to hard drinking. The habitual use of distilled liquors is more influential in this regard than excess in wine or beer. The harm done by alcohol may be re-enforced by other dietetic causes. We have often met with genuine chronic gastritis in extremely destitute persons, who for a long time had to eat food that was insufficient, bad, or even decayed. It should also be pointed out that abnormal conditions of the mouth, and particularly dirty or carious teeth, may occasion dyspeptic and catarrhal conditions. Habitual excess in smoking is also said sometimes to occasion chronic gastritis; and finally, it is probable that the bad habit of rapid eating with imperfect mastication leads at last to gastric disturbance. As is the case with all external irritants of this sort, the results are influenced by individual differences in the resisting powers of the affected organ, and personal predisposition to disease plays a certain part in the development of chronic gastric catarrh. Often, as we have already noted, this predisposition seems, to a certain extent, a matter of family and heredity.

Chronic gastric catarrh is not always a primary affection. It may be secondary to some other disease. In particular, all diseases associated with portal congestion are apt to lead to secondary catarrh of the stomach. This includes hepatic cirrhosis and hepatic syphilis. Again, the gastric catarrh associated with chronic cardiac, pulmonary, or renal disease is in part to be regarded as due to congestion. On the other hand, however, we should always consider how manifold are the other conditions, associated with all sorts of chronic diseases, which may lead to chronic gastric disturbance. We may mention, for instance, anæmia, muscular insufficiency, and auto-intoxication.

Pathology.—The macroscopic changes in the gastric mucous membrane are in most cases very moderate. Usually it is thickened and coated with a layer of tough, grayish-white mucus, in which is suspended a greater or less amount of detached epithelium. The membrane is brownish red, unless rendered gray by excessive pigmentation. Under the microscope we find a well-marked infiltration of the interstitial tissue with fine cells, and well-marked parenchymatous changes, particularly extensive degeneration of the glandular cells. All these changes are especially marked in the pyloric portion of the stomach. If the catarrh has lasted a long time there may be still further changes in the mucous membrane. In many cases it appears smooth and atrophic. The glandular layer of the stomach may at last be almost destroyed, and the muscular and submucous coats share in the atrophy, while the organ as a whole is usually dilated. In other less frequent cases there is a marked growth of interstitial connective tissue, with resultant contraction. In this variety, also, the glandular layer atrophies, the stomach walls grow firmer, and the whole organ shrivels (cirrhosis of the stomach). Contrasting with these forms which lead to atrophy or sclerosis, there are others which occasion a hyperplasia of the mucous membrane. Its inner layer is thickened and mammillated (*état mamelonné*), and there may be actual polypi formed. The hyperplasia in these cases affects mainly the gland-tubes of the mucous membrane, but there is also a considerable thickening of the submucous coat.

Symptoms on the Part of the Stomach.—The symptoms of chronic gastric catarrh are the same dyspeptic symptoms which we meet in various combination and degree in all diseases of the stomach. They include disturbance of the appetite; pressure or pain in the region of the stomach; a bad taste in the mouth; abnormal sensations in the throat; and finally, motor phenomena, including rumbling, eructations, and vomiting.

The appetite is usually impaired in chronic gastric catarrh. There is sometimes a moderate appetite, but it is soon changed to a feeling of repletion upon the ingestion of even a slight amount of food. In other cases there is actual dislike for any form of nourishment; the patient eats little, and prefers highly spiced, piquant dishes. There is often a persistent bitter, flat, offensive, or otherwise abnormal taste in the mouth.

Subjective sensations in the region of the stomach are rarely entirely absent. As a rule, there is a feeling of fullness or pressure and of dull pain. These troubles may either be constant, or occur after meals. Sometimes, especially after errors in diet, there may be actual pain. This is termed cardialgia or gastralgia, and is probably due in most cases to cramp-like contractions of the muscles; but in general, habitual and severe pain does not belong to the clinical picture of simple catarrh. A very frequent and annoying symptom, which is particularly apt to come on after eating, is the eructation of gas, usually air, but sometimes gases which are formed in the stomach by the processes of decomposition. Such gases have a disagreeable odor. The eructations may bring up, also, some of the fluid contents of the stomach. If there is a bitter taste associated with it this is usually occasioned by peptones, but sometimes perhaps by bile (*vide infra*).

Acid eructations indicate excessive acidity of the gastric contents, ordinarily due to hydrochloric acid but exceptionally to lactic acid (*vide infra*), or to the fatty acids, which are recognizable by their odor. The burning feeling in the pharynx and lower down, which is occasioned by acid eructations and which is usually termed heartburn, is almost always caused by an excess of hydrochloric acid in the gastric contents, and consequently is not a frequent symptom of genuine chronic gastric catarrh (*vide infra*).

In many cases the feeling of nausea increases, especially after eating, to actual vomiting, but in chronic gastric catarrh vomiting is not very frequent, and if it occurs it is the result of some special cause, such as errors in diet. There is this exception, that in the chronic gastric catarrh of drunkards a peculiar form of vomiting is very common. This is called morning vomiting. By this is understood a fairly regular occurrence of vomiting in the morning before breakfast, with the ejection of considerable amounts of watery mucus, usually alkaline. This vomiting is due not only to the chronic catarrh of the stomach, but also to the chronic catarrh of the pharynx, which latter occasions a great tendency to gagging—e. g., when rinsing out the mouth. The vomitus consists not only of mucus from the stomach but in large part, also, of a mixture of saliva and mucus from the mouth and throat. In the other forms of chronic gastric catarrh the vomitus consists often of ill-digested food and more or less mucus. If there are vigorous efforts at vomiting and retching, the vomitus may contain also some bile and small amounts of blood, which have no serious significance.

The external examination of a patient with chronic gastritis shows little unusual. The general nutrition, of course, is often impaired, although in drunkards and gourmands an abundant fat layer persists for a long time. The tongue is usually coated, especially in its center, while its sides and tip are red. A thickly coated tongue and salivation are both of them due rather to accompanying stomatitis and pharyngitis—complications which are present in the case of most drunkards, and sometimes also in excessive smokers. Examination of the epigastrium shows often a considerable prominence of the stomach and tenderness on pressure, but the only means of obtaining an actual insight into the abnormal process is a careful investigation, by means of the stomach-tube. This should not be omitted in any case of chronic gastritis of any severity, especially as the results of it furnish the only true guide for a rational treatment of the disease. If we examine the gastric juice after the manner already described, we shall find that free hydrochloric acid in chronic gastritis is scanty (below 0.1 per cent.), or even absent. Its complete absence is particularly common in chronic alcoholic catarrh, and also in cases of chronic gastritis in which there is atrophy of the mucous membrane. In the latter case pepsine, also, is often absent. With regard to abnormal acids, we may find lactic acid, but seldom in large amounts, because in simple chronic catarrh there is scarcely ever much stagnation of the gastric contents. If there are abnormal processes of decomposition, we find acetic acid, butyric acid, and volatile fatty acids, but these are very rarely present.

The demonstration of an excessive production of mucus in the stomach is of great diagnostic importance. We have mentioned that sometimes the vomitus contains a distinct admixture of mucus, but the only way of forming an accurate opinion with regard to the amount of mucus in catarrh of the stomach is by siphonage, especially in cases in which vomiting is absent or infrequent. If we examine the stomach when fasting we shall, in many cases, find at every trial an abundance of mucous fluid with scarcely any other ingredients. In other cases there is more apt to be an abundance of mucus after a meal—either in a viscid, ropy fluid, or in separate transparent masses of varying size. Excess of mucus in the stomach is usually associated with anacidity, but there are a few exceptional cases in which it is associated with increased secretion of hydrochloric acid (gastritis acida; acid catarrh of the stomach. Boas, Riegel, and others). We must avoid confounding secretion from the mouth and pharynx that has been swallowed with mucus produced in the stomach. The latter is intimately mixed with the food, if there be any.

The motor function of the stomach is seldom much disturbed in chronic gastritis. Often it is surprising how rapidly the stomach empties itself; but there may be delay, partly because digestion is impaired and partly because there is a gradual development of weakness and atrophy of the muscular coat of the stomach, analogous to the weakness of the muscles of the vocal cords in chronic laryngitis. If food is long retained in the stomach, there is probably some other disease than simple catarrh. The size of the stomach in chronic catarrh may be somewhat increased, but this has no special importance.

Symptoms on the Part of other Organs.—Course of the Disease.—Of the other organs, the intestine most frequently suffers in chronic gastric catarrh. In almost all cases of chronic gastric catarrh the bowels are irregular. Habitual constipation is the rule, but sometimes there is diarrhœa. If much gas is generated in the stomach, the intestinal canal often becomes implicated, and tympanites and flatulence develop. There may be catarrh of the duodenum leading to jaundice.

The urine is often only feebly acid in reaction, and therefore frequently deposits a large amount of phosphates. We must be cautious, however, in the interpretation of this sign and the value we lay upon it, since the reaction of the urine is influenced by a great variety of conditions, including food, drugs, and the loss of acid by vomiting.

It is often asserted that chronic cutaneous diseases and, in particular, certain forms of eczema, are due to catarrh of the stomach, but this is not certain, though not impossible.

There has been great stress laid, particularly in recent times, upon the frequent occurrence of nervous disturbance in association with chronic catarrh of the stomach. This takes the form of hypochondriasis and depression of spirits in association with all sorts of other nervous symptoms, including pressure in the head, headache, dullness of mind, and dizziness (*vertigo e stomacho laeso*); but, while we may admit that the disease of an organ which, for many human beings, is the main source of happiness, may well put the patient out of sorts, yet there is no doubt that many earlier reports about nervous disturbances were due to a confusion of chronic gastric catarrh with nervous dyspepsia (*vide infra*). Genuine chronic catarrh of the stomach, as such, has no peculiar relation to nervous disease, except in the rare cases in which certain unusual symptoms develop, not improbably from the absorption of toxines.

When the catarrh is of any duration and severity, the general nutrition is apt to be seriously impaired. The diminished appetite and the imperfect digestion and absorption of what is eaten contribute to produce a gradual and considerable loss of weight. The fatty and muscular tissues atrophy. The skin grows dry and harsh, and usually has a dirty-pale color. In rare cases of extreme atrophy of the mucous membrane (probably almost always combined with atrophy of the intestinal mucous membrane) there are developed symptoms like those of progressive pernicious anæmia (*q. v.*).

Individual cases differ greatly in the combination of symptoms they present and in their course. The anorexia, gastric oppression, eructations, vomiting, and other important disturbances already mentioned, exhibit the greatest diversity in their intensity and their grouping. In the milder cases, loss of appetite and moderate local uneasiness may be the only symptoms. Frequent vomiting is confined to the severer cases. The disease often lasts for years, especially if the patient neglects himself. In most cases there are frequent remissions and exacerbations, usually dependent upon external causes.

The disease is not intrinsically fatal except in the above-mentioned rare cases of complete atrophy of the mucous membrane, but the general debility consequent upon it may indirectly shorten life.

Diagnosis.—If we except the self-evident cases of chronic alcoholic catarrh, the only proper way of making a diagnosis of chronic gastritis is by means of a careful examination with the stomach-tube, along with a consideration of the associated conditions. If a patient has for some time been suffering from the ordinary gastric symptoms of anorexia, gastric oppression, eructations, and vomiting, we must first determine whether we have to deal with a primary disease of the stomach, or with a secondary disturbance referable to such conditions as cardiac disease, renal disease, or pulmonary tuberculosis. If the indigestion is not symptomatic we must then consider in order each of the primary diseases of the stomach. If there is no indication of ulcer or carcinoma, we have remaining actual gastritis, nervous dyspepsia (*q. v*), and a dislocation of the organ (gastroptosis). One important factor is the general condition of the patient—for instance, if there is neurasthenia or the like. No actual conclusion can be reached, however, except by examining the contents of the stomach. We can not assume that there is a chronic gastritis unless there are direct objective changes to be demonstrated, chief among which is an abnormal production of mucus, associated with a change in the amount of hydrochloric acid in the gastric juice. This change is in most cases a diminution, but exceptionally an increase of the amount secreted. We wish, in this connection, to emphasize once more the statement that genuine chronic gastritis, except in drunkards, is by no means a common disease and is much less frequent than nervous dyspepsia.

Treatment.—If the disease seems to be merely symptomatic—the result, for instance, of venous stasis due to chronic cardiac, pulmonary, or hepatic disease—our efforts must, of course, be directed chiefly to the relief of the original trouble. We must also take into consideration all other causative influences, such as neglected or decayed teeth, improper mode of life, and unhealthy occupation.

The direct treatment of chronic gastric catarrh must always begin with a regulation of the diet. Such vague injunctions as " to be cautious " or " to avoid indigestible articles of food " are useless. The patient must have a perfectly definite bill of fare prescribed for him; nor can any universal one, suitable for all cases, be drawn up. In each individual instance the individual circumstances must be considered. The personal experiences of the patient himself are by no means to be disregarded. One man may be quite unable to digest what is well borne by others, and *vice versa.*

In the first place, certain foods must be utterly forbidden to such patients as do not themselves avoid whatever disagrees with them. All articles must be prohibited which may irritate the mucous membrane, either mechanically or chemically. This includes all the coarser sorts of vegetables or fruits, containing a large proportion of indigestible cellulose; and all dishes that are very sour, strongly salted, or highly spiced. Potatoes, farinaceous food, and all substances composed mainly of hydrocarbons, must also be interdicted; because almost all the abnormal fermentative processes, the evil consequences of which have already been considered, are promoted by the hydrocarbons. Fat is also harmful. It impedes digestion by protecting the contents of the stomach from the action of the gastric juice, in a purely mechanical way; and then, being changed into the fat acids, it causes sour eructations and pyrosis. Yet we must not go too far in forbidding starches and fats. Particularly if the patient is emaciated it is advisable to give such fatty substances as good butter and cream in cautious amounts, and often the result is satisfactory. In general, we should not forget that with regard to diet the skill of the physician often lies more in what he permits than in what he forbids. The limitation or withholding of alcoholic beverages is an important point. As we have already mentioned, the great majority of cases of chronic catarrh are the direct result of excess in alcohol—the most important factors being chemical irritation and mechanical distention of the mucous membrane. Moreover, the experiments of Fleischer and others have shown that alcohol prolongs and hinders the process of digestion. In every severe case it is better to forbid alcoholic beverages entirely, and ordinarily such an injunction is much better obeyed than advice to use them sparingly. In mild cases we admit that small portions of beer and wine may be permitted unhesitatingly, especially if the patient himself experiences no feeling of discomfort from them, but rather an improvement in his appetite.

In determining what the patient may be allowed to eat, we are to consider, as already mentioned, his own personal experience as well as our more general knowledge. An intelligent patient will often be himself the best judge of what agrees or disagrees with him. The following foods are very easy to digest: milk, soft-boiled or raw eggs, broths (especially veal and chicken broths), and certain artificial preparations, chief among which stand the Leube-Rosenthal meat solution and the meat juice and peptones which have been lately put upon the market. It must be confessed that patients soon tire of these last. The brain and sweetbread of calves are easily digestible; also birds, such as pigeons, chickens, and partridges, thin shavings of raw beef or raw ham, rice, potato purée, etc. Gradually we may proceed to somewhat heartier food—veal, game, roast beef, trout, and light farinaceous dishes. The worse the symptoms are in any case, the more strict must we be in regard to diet. For drink, besides water or Seltzer water, very weak tea, cocoa, chocolate, and water reddened with claret are allowable.—Shall we permit coffee? This is often a question of great interest to the patient: it must be answered according to his individual experience. Coarse bread is to be forbidden. Ordinary white bread, toasted, if it seems desirable, may be allowed in moderate amount, also rusks.

Solid articles of diet must be finely cut up and well chewed before being swallowed. The food must not be very hot or very cold. It is sometimes advantageous to take more than three meals a day, each one being proportionally smaller. Other patients relish their food better if the intervals between eating are prolonged. It should be added that excessive smoking, also, is harmful in chronic dyspepsia.

There are other special indications to be met. In all severe cases by far the most efficient mode of treatment consists in methodical washing out of the stomach. This is especially true when there is excessive production of mucus, or a tendency to decomposition of food in the stomach. In this way we prevent the accumulation of any great amount of undigested food. We remove the excreted mucus and the products of abnormal fermentation and decomposition, and moreover, we may perhaps exert a direct and favorable influence upon the mucous membrane. In such cases, we are accustomed to rinse out the stomach daily, and the best time for this is in the morning before the patient has had breakfast. Fresh fluid must be poured in and emptied out rapidly, until the stomach is freed from all foreign material, especially mucus. We use for the rinsing fluid a weak solution of hydrochloric acid (one to two per mille), or a one-per-cent. solution of either common salt or bicarbonate of soda; and, if there is much mucus, a favorite remedy is very dilute lime-water, two to four tablespoonfuls of liquor calcis to a litre (quart) of water. If fermentation is going on, we employ weak solutions of hydrochloric, boric, or salicylic acids, or resorcine. It has also been recommended to spray or douche the mucous membrane of the stomach, for which purpose stomach-tubes are employed with numerous small openings, and such fluids are chosen as weak solutions of alum, tannin, and condurango.

If we now turn to a consideration of the internal remedies used in chronic gastric catarrh, it is possible to decide with regard to their employment in a much more intelligent fashion than heretofore, because in all severer cases we make a more accurate investigation of the digestive process. If the examination of the gastric juice shows a diminution of hydrochloric acid, this may be artificially supplied. We prescribe, thirty to sixty minutes after each meal, ten to fifteen drops of dilute hydrochloric acid in half a glass of water. The benefit to be thus derived must not be overestimated. In appropriate cases, however, its influence is favorable. If there is no pepsine in the gastric juice we may prescribe pepsine as well as hydrochloric acid. Pancreatine and papaine are also recommended, but their usefulness is very doubtful. The popular wines of pepsine are not to be recommended, because of the alcohol in them.

Alkalies have long been employed as well as hydrochloric acid, and often with good results, although the most common form of chronic gastric catarrh has a less than normal secretion of hydrochloric acid, and consequently the use of alkalies would seem to be contra-indicated; yet experience shows them to be useful. Probably their beneficial effect is that they neutralize the abnormal acids, promote the emptying of the stomach into the duodenum, dissolve the mucus, and finally stimulate the secretion of the gastric juice. This has been experimentally demonstrated with regard to carbonate of soda, common salt, and carbonic acid. From what has been said it is evident that in chronic gastric catarrh alkalies are not usually given during digestion, but when the stomach is empty and before meals. As is well known, the alkalies are prescribed more especially in the form of alkaline mineral waters, which the patient drinks at home, or at the various health-resorts. The springs of Carlsbad possess the greatest reputation. Those at Ems, Kissingen, Tarasp, and Vichy also deserve to be mentioned as health-resorts for patients with gastric trouble. A good portion of the benefit at these places is, to be sure, dependent upon the fact that many patients are much more apt to follow a strict regimen when they make use of a particular "cure" than when they remain at home. It is self-evident that large doses of bicarbonate of soda are indicated in that form of chronic gastritis which is associated with hypersecretion.

If the character of the vomitus, or gastric contents obtained by rinsing, indicates abnormal fermentation, we may not only wash out the stomach (*vide supra*), but also try certain antifermentative remedies. First among these is hydrochloric acid in rather large doses, and we also give salicylic acid (in powders, containing 8 grains, gramme 0.5); creasote (two or three half-grain pills daily, gramme 0.03); benzine (20 drops in capsules or in milk); and similar drugs. As a rule, however, the last-named remedies are seldom employed.

The best drugs to stimulate the secretion of gastric juice are the bitters. It is this property which has earned them the name of stomachic tonics. Compound tincture of gentian, tincture of nux vomica, tinctura amara, and tinctura calami, P. G., quassia and columbo—these are much employed. In general, however, they are not very efficient. An excellent tonic in many of these cases is condurango bark. A decoction may be made (15 parts to 200 of water); or we may employ the fluid extract, giving a teaspoonful in water two or three times a day.

A few remedies remain to be mentioned, which are said to exert a direct beneficial influence upon the catarrh, and which many physicians extol highly. Their efficacy, however, is somewhat problematical. We refer chiefly to subnitrate of bismuth, sulphate of zinc, and nitrate of silver.

Certain symptoms may demand especial treatment, such as vomiting. It will usually yield to regular and persistent washing out of the stomach. Other remedies are small bits of ice, and minute doses of opium or chloral. Potassic bromide and chloroform, internally administered, may also be tried.

Violent gastralgia requires narcotics, such as morphine and opium. The best external applications are poultices or wet compresses. "Sour stomach" may be relieved by a pinch of bicarbonate of soda, or of calcined magnesia. Persistent anorexia may yield to the bitters mentioned above, or to small doses of quinine or compound tincture of cinchona. If the bad taste in the mouth is annoying, the mouth should be frequently rinsed out with Seltzer water, a one-per-cent. solution of carbolic acid, or five drops of tincture of myrrh to a glass of water. For habitual constipation, enemata, or the various mineral waters, are good; also Carlsbad salts. In obstinate cases pills of rhubarb or aloes may be employed. Still, we ought never to forget that the infrequency of the stools is often merely a natural consequence of the scanty diet, and that it is therefore possible to do harm with our purgatives. Iron is often prescribed for the concomitant anæmia; but it should be employed cautiously, for it is often ill borne in gastric disease.

CHAPTER IV

PHLEGMONOUS GASTRITIS

(Purulent Inflammation of the Stomach)

Purulent inflammation of the stomach is very rare, and little is yet known about it. In most cases no special causes for it have been ascertainable. It is occasionally one of the symptoms of grave pyæmic or puerperal inflammation. We have seen one case in which suppuration of the pharynx led to an acute phlegmonous inflammation of the submucous coat of the œsophagus, also involving a large portion of the stomach, with a fatal termination.

Two forms are distinguished—a diffuse and a limited variety. The latter is equivalent to gastric abscess. The submucous layer is almost invariably the chief seat of suppuration. From this starting-point the process invades the muscular and serous coats on the one hand, and the mucous membrane itself on the other—the usual result being multiple sievelike perforations inward or outward.

The usual symptoms are violent gastric derangement, with pain and vomiting, high fever, and the indications of constitutional infection, namely, headache, delirium, and general prostration. Sometimes the disease is quickly fatal, sometimes it runs a more chronic course. The few cases in which recovery has been reported are somewhat obscure.

The disease can never be diagnosticated with absolute certainty. Treatment must be purely symptomatic. Ice, both internally and externally, and the narcotics, are chiefly employed.

CHAPTER V

GASTRIC ULCER

(Simple or Round Ulcer of the Stomach)

Ætiology.—Since Cruveilhier gave the first accurate description of gastric ulcer, numerous explanations of its occurrence have been propounded. Even yet there is no universally accepted view. We may, in general, hold to the assertion that the development of an ulcer is preceded by a local disturbance of nutrition, or necrosis of the gastric mucous membrane, and that then the enfeebled or necrotic tissue is dissolved by the gastric juice—i. e., digested ("peptic ulcer"). But it is not known what are the special causes which occasion the primary damage to the tissue, nor why the loss of substance when it occurs is not at once healed, but extends in width and depth. There is, indeed, no lack of theories to solve these questions, and pathologists have made many experiments with regard to the pathogenesis of gastric ulcer.

The explanation of the fact that the normal gastric mucous membrane is not attacked by gastric juice lies probably not so much in the presence of the alkaline blood flowing through the membrane as in the intrinsic vital resisting powers of the normal living cells. If the cells are in any way damaged there occurs at once an auto-digestion at that spot. Damage of this sort may be produced experimentally in different ways; for example, by artificially produced embolism of the smaller arteries of the stomach, and by wounds, including bruising, burning, and cauterization of limited portions of the mucous membrane. In all such cases a peptic ulcer is formed, but almost invariably this promptly heals, or, at any rate, it shows no tendency at all to extension. If, as a symptom of severe gastritis or portal congestion, there are small hæmorrhages into the mucous membrane, they result in so-called hæmorrhagic erosions; but these again are limited to the small spot destroyed by the hæmorrhage.

Virchow assumed that ordinary round ulcer of the stomach in man is, in most cases, the result of the plugging of the small vessels with a thrombus or embolus, because of various diseased conditions of their walls, but this supposition remains entirely without proof. One fact that tends to contradict this view is the appearance of ulcer in young individuals without any signs of cardiac or vascular disease. Other investigators thought of the possibility of accidental injuries, and, perhaps in some few cases, even, of external trauma. Such suppositions are perfectly possible, although they, again, in most cases are incapable of demonstration. Then we have still to meet the second above-mentioned question: Why, in all such cases, the little erosions, if they are formed, do not immediately heal, as the experimentally produced ulcers almost invariably do. Lately the attempt has been made to settle this difficulty by pointing out that in ulcer of the stomach the acidity of the gastric juice is shown to be abnormally great. This hyperacidity is assumed to exist even before the development of the ulcer, and in a certain degree to cause a predisposition for its development, as well as to explain the unwillingness of the ulcer to heal and its proneness in many cases to extend farther, to spread. If a gastric ulcer is artificially produced, the process of healing may be considerably delayed by introducing solutions of hydrochloric acid into the stomach. It is very possible that hyperacidity of the gastric juice is a factor in the ætiology of ulcer, but the whole matter is by no means easily intelligible. We remain uncertain as to what is the primary cause of the ulcer, and it has not yet been proved that hypersecretion and hyperacidity exist before the formation of the ulcer. We might also believe that the excessive acidity is secondary to the sensory irritation occasioned by the raw spot. In a word, our present knowledge of the development and extension of gastric ulcer is very limited.

Gastric ulcer occurs but seldom in childhood. It is most often seen between the ages of eighteen and thirty. The cicatricial stenosis of the pylorus and other sequelæ are often seen in still older individuals, between thirty and forty-five years; but in most of these cases the disease probably originated much earlier. In still later life gastric ulcer is rare. In general, the disease seems to be more frequent in the female sex than in the male; but if we count only the absolutely demonstrated cases, the difference is not a very great one. The view that ulcer of the stomach attacks by preference anæmic and chlorotic girls is very general, but to the author seems much exaggerated. The probable explanation is that gastric disturbances in chlorotic persons are often ascribed to gastric ulcer without sufficient reason. Taking into consideration only the absolutely certain cases, the author's experience does not show any especially close connection between chlorosis and gastric ulcer. Yet it must of course be confessed that indubitable cases of gastric ulcer occur in chlorotic girls.

Pathological Anatomy.—The ulcer is usually approximately round or oval. Its borders are sharp; the walls often slope inward, giving the ulcer the form of a shallow funnel. The base of the ulcer is almost always perfectly clean, so that in microscopic sections we see the ends of the gland tubes remaining unchanged, and reaching out into the surface of the ulcer. After an ulcer has lasted some time, a reactive inflammation develops around the necrotic area, leading to the formation of connective tissue and cicatrization. If superficial, it does not extend farther than to the muscular coat, but it may be deep enough to expose the serous membrane, or even to perforate it (*vide infra*). The size varies greatly. Some are hardly as large as a pea; others may measure ten to fifteen centimetres in their greatest diameter. As to position, most of them are found near the pylorus. They attack the posterior wall of the stomach, particularly the neighborhood of the lesser curvature, far more frequently than the anterior wall, but the greater curvature is not infrequently the seat of an ulcer. As a rule, we find but a single ulcer, although exceptions to this statement are not very rare.

If an ulcer of any size heals, a scar is formed, with radiating lines and often of considerable size; smaller superficial ulcers heal with a smooth scar. Cicatricial contraction may alter the shape of the stomach considerably. If a deep constriction is formed around the middle of the stomach, we have what is called the hour-glass shape. Scars of pyloric ulcers are of the greatest clinical importance, because they lead to cicatricial stricture of the pylorus, with resultant dilatation of the stomach.

If the gastric ulcer extends to the serous membrane, the final result may be perforation into the abdominal cavity, and general peritonitis, unless the stomach previously becomes attached at the point threatened to some neighboring organ, because of adhesive inflammation. The ulcers being usually on the posterior wall of the stomach, it is oftenest the pancreas to which the stomach becomes adherent. In other instances it is the liver, transverse colon, diaphragm, or spleen. If adhesions are formed before the perforation of a gastric ulcer, there may be circumscribed peritoneal abscesses. These are situated with comparative frequency between the upper surface of the liver and the diaphragm, or between the stomach and the diaphragm (subphrenic abscess). Again, after such adhesions there may be perforation into the pleural cavity, the transverse colon, the pericardium, or the lungs.

The ulcer may cause erosion of a blood-vessel, and thus give rise to one of the most important symptoms of the disease, namely, gastric hæmorrhage.

Clinical History.—There may be absolutely no symptoms. It is not a rare thing to find at autopsies a still active ulcer of the stomach, or the cicatrix left by one, in subjects who never had during life any gastric disturbances whatever. Nor is it very exceptional for a person suddenly to exhibit grave symptoms, such as gastric hæmorrhage, or peritonitis due to perforation, when there has been no reason previously to apprehend the existence of an ulcer.

In other instances the ulcer does, indeed, give rise to symptoms, but they are not sufficiently characteristic to point to the correct diagnosis. Usually the gastric symptoms are long continued, but comparatively slight. They consist, for instance, of a sense of oppression or slight pain in the epigastrium, eructations, and occasional vomiting. In these cases, also, grave symptoms consequent upon the ulceration may suddenly arise.

In a third class of cases there are symptoms which are to a certain extent characteristic, and lead with more or less definiteness to the true diagnosis. These "symptoms of ulcer" are chiefly a peculiar epigastric pain, which is usually intermittent, and vomiting, or, what is yet more distinctive, the vomiting of blood, or hæmatemesis; also the signs of an increased secretion of hydrochloric acid in the gastric juice (hyperchlorhydria and hypersecretion). These symptoms and their diagnostic value we must now consider in detail.

Pain in the stomach is one of the most frequent symptoms of round ulcer. Its forms are very diverse. Often the patient complains only of a diffuse, painful sensation of pressure referred to the entire region of the stomach. This may be uninterrupted, or it may occur only after meals, or after excessive exertion, or as the result of some other special cause. This sort of pain is the least diagnostic of any, inasmuch as exactly similar sensations may be caused by other chronic disorders of the stomach. More characteristic of ulcer is a decided cardialgia, or, more correctly, gastralgia—that is, a very violent pain, coming on at intervals like neuralgia. It is described as "cutting," "tearing," "boring," and the like. These attacks of pain occur at various times, but oftenest after eating, and particularly after the ingestion of a large amount of food, or of food of a rather coarse sort. Often they occur with tolerable regularity a definite time after eating, say half an hour or an hour. This is partly to be explained by the beginning of the expulsion of food through the pylorus, and partly by the acme of the secretion of acid being due at this time. The pain is felt chiefly in the epigastrium, but not infrequently it extends toward the umbilicus, backward toward the vertebræ, into the thorax, or even into the upper extremities. In many instances a marked sensation of thoracic oppression accompanies it. A change of position may sometimes affect the severity of the pain. It is sometimes observed that the patient, when lying upon his right side, feels violent pain, which is at once relieved by changing to the left side, probably because the ulcer is located near the pylorus. An attack of cardialgia may last for a few minutes or for several hours. A third variety of pain may be observed in gastric ulcer. The suffering may be localized in a very limited area. Such pain is thought to be due to irritation of the floor of the ulcer by food, or to its edges being pulled upon during the movements of the organ. It generally comes on after eating, and ceases if the stomach is perfectly quiet. In position, this pain is generally epigastric, but sometimes it is umbilical, or even, now and then, more toward the back. In many cases of gastric ulcer there is also tenderness on pressure in a quite sharply defined area and at one particular spot. Most authors regard the accurately localized pain as the most nearly pathognomonic; but it must be said that it is decidedly the least frequently exhibited of any. Transitional forms and combinations of the various kinds of pain are often observed. Two things may be said to be characteristic in all the sorts of pain connected with ulcer of the stomach: the localization of the pain each time in the same spot; and the relation of the pain to the ingestion of food. Complete bodily rest is beneficial; external pressure upon the epigastrium usually increases the pain.

Vomiting is a frequent symptom in gastric ulcer, although it may exceptionally be absent or nearly so. Ordinarily, vomiting occurs, like the cardialgic attacks, after eating, and particularly after partaking of indigestible dishes. The vomitus in such cases consists mainly of partially digested food. It usually has a strongly acid reaction. If there is hypersecretion there may sometimes occur vomiting of a strongly acid fluid, not containing much that has been swallowed. If the ulcer has led to stenosis of the pylorus, the way in which the vomiting occurs is very characteristic. We shall describe this form of vomiting later, in detail. Vomiting is of the greatest diagnostic importance when blood appears. This may be intermixed in greater or less amount with the other contents of the stomach, or it may be vomited as pure blood (hæmatemesis). A gastric hæmorrhage demonstrated by the vomiting of blood is the most valuable diagnostic sign of gastric ulcer. If there is no hæmorrhage at all, the recognition of gastric ulcer is almost always rather difficult and uncertain.

Hæmatemesis is frequently the symptom which first leads the patient to apply to a physician. Up to this time he may have felt perfectly well, or, although there may have been some gastric derangement, he has not thought anything of it. The patient suddenly becomes faint, perhaps while he is pursuing his regular occupation, or it may be at night. He feels dizzy, and everything looks black. Then he has nausea, and finally is obliged to vomit. The vomitus is either pure blood, or a mixture of blood and food. It is partly coagulated, and often has a rather dark or blackish color, like tar. This change in color, as well as the coagulation, is due to the action of the gastric juice. The hæmoglobine is transformed by the action of the hydrochloric acid into hæmatine. If the blood, before it is vomited, has remained for some time in the stomach, the vomitus has the appearance of "coffee grounds," and no longer contains unchanged red blood-corpuscles (for the chemical demonstration of blood, *vide supra*, page 422). The amount varies greatly in different cases: there may be a quart or more. Sometimes there is a single hæmorrhage, but not infrequently blood is repeatedly vomited either at short intervals or on successive days. Part of the blood escapes through the pylorus, so that, after a profuse gastric hæmorrhage, blood is sure to be found in the stools. In them it is black and tarry. Exceptionally it happens that all the blood, beyond what is absorbed from the intestinal canal, passes off *per anum*, so that none whatever is vomited. In such cases it is often a difficult matter to locate the hæmorrhage. If there is a sudden faintness with pallor, and without vomiting, but followed by black fæcal discharges, we should always think of the possibility of a gastric hæmorrhage.

The consequences of gastric hæmorrhage depend, of course, chiefly on the amount of blood lost. Sometimes, although fortunately rarely, a large blood-vessel is eroded and the patient dies. This event may be sudden, or it may occur more gradually under the influence of repeated hæmorrhages and after a few days, during which all the symptoms of acute anæmia are exhibited. On the other hand, the loss of blood may be so insignificant as to produce no especial symptoms. In most instances life is not actually threatened, but yet the signs and results of a more or less marked general anæmia are clearly visible.

In such cases the patient feels extremely exhausted, and at once takes to his bed. He has also all the subjective symptoms of cerebral anæmia. There are vertigo, tinnitus aurium, specks before the eyes, frequent gaping, and sometimes headache. To assume an erect posture aggravates the disturbance. There is usually excessive thirst. Now and then a temporary amaurosis has followed an excessive hæmorrhage.

Objectively, we notice at once the excessive pallor of the skin, particularly of the face. The lips and conjunctivæ are also blanched. The pulse is rapid, and often ill sustained. For some days there may be anæmic murmurs over the heart, and there is a distinct sound to be heard in the femoral arteries. A moderate rise of temperature is very common, probably due to the absorption of the blood decomposing in the intestines. This is known as anæmic fever. The urine is pale, and usually rather abundant. Its specific gravity is not infrequently relatively high, namely, 1015 to 1020. All these symptoms are directly referable to the loss of blood, and they will be discussed with greater detail in the section on anæmia.

If the hæmorrhage is not repeated, the patient gradually regains his strength. To be sure, the pallor usually persists for a long time, but the disagreeable symptoms gradually abate. When gastric discomfort has existed previously to the hæmorrhage, it often disappears entirely after it—a circumstance which is probably due in part to the excessive caution of the patient thereafter. At the end of a few weeks the patient often feels perfectly well again; and, indeed, recovery is not infrequently complete and permanent. In other cases, however, the symptoms of ulcer return, sooner or later.

The third group of symptoms in ulcer of the stomach is furnished by an examination of the gastric contents, with the aid of the stomach-tube. These are of extreme importance. Earlier, when the soft tube now universally employed was not in use, we feared to introduce a tube into the stomach in case of ulcer, and even now we should not do it, in case of hæmatemesis, if it can be avoided, until some weeks have elapsed; but with this exception abundant experience has shown that we do not need to be at all timid about the introduction of the stomach-tube; and particularly if there is doubt about diagnosis we should never fail to avail ourselves of it. The most important fact which the investigations of the gastric contents in patients with ulcer has taught us is that there are almost invariably present hyperacidity and hypersecretion of the gastric juice. If the stomach is examined when fasting, we shall find a fluid containing a large amount of hydrochloric acid, and if we examine it an hour after a test breakfast, we shall find an extreme amount of acid (*vide supra*, page 417). This fact was discovered by Riegel, and we have found it true in all uncomplicated cases of ulcer of the stomach, indeed it seems to be so much the rule that if anacidity were found it would render any previous suspicion of ulcer extremely improbable. If, however, the ulcer has led to stenosis of the pylorus, and so to extreme stagnation of the gastric contents, there may be temporary subacidity or anacidity (*vide infra*). Likewise—and this is a point of great practical importance—hydrochloric acid may be absent if a gastric carcinoma has developed on the base of a former gastric ulcer (*vide infra*). Lactic acid is invariably absent when there is hyperacidity from hydrochloric acid; or, at most, if there is great stagnation of the gastric contents, there may be a moderate formation of lactic acid. It is also very important in cases in which there is no vomiting at all, to examine the gastric contents obtained by rinsing out the stomach, with regard to the possible presence of blood. Small admixtures of blood in association with hyperacidity are strong evidence of gastric ulcer.

The motor efficiency of the stomach in ulcer is at first normal or even somewhat exaggerated, because the digestion of albumen is very energetic owing to the excess of hydrochloric acid in the stomach. But if the ulcer is located in the pyloric region and leads to a cicatricial stenosis, the emptying of the stomach is delayed and a greater or less amount of food will be found seven or eight hours after the test meal. We shall later describe the symptoms of well-marked obstruction of the pylorus. If there is suspicion of an ulcer, the stomach is usually not inflated at all, or if so only by means of the condom (see page 421). If, however, there is a cicatricial stenosis of the pylorus, or an hour-glass contraction of the stomach, our best means of determining the form and size of the organ are by the introduction of air.

There are no other symptoms in gastric ulcer so important as those already described. The appetite in many cases is well maintained, and if the patient takes little food it is only because he dreads the ensuing pain. Eructations may be absent or there may be sour eructations and heartburn, symptoms referable to the existence of hypersecretion and hyperacidity. The tongue is seldom coated, being usually smooth and red; the bowels are apt to be somewhat constipated; the urine is often very faintly acid, so that there is a tendency to a deposit of phosphates.

The general nutrition of the patient often remains good, but there may be marked emaciation if there is a long-continued diminution in the amount of food taken, or if there is obstinate vomiting. Sometimes, but by no means invariably, the patients display great anæmia. This may precede the ulcer (*vide supra*, ætiology), or it may develop in association with the symptoms of ulcer, and even without any severe hæmorrhage to explain it; but that after severe hæmatemesis there should be a severe anæmia is a matter of course, and has already been alluded to.

An event which has been already mentioned under pathology—namely, perforation due to the ulcer—is of great clinical importance. It would be impossible to particularize here all the possibilities incident to it. We shall confine ourselves to the two most important because most frequent varieties of perforation: (1) into the peritoneal cavity, causing peritonitis, and (2) into the left pleura, or left lung.

Perforation into the peritoneal cavity leads almost invariably to a quickly fatal peritonitis. When the ulcer has previously caused few symptoms, if any, the excruciating abdominal pain, tympanites, vomiting, collapse, and sudden death of peritonitis may abruptly supervene upon a state of apparently perfect health. In other cases in which there has been a previous formation of adhesions, we have an encapsulated abscess, either subphrenic, between the stomach and the diaphragm, or elsewhere in the abdomen. The subphrenic abscess may contain air as well as pus, and is then termed pyo-pneumothorax subphrenicus. In making a diagnosis of any subphrenic abscess, we should consider the direct and constitutional symptoms, and also the thoracic signs, for the respiration and the inspiratory expansion of the lungs should be normal. If there is perforation outward or into a coil of intestine, recovery may result. More often, however, the termination is fatal, unless surgical intervention is successful.

Perforation into the left pleural cavity we have observed repeatedly. It causes a purulent or septic pleurisy on that side, and pulmonary gangrene may develop at the same time or later, as a result of perforation into the lung. Whenever we meet a case of apparently spontaneous, left-sided empyema, we should at any rate always think of the possibility of gastric ulcer.

In many cases of ulcer we observe signs of threatening perforation—that is, localized pain due to peritonitis, vomiting, and similar symptoms, and yet the case finally clears up. Probably in most such instances there is a limited peritonitis resulting in adhesions.

The general course of round ulcer of the stomach varies greatly, as we can see, in different cases. Complete recovery is by no means rare. In other cases the symptoms persist for years with varying intensity. We have already spoken of the hæmorrhage and perforation which may suddenly intervene, and of their significance. Relapses are not infrequent, even after apparent recovery. If the ulcer cicatrizes, the scar itself may give rise to persistent disturbances: there may be obstinate cardialgia, or, if the scar is at the pylorus, dilatation of the stomach (*vide infra*) may gradually be developed. This extremely important sequel of gastric ulcer deserves special consideration.

The cicatrization and contraction of a gastric ulcer which result in stenosis of the pylorus may be complete, but it is by no means exceptional for the ulcerative process to persist in other places while there is a scar at the pylorus, so that we may have the symptoms of stenosis combined with all the other symptoms of ulcer, such as pain, hyperacidity, hæmorrhage, and perforation. When there is an obstruction of the pyloric orifice as a result of interstitial thickening and scar formation at that place, a group of extremely characteristic symptoms is superimposed upon the ordinary symptoms of ulcer. Perhaps one cause of the change in the pylorus is the destruction of the muscular coat at that point.

The first and most important result of pyloric stenosis is prolonged retention of the gastric contents. So long as the muscular coat of the stomach is able by increased effort to overcome the increased resistance, there is no disturbance to be noticed. It is proper to speak of a compensated pyloric stenosis, just as we speak of a compensated valvular disease of the heart; but if the stenosis becomes greater, so that even the hypertrophied muscular coat ceases to be able to discharge the chyme into the intestine, there is a gradually increasing collection of food in the stomach, and the only way for the organ to relieve itself is by vomiting. The vomiting, in case of pyloric stenosis, is somewhat characteristic. It occurs from time to time, perhaps at intervals of two or three days, and is very profuse. Then, the stomach being partially emptied, new collections may form until their large amount leads to vomiting again. The vomitus in cases of stenosis due to ulcer is usually very acid and contains much free hydrochloric acid, and sometimes blood. The stomach itself becomes gradually and increasingly dilated, because of the stagnation of its contents. Usually this secondary dilatation of the stomach may be recognized by mere external examination. We can feel the stomach as a great limp, flabby bag; and from time to time we can see its walls bulge out from muscular contraction. We can often distinctly recognize the peristaltic waves passing from the fundus to the pylorus. In the region of the pylorus we may sometimes, though by no means invariably, make out a distinct resistance on palpation, suggesting a tumor and corresponding to the thickening of the gastric walls from scar formation. In such cases it is often especially difficult to distinguish between the scar of an ulcer and a carcinoma. Even if we can feel no tumor, visible contractions of the stomach with distinct peristalsis are almost invariably pathognomonic of pyloric stenosis. In such cases there is usually a cramplike, painful sensation, associated with every marked contraction of the muscular coat. During these contractions of the stomach its contour is often so evident that we can obtain a perfectly distinct idea of its size, extent (often reaching below the navel), and position. But we must especially emphasize the fact that the size of the stomach, its dilatation, is not the essential point. The main thing is the stenosis of the pylorus, and, above all, the degree of consequent motor inefficiency. With regard to the latter, we can reach no complete conclusions without using the stomach-tube. With its aid we can determine the amount the stomach contains, and recover ingesta, such as cranberries and green-colored vegetables, which have been eaten many days before. If we empty the stomach completely and give a test meal, we can gauge more accurately the degree of motor insufficiency. We may also determine precisely the position, form, and size of the stomach when emptied, by means of inflation. In exceptional cases there is a large amount of bile mingled with the gastric contents. The author surmises that in such cases the cicatricial process has changed the pylorus into a rigid, though narrow, tube, which can neither be opened nor closed. Thus we have an "insufficiency" associated with the stenosis of the pylorus, so that no obstacle is opposed to the entrance of bile into the stomach.

When there is a well-marked uncompensated stenosis of the pylorus, there is always a decided impairment of the general health. Nutrition grows more and more imperfect because of the persistent vomiting, and there may be extreme emaciation. Still, many patients with stenosis due to ulcer may maintain a tolerable condition of health for many years, if they take proper care of themselves by regular washing of the stomach and by eating proper food (*vide infra*); but finally, even in such individuals, the symptoms grow worse, or a new trouble develops, one of which there is always danger in any case of stenosis due to ulcer. We refer to the secondary development of carcinoma at the seat of the former ulcer. This happens not infrequently, and is to be considered at length in the next chapter. There remain to be mentioned some special symptoms, associated with ulcers of the pylorus when they lead to stenosis. There is usually a decided tendency to constipation; the urine is faintly acid and often has an abundant deposit of phosphates; the pulse is often remarkably infrequent; and finally, there are sometimes attacks of tetany.

Diagnosis.—The diagnosis can be made only when the above-mentioned characteristic symptoms are present. Of these, hæmatemesis is by far the most significant, for it is with very few exceptions the result of gastric ulcer. Particularly is this true of individuals under middle age.—But how shall we determine whether the blood ejected did not come from the nose or the lungs, rather than the stomach? The answer is not always easy. If an epistaxis occurs at night, a part of the blood often flows back into the naso-pharynx, and, being swallowed, excites vomiting, so that a gastric hæmorrhage is suggested.

In doubtful cases we must, therefore, make an accurate examination of the nose. It is also very important for the physician to remember that blood may be "vomited" in hysterical cases. If there is also nervous cardialgia, one might easily be misled to assume the existence of a gastric ulcer. Usually, however, we can make sure of the diagnosis by means of the other symptoms of hysteria (see the chapter on hysteria), and by examining the "vomited" blood. The latter does not come from the stomach at all, but from the gums, the pharynx, or the naso-pharynx, and consequently it is almost always comparatively bright red, rather fluid, and mixed with mucus and saliva; and on microscopic examination it shows pavement epithelium and micro-organisms from the mouth.

The diagnosis between gastric and pulmonary hæmorrhage in doubtful cases depends on the following factors: 1. The previous condition of the patient—whether he has had cough, expectoration, and other pulmonary symptoms, or, on the other hand, gastric pain and vomiting. 2. On the character of the hæmorrhage, whether accompanied by vomiting or by cough. But there may have been both. Violent vomiting may excite a cough; and, on the other hand, blood which has been coughed up may be in part swallowed and induce vomiting. 3. On the character of the blood: if from the lungs, it is usually bright-red and frothy, containing bubbles of air, with few clots, and of alkaline reaction. In gastric hæmorrhage it is usually dark, mixed with food, partly clotted, and acid in reaction from admixture with the gastric juice. 4. On the results of physical examination. In this, of course, we must be extremely cautious after a hæmorrhage, lest the movements of the patient excite fresh bleeding; and yet we may be able to perceive from the general condition of the patient, or from dullness at the apices, or moist râles, that pulmonary disease is probable. If the blood came from the stomach, we usually detect nothing but the signs of anæmia. 5. The subsequent symptoms. In cases of pulmonary hæmorrhage there is almost sure to be an expectoration, for the next few days, either of pure blood or of matter stained with blood; and, in gastric hæmorrhage, the next dejection will almost certainly be black, from the presence of decomposed blood. In doubtful cases, the appearance of blood in the stools almost invariably settles the question in favor of gastric hæmorrhage. The best way of testing for blood in the fæces is by means of guaiac and turpentine (*vide supra*).

If no gastric hæmorrhage has ever occurred in the course of the disease, violent and persistent gastralgia of the peculiar character above described is that symptom which would first arouse suspicion of an ulcer, particularly if the pain is from time to time associated with vomiting. If hyperacidity or hypersecretion has been demonstrated, this condition in association with other symptoms may greatly strengthen the diagnosis. Severe gastric pain, combined with hypersecretion, makes the diagnosis of gastric ulcer very probable. To make an absolutely certain distinction between ulcer and nervous hypersecretion (*q. v.*) is impossible, if there is no hæmatemesis. In general, we must remember that it is often possible to observe more or less distinct "symptoms of ulcer," without being absolutely certain that an ulcer exists. Often we are obliged to await the further course of the disease, and particularly the results of treatment, in order to settle our doubts.

The two diseases which it is most difficult to distinguish from ulcer are nervous dyspepsia with nervous gastralgia, and carcinoma. When we consider these diseases we shall discuss at greater length the differential diagnosis between them and ulcer of the stomach. Chlorotic patients, also, often have gastric symptoms (see the chapter on chlorosis) which excite suspicion of an ulcer, especially as hypersecretion of the gastric juice is often associated with chlorosis. Frequently it is impossible to make an absolute diagnosis in these cases, but the results of treatment may be decisive. Preparations of iron, particularly Blaud's pills, are ill borne in ulcer, while in case of genuine chlorosis they usually cause rapid improvement in the gastric symptoms, as well as in the general condition.

Prognosis.—The chief dangers in ulcer of the stomach, hæmorrhage and perforation, have already been spoken of. Whether these complications will actually occur in any individual case, and when, we can not determine.

There can be no doubt that a large number of ulcers heal perfectly; but, as we have already said, even the resulting scar may cause trouble. We must remember the possibility of persistent gastric disturbance, particularly cardialgia, and also the development of a cicatricial stenosis with its consequences. Finally, we have the danger, already mentioned, of the eventual development of carcinoma in the scar of an old ulcer.

Treatment.—If the diagnosis of gastric ulcer is evident, or if the symptoms are of such a nature that there is a justifiable suspicion of an ulcer, the patient should be urgently advised to submit himself to careful and methodical treatment; for it is only by means of a sufficiently persistent and properly conducted treatment that we can hope for good therapeutic results in ulcer of the stomach.

One essential condition is, that the patient should keep his bed for the first part of the treatment, at least for three or four weeks. Complete bodily rest is certainly important in promoting healing of the ulcer. The patient should also have moist compresses, or, still better, as Leube has advised, warm poultices applied all day long to the epigastrium. These applications are very good for the pain, and they also have a certain psychological importance, for they reconcile the patient to keeping quiet in bed. Thirdly, and this is probably the most important point, the patient must keep strictly to a prescribed diet. This diet we are about to describe at length. The main consideration in determining the patient's diet is that all mechanical and chemical irritation of the floor of the ulcer must be avoided. Fourthly, we should have regard to the almost invariable existence of hyperacidity of the gastric juice. We, therefore, give to patients alkaline carbonates, either in the form of Carlsbad water (on waking a large glassful of the Mühlbrunnen warmed to about 98.6° F., 37° C.; then, in the forenoon, between ten and eleven, as much more), or, what we consider in many ways still better, bicarbonate of soda (either alone or combined with subnitrate of bismuth). Of the soda we should give as much as is held upon the point of a knife, every hour, so as to keep the gastric juice constantly neutralized. With regard to the dietetic treatment of gastric ulcer, the best way is to follow Leube's example, and have four diet lists. Beginning with the first, we gradually go on at definite intervals of time to the second, third, and fourth. For the first ten days of treatment the patient follows the first diet list. This contains boiled milk; broth; unsweetened rusks, softened by soaking; to this we may add the artificial solutions of meat, particularly that of Leube-Rosenthal. Then, during the next week, we have the second diet list, comprising gruel, soft-boiled rice and oatmeal, eggs, boiled calf's brain and sweetbread, boiled pigeon and chicken. From the eighteenth to the twenty-fourth day of treatment we have, in addition, the third diet, with shavings of raw beef and beefsteak, potato purée, boiled calf's feet, and for beverage some tea or weak coffee. From the twenty-fourth to the thirtieth day the patient has the fourth diet list—that is, broiled fowl or pigeon, partridge, venison, roast beef, tenderloin, finely chopped macaroni, and bread. It is understood that with each advance in diet the previous diet lists are also permitted. In general, the patient has about five meals a day, the amount of each being determined by the appetite and symptoms of the patient. If everything goes favorably, the gastric pain will wholly cease after a few days of this diet, under the other regulations above enumerated. The patient then proceeds toward recovery without any discomfort, except possibly hunger. If the transition to a more advanced diet causes pain, we must resort for a few days to the simpler foods. In general, the above suggestions must be regarded as an outline from which certain variations may be made to suit the individual case, yet we may be well assured that the results are better and more certain the more strict and methodical the treatment is. If the patient has borne the articles of the fourth diet list for about a week without any discomfort he may go on by slow degrees to other simple dishes, such as veal, pike, trout, porridge, vegetables, and other similar articles, and so to an ordinary diet. Of course, caution in eating and drinking is necessary for a long time.

With the mode of treatment just described, we shall in most cases accomplish as much as possible; but if, despite this regimen, the symptoms do not cease, there are two other remedies which deserve to be tried—viz., subnitrate of bismuth and nitrate of silver. These are specially employed, also, in treating poor persons whose circumstances often render it impossible to carry out strict dietary rules. The subnitrate of bismuth is almost always given in combination with bicarbonate of soda: of a mixture containing 3 to 5 parts of bismuth and 30 parts of soda we give several times a day a generous "knife-point full" before meals. In severe and obstinate cases of ulcer, we can from our own experience recommend also "injections" of bismuth (Fleiner). The patient's stomach is washed out in the morning before breakfast, and then ʒ iv–v (grm. 15–20) of the subnitrate of bismuth suspended in a glass of warm water is poured slowly into the stomach-tube; the patient should at the same time lie on his right side, and so remain for at least half an hour. The heavy bismuth powder is expected to deposit itself upon the surface of the ulcer (which is probably in the pyloric portion of the stomach), and thus in a mechanical way protect it from irritation and promote its healing. The symptomatic results of this method are sometimes excellent. Often, also, favorable results are obtained from nitrate of silver. This may be partly explained by its neutralization of the hydrochloric acid, and the formation of chloride of silver. The drug is prescribed in a solution of six grains to four ounces of water (gram. 0.3–120), to be kept in opaque glass, and of this a teaspoonful should be given three or four times a day before meals.

Sometimes the violent pain does not abate under the ordinary treatment, and demands symptomatic remedies. Of course, morphine is the most efficient, given internally or subcutaneously. Chloroform may also be tried: we give a tablespoonful of a mixture containing 1 part to 120 of water. Gerhardt recommends for the gastric pain 3 or 4 drops of the solution of perchloride of iron in a wineglass of water.

Excessive vomiting and persistent nausea are likewise to be combated by the narcotics. Opium is the best; morphine, cocaine, chloral, and bromide of potassium may also be tried. At the first appearance of blood in the vomitus the greatest bodily quiet and most careful dieting is absolutely indispensable. For the first day or two it is best to allow nothing except ice-cold milk and bits of ice in the mouth to appease the burning thirst. In severe cases we may try giving water and milk by the rectum, so as to afford the stomach complete rest. The patient must lie as quietly as he can. A flat ice-bag, not too heavy, should be placed on the epigastrium. In case of persistent nausea or eructations, small doses of opium or morphine are to be prescribed. If the hæmorrhage is obstinate we may try the subcutaneous injection of ergotine, or, by mouth, acetate of lead or chloride of iron. We must wait four or five days after a hæmorrhage before we can give a little more food by the mouth, and this must be allowed cautiously and in a liquid form.

If peritonitis appears as the result of perforation, the best means to try are the outward application of ice to the epigastrium and the internal use of opium in large amounts—that is, half a grain to a grain (grm. 0.03–0.05) every two or three hours, or twenty to thirty drops of laudanum. Unfortunately, however, the cases are exceptional in which the peritonitis does not become general. The only remaining hope lies in surgical interference, although the results even of laparotomy are dubious.

If the symptoms of pyloric stenosis are growing more and more distinct—that is, if the hypertrophied muscular coat of the stomach is no longer vigorous enough to prevent the stagnation of food, with consequent dilatation of the stomach and vomiting, we can perhaps restore the patient to a comfortable condition by means of regular washing out of the stomach, combined with a suitable diet. This irrigation prevents any great and burdensome accumulation of food in the stomach. Moreover, it rids the patient, for a time at least, of the large amount of strongly acid gastric juice which is usually present. These beneficial and agreeable effects are soon so noticeable that often the patient learns to rinse out his stomach himself, or even, by bending over and pressing, to empty out the gastric contents through the tube. As a rule, in cases of gastrectasia resulting from pyloric stenosis, we wash out the stomach every morning before breakfast. Ordinary lukewarm water suffices, but if there is an excessive secretion of hydrochloric acid it is satisfactory to employ 1- or 2-per-cent. solutions of bicarbonate or borate of sodium. In severe cases the operation may be repeated in the evening before supper.

The diet in stenosis following ulcer should consist of such articles of food only as can pass with comparative ease through the narrow pylorus, being at the same time highly nutritious. Most suitable are milk, eggs, soups with the addition of meat-juice, somatose, oatmeal, rice, or flour; gruel made from rice, oatmeal, or preparations like Mellin's food; and finally, finely chopped meat of all sorts. The meals should not be too large, but should rather be frequently repeated—a little food often.

In this way it is often possible to give the patient considerable relief for many years, and even to make him gain weight. Other methods of treatment—by applying electricity to the stomach, by cautious massage of the organ, or by the administration of strychnine—may be employed now and then in practice, but they have no special value in actual stenosis.

It may well be questioned whether the existence of such patients, who are preserved from excessive discomfort only by means of daily lavage and a monotonous and limited diet, is really bearable; and, still further, what are we to do when, in spite of the most skillful treatment by the method described, the symptoms persist and the nutrition and strength of the patient grow gradually more and more impaired? For such there is still the possibility of actual and complete recovery. We refer to surgical intervention. In no other severe disease of the stomach do operative procedures offer so great a prospect of favorable result as in stenosis of the pylorus following ulcer, and therefore it has been our rule for a series of years, in every severe case of this sort, to urge the patient strongly to an operation. Moreover, we should not delay too long, if the patient is already enfeebled, or if there is reason to suspect that carcinoma is developing.

The operation is usually gastro-enterostomy, or sometimes pyloroplasty; but we can not enter here into particulars. Certainly we ought not to conceal from the patient that we have to do with a serious operation, which is not absolutely without danger; yet in the great majority of cases the results of treatment are excellent. After a successful operation the patient is freed from all discomfort, he regains the appearance of health, and sometimes even the dilatation of the stomach completely disappears. It is true that later, perhaps years later, relapses may occur, whether because of special mechanical conditions or from the development of secondary carcinoma; but even then perhaps assistance will be given by a second operation.

[In cases of recent hæmorrhage or markedly painful digestion, the editor has pursued the following plan of treatment with apparently good results: The most powerful agent in the promotion of cicatrization is rest. Absolute rest in bed is therefore enjoined, with the secondary end in view of reducing the demands of the system. The patient is then fed exclusively by the rectum for at least one week, generally for two weeks, nothing but a little water being given by the mouth. A simple cleansing enema is given once daily; and at six-hour intervals a nutrient enema, six to eight ounces in bulk, and composed of one or two raw eggs, an ounce of expressed beef juice, and fully peptonized milk, is administered. Tolerance by the rectum is promoted by the addition to the enema of a few drops of laudanum. After one or two weeks small quantities of milk at frequent intervals are given by the mouth, and the rectal alimentation is gradually diminished as that of the stomach is increased. Pain usually ceases immediately on the cessation of gastric ingestion, and the loss of weight during a fortnight of rectal feeding is surprisingly small.]

CHAPTER VI

CANCER OF THE STOMACH

Ætiology.—We can not here discuss the ætiology of carcinoma in general, and we shall therefore merely enumerate the factors which experience has shown to favor the development of cancer in the stomach.

Age has a remarkable influence. Gastric cancer is decidedly most frequent late in life, between the fortieth and sixtieth year. Still it is occasionally seen in younger persons. We have ourselves seen several cases in persons between twenty-two and twenty-five years of age.

Sex is of no importance.

Heredity has a slight but undeniable influence. The most famous example of the transmission of cancer is presented by the family of Napoleon.

The relations of gastric cancer to previous disease of the stomach is very important. It is not probable that the tendency to cancer is increased by frequent errors in diet, indulgence in alcohol, or similar causes. Most of those who are attacked by gastric cancer have previously lived in a perfectly temperate manner, and report that they have thus far always enjoyed a good digestion. The exception is to discover that there has been any special injury or trauma, such as scalding or corrosive poisoning, and if there is a history of them they are to be regarded as merely contributory causes. Far more important, however, is the fact which the investigations of Hauser have lately confirmed, that gastric carcinoma is not infrequently developed in the cicatrized edges of a previous gastric ulcer. We have ourselves had a considerable number of cases in which both the clinical course of the disease and the results of the autopsy illustrated this mode of development.

Pathology.—The stomach is a favorite seat for cancer. About a third of all cases of cancer are gastric. The parts of the organ most often attacked are the pyloric end and the lesser curvature. Less frequently the cardiac end and the fundus suffer.

The new growth takes the form either of a circumscribed tumor or of a diffuse infiltration, thickening the walls. The disease invariably originates in the mucous layer, extending thence into the submucous, muscular, and serous coats. Often the cancer extends by way of the lymph-channels, or sometimes the veins. The lymph-glands and the omentum are frequently attacked by the new growth. Moreover, we find metastases in the peritoneum, and also with particular frequency and abundance in the liver, whither cancerous cells are carried by the branches of the portal vein. The connective and muscular tissues in the neighborhood of the cancer are quite often considerably hypertrophied.

Histologically, gastric cancer is of the cylindrical-cell variety, starting from the glandular epithelium. The soft tumors are termed medullary; the firm and hard, scirrhous or fibroid. The medullary cancers are particularly apt to be quite extensively broken down on their exposed internal surface, thus forming what are known as cancerous ulcers. This seems to be mainly the result of the gastric juice acting on the superficial and insufficiently vascularized portion of the tumor. The base of these ulcers is usually clean, as we should expect from the mode of their production. In many cases of rather young subjects, and sometimes in others, we find colloid cancer. This form also may appear either in nodules or as a diffuse growth, infiltrating the tissues. It is peculiarly apt to involve the peritoneum extensively, with consequent ascites.

Clinical History.—Most cases of gastric cancer exhibit a combination of grave digestive disturbances with a relatively rapid loss of flesh and strength. Now and then the gastric symptoms assume less prominence. The chief sign of disease is a constantly progressive marasmus or anæmia, the true cause of which is either entirely latent or not unmistakably recognizable till late in the illness.

Some of the gastric symptoms are not very characteristic. They merely show that digestion is disordered. The tongue is usually thickly coated and dry. There is loss of appetite and distress after meals. The patient complains of a disagreeable sensation of pressure in the epigastrium, increased by food. Sometimes this amounts to actual cardialgia. Many patients are annoyed by eructations. Occasionally vomiting is troublesome; in other instances there is scarcely any. The constant recurrence of vomiting is an almost sure sign that the cancer is situated in the pyloric region and obstructs the orifice. The vomitus may contain nothing but mucus and ingesta; or it may assume, from the admixture of blood, a very characteristic and somewhat pathognomonic appearance.

Free gastric hæmorrhage and consequent hæmatemesis is exceptional, or at least it is much less frequent than in ulcer of the stomach; but the vomitus often contains decomposed blood, and in many cases this will be for a time almost a constant appearance. Most of the ulcerated cancers bleed frequently, a little at a time. When it reaches the stomach the blood is decomposed and gives that well-known "coffee-grounds," or "chocolate-colored" appearance to the vomitus, or to the gastric contents obtained by means of a stomach-tube, which is so important a symptom for the diagnosis of cancer of the stomach. In such cases the best means for demonstrating the presence of blood, in the brownish or blackish fluid, is the guaiac-turpentine test (*vide supra*, page 422). This gives an extremely distinct reaction in the case of carcinoma of the stomach, because lactic acid also is usually present. We should mention that in the case of ulcerating carcinoma ventriculi the vomitus may have so foul an odor that it may even be regarded as stercoraceous.

Immediate microscopic examination of the vomitus may reveal red blood-globules. Further, the vomitus consists of partially digested food, and also contains an abundance of micro-organisms. Sarcinæ are found much less often in carcinoma than in ulcer of the stomach, but we frequently see under the microscope an abundance of yeast cells, and of long threadlike bacilli which perhaps have a relation to lactic-acid fermentation. It is extremely rare to find in the vomitus characteristic particles of cancer. It is somewhat less unusual to find little pieces of the growth attached to the stomach-tube, when the carcinoma is at the cardiac end or in the fundus of the stomach. These, if obtained, are of course to be submitted to microscopic examination.

The external examination of the stomach should always be made when the patient is lying flat on his back. Sometimes we can clearly recognize by mere inspection the position and size of the organ, any peristaltic motions (*vide infra*) that there may be, and even in some cases the presence of a tumor. Palpation is, however, more important. In a great many cases the new growth can be felt with distinctness through the abdominal wall as a hard irregular tumor. Its situation is, in a majority of cases, the epigastrium. Carcinoma of the pylorus, which is the most common, is usually felt in the right half of the epigastrium; carcinoma of the greater curvature somewhere in the level of the navel. But it should be considered that the position of the stomach may be considerably altered by the presence of a growth—for example, we have seen a case of pyloric cancer with secondary dilatation of the stomach, in which the pylorus had sunk so low that the tumor could be felt about a hand's breadth above the symphysis. In some cases the tumor varies its position (*vide infra*) according to the degree of distention of the stomach. The influence of respiration upon the tumor is various. As a rule, cancer of the pylorus does not change its place much with respiration, unless it is adherent to the liver. Carcinomata of the lesser and greater curvatures usually do move with respiration; and when displaced downward they may be held in that position with the hand, returning to their ordinary location when the hand is removed. Tumors of the liver and cancers of the stomach which are adherent to the liver can not be thus held down. When the carcinoma is situated high up, in the lesser curvature or near the œsophagus, it may not be felt at all except during inspiration.

In a minority of the cases no tumor can be felt at any time. This is true in most instances of diffuse cancerous infiltration of the walls of the stomach. Under such circumstances the diagnosis can not often be definitely established. The tumor is undiscoverable, first, in most cases of diffuse cancerous infiltration of the stomach-walls. We may, indeed, notice an increased sense of resistance and hardness in the epigastrium, but we can not refer this condition with certainty to a new growth. Secondly, the new growth may extend chiefly inward, toward the cavity of the organ, and may thus escape detection. And, finally, the tumor may be so concealed by the liver or the edge of the ribs that it is inaccessible to the touch. Such cancers as attack the cardiac extremity, the posterior wall, or the lesser curvature of the stomach, are particularly apt to be out of reach of palpation.

Percussion of the cancer rarely gives flatness, but instead a muffled tympanitic resonance. This is sometimes an influential factor in the differential diagnosis from cancer of the liver. In a few cases, upon auscultation in the neighborhood of the tumor, we may hear a soft systolic murmur, probably due to compression of a rather large artery.

The examination of the gastric contents is of the greatest diagnostic importance. While introducing the stomach-tube, or perhaps, if need be, a less flexible instrument, we should notice whether the tube enters the stomach with ease. Carcinoma at the cardia is not infrequently the cause of a sensible resistance to the tube when it passes the lower end of the œsophagus. With regard to the examination of the gastric contents, the chief point is about free hydrochloric acid. Von der Velden first pointed out what numerous investigations have since confirmed, that in almost all cases of gastric carcinoma there is no free hydrochloric acid, so that the phloroglucine-vanilline reaction an hour after the test breakfast is negative. Associated with other symptoms this is an extremely important diagnostic sign (*vide infra*). The cause of this almost invariable absence of free hydrochloric acid in carcinoma of the stomach has not yet been determined, especially as this symptom has often been observed very early in the course of the disease. The associated catarrh of the gastric mucous membrane and its secondary atrophy may have some influence, but probably there are also other unknown causes at work. An examination for pepsine may be omitted in practice: it also often turns out negative. Far greater importance attaches to the fact that the gastric contents in carcinoma are very apt to show a surprising amount of lactic acid. This is almost invariably so when the carcinoma obstructs the pylorus. The explanation of this is very simple. There is no hydrochloric acid to exert an antiseptic action, and the food is stagnant in the stomach, so that there is an especially favorable opportunity for the development of a vigorous lactic-acid fermentation. There is, then, nothing specific about the presence of lactic acid in the gastric contents in cases of carcinoma, only it is a very frequent and therefore not unimportant symptom.

The motor efficiency of the stomach is impaired in most cases of carcinoma, except in cancer affecting the cardia, or in small cancers on the fundus. Whenever there is an extensive development of carcinoma of the stomach, the muscular coat of the organ is sufficiently damaged to embarrass its motor activity; but of course the hindrance to the discharge of its contents is greatest when the new growth is at the pylorus. In this case, besides the direct destruction of the muscular tissue, we have the purely mechanical stenosis, and this may at last become extreme. For this reason, if we find a marked stagnation of the ingesta we can usually infer that the seat of the carcinoma is the pylorus (*vide infra*). We should not omit to inflate the stomach when it is possible. We can thus determine not only the size and position of the stomach, but also its relations to any tumor that may be felt; and finally, it should be added that a test for blood (*vide infra*) should be made with the fluid obtained by washing out the stomach.

Certain peculiarities in the clinical picture of gastric carcinoma are occasioned by its special location; if at the cardia, the symptoms are similar to those of cancer of the œsophagus. As soon as the cardia is narrowed, the patient himself feels that it is difficult for food to enter the stomach, and speedily there appears vomiting, or, more accurately, regurgitation of food, so that the patient can take nothing but liquid nourishment. The diagnosis of carcinoma at the cardia is usually easy. We feel the resistance at the cardia with the stomach-tube some 16 to 18 inches (40–45 centimetres) from the front teeth. Externally the growth can not be felt until it invades the lesser curvature of the stomach. Then we often feel the tumor in the epigastric angle, especially on deep inspiration. In carcinoma of the fundus the symptoms may be indefinite for a long time. The pylorus remaining permeable, there may be no vomiting at all. In late stages the tumor which may be felt is often very large, particularly if there is involvement of the omentum, which changes it into a great cancerous mass.

When cancer at the pylorus leads to stenosis, the clinical picture is especially important as well as very common and very characteristic. As soon as there is a hindrance to the emptying of the stomach, whether because the muscular coat is impaired by infiltration with carcinoma or because there is the direct mechanical obstacle of stenosis, there result retention and accumulation of food in the stomach, with exaggerated peristalsis, and finally gastric dilatation. The circumstances are precisely similar to those depicted in the preceding chapter when speaking of cicatricial stenosis (cf. page 438). In advanced cases the contours of the dilated organ are clearly seen through the emaciated and collapsed abdominal walls. From time to time a contraction occurs, and we see the peristaltic wave slowly travel toward the pylorus. Very often the carcinomatous growth near the pylorus is distinctly palpable, and even visible. Often it moves with respiration. Washing out the stomach will demonstrate the stagnation of its contents, and by inflation we determine its size and position. If the stomach is not emptied artificially, from time to time very large amounts are vomited, the vomitus being invariably devoid of hydrochloric acid but containing much lactic acid, and sometimes also showing fermentation. If we let some of the gastric contents, obtained by vomiting or through the tube, stand in a glass vessel, it divides into layers. If fermentation has set in, a foamy layer is formed on top.

We have now enumerated the symptoms of gastric cancer, referable to the stomach itself. The greatest attention should also be paid to the general disturbance of nutrition occasioned by this gastric disease. Loss of flesh is not rarely the very first symptom which calls the patient's attention to his disease. This wasting is observed earliest in cases which are attended with anorexia and vomiting. The patient also gradually takes on that familiar sallow cachectic look which is characteristic of most cases of cancer. Some patients become excessively anæmic. The skin acquires a waxy pallor, and there are all the symptoms which result from great anæmia, such as cerebral disturbances and functional cardiac murmurs. Sometimes the blood itself presents decided peculiarities in such cases. Thus we may find microcytes and poikilocytes in it. Gastric cancer and pernicious anæmia (*q. v.*) have been repeatedly confounded. In one such case we made the interesting discovery of extremely abundant metastatic cancer in the bones. As the bone marrow is known to have something to do with the production of the blood, it may be that the anæmia was due to this abnormal condition. At any rate, the grave anæmia which results from cancer can not be regarded in just the same light as the loss of flesh and the cachexia. We often find extreme anæmia in patients who are tolerably well nourished, while, on the other hand, many patients with carcinoma, though emaciated to a skeleton, do not display this peculiar anæmic pallor. The anæmia must, therefore, depend upon some special circumstances. Probably it is most frequently caused by continued though small hæmorrhages, due to ulceration of the new growth.

Special derangements of other organs are relatively infrequent. Metastatic cancer is of importance. It attacks the liver chiefly. If the hepatic new growth is considerable, it may quite overshadow the primary cancer—there being jaundice and a greatly enlarged, nodular, and tender liver. Secondary carcinosis of the peritonæum is also apt to cause marked symptoms, such as ascites and abdominal pain. Secondary cancer may also involve the mesenteric and retroperitoneal lymph-glands, the lungs, and other organs, but it does not usually give rise to striking symptoms when so situated, although the discovery of even small metastases by palpation may have great diagnostic significance. Older physicians frequently mention the occurrence of swollen lymph-glands above the left clavicle, but we have scarcely ever found them there, although we have paid special attention to this point. Swelling of the inguinal glands is more important. We have seen small metastases in them several times. Finally, we would like to call special attention to the appearance of small secondary nodules at the navel, a fact which we have repeatedly noticed. The discovery of such a growth is of great diagnostic value when no primary tumor can be made out by palpation.

Direct extension of the new growth into neighboring organs is not very frequent. We will venture to mention one case which we saw, on account of its great rarity. The new growth caused adhesion of the anterior wall of the stomach to the abdominal wells, and then, penetrating through them and the skin of the epigastrium, finally appeared as a tumor, of about the size of one's fist, projecting outward. If a cancer ulcerates, it may destroy all the layers of the stomach, and result in perforation and secondary peritonitis; or, if previous adhesions have been formed, the perforation may open up an abnormal communication between the stomach and some neighboring part of the intestine. The transverse colon is the part usually perforated; less often the small intestine.

As to the bowels, constipation is the rule. Diarrhœa is rare. The urine is usually pale and but slightly acid. Its amount is diminished, as we should expect from the slight amount of nourishment taken, and from the vomiting. Over the heart we may sometimes hear soft anæmic murmurs. The pulse is usually accelerated, although, if there be extreme marasmus, it may be slow.

The temperature is often normal. If the patient is greatly emaciated it is not infrequently subnormal, but, on the other hand, if the thermometer is carefully used we shall very often find occasional irregular elevations of temperature, 100.5°-102° (38°-39° C.), or even a persistent or intermittent fever. The cause of these elevations, exclusive of complications, is probably, in most cases, the absorption of septic material from the ulcerated surface of the carcinoma. If there are hæmorrhages, another factor may be the absorption of the decomposed blood (fibrine ferment). Not infrequently, in advanced stages of the disease there is more or less œdema of the ankles, hands, and other parts. The explanation of this is the same as in most cases of œdema in cachectic and anæmic patients—viz., the impaired nutrition of the vascular walls, the hydræmia, and the associated cardiac weakness. Sometimes there also occur marked pains in the arms and legs. The temperature is normal, or even subnormal. If there is some inflammatory complication, or if the anæmia is extreme, fever may occur.

The entire duration of the disease may be one or two years. It is exceptional for it to last longer, except when the cancer develops in the floor of a preexisting ulcer. In this case the symptoms of gastric ulcer pass into those of carcinoma. The change may be gradual, but there may be an intervening period of apparent health. We have by careful questioning repeatedly been able to make the diagnosis of this transformation during the life of the patient, in cases later confirmed by autopsy. In individual instances the disease, of course, exhibits many variations and departures from the typical course. Sometimes the constitutional symptoms of weakness and emaciation are more prominent, and sometimes the distinctively gastric disturbances.

The fatal termination is usually preceded by the symptoms of constantly increasing weakness. It may be hastened by complications, such as perforative peritonitis. Now and then grave nervous symptoms appear, often quite suddenly. The patient falls into a condition resembling that of diabetic coma (*q. v.*), he is somnolent, and has a peculiar dyspnœa, with deep and labored respirations. Such an attack is probably due to auto-intoxication, and almost always ends fatally. Recovery from cancer of the stomach is unknown.

Diagnosis.—Gastric cancer is a comparatively frequent disease, and in every case in which there are well-marked gastric symptoms, particularly in an elderly person, the physician should think of the possibility of this grave disease. The suspicion is all the more justified when the symptoms occur in a previously healthy individual without known cause, and if they are associated from the start with a cachectic look and a feeling of weakness and languor, as well as with marked emaciation. If now, having such suspicions, we wish to reach a diagnosis, the first requirement is a thorough investigation of the stomach. External examination should be particularly directed to the discovery of a palpable tumor, if any exists. We should examine while the patient is lying on his back or on his side. If possible, the stomach should be empty and we should palpate while the patient is breathing quietly, and also while he is taking deep inspirations. If a tumor can be felt, the next question is whether it actually arises from the stomach, or from some other organ. In most cases the other clinical symptoms, such as vomiting, have already indicated the stomach as the seat of the disease, and thus we know the origin of the tumor. On the other hand, however, there may be great obscurity, and many cases of gastric tumor have been confounded with carcinoma of the left lobe of the liver, the pancreas, the omentum, the transverse colon, and other parts. It is difficult to lay down special rules for diagnosis, because the difficult cases almost invariably have their own special peculiarities. Of importance in every case are an accurate local examination, a mapping out of the neighboring organs, inflation of the stomach and perhaps also of the colon, determining whether the mass moves with respiration, and a consideration of all the other factors. Among these last the most important is the result of an examination with the stomach-tube. The main question in every case is the presence or absence of free hydrochloric acid in the gastric juice. If it is absent on repeated examination our suspicion of carcinoma is confirmed, provided the other symptoms point in the same direction; but if they do not, the absence of free hydrochloric acid is by no means so grave a matter, for it is absent often enough under other circumstances. It is, however, a fact of great practical importance that when free hydrochloric acid has been distinctly and repeatedly demonstrated in the gastric juice, we can usually absolutely exclude gastric cancer, even if other symptoms point to it. If there is no free hydrochloric acid, but lactic acid, and also impaired motor efficiency, the circumstances are different. In such a case, even if no tumor can be felt, there is great probability of a gastric carcinoma, probably involving the pylorus. Another very important point is the demonstration of blood in the gastric contents (*vide supra*, page 422). If we have distinct "coffee-ground" vomitus, and at the same time achlorhydria, these factors alone almost establish the diagnosis, but even smaller amounts of blood obtained by lavage are important. Finally, we should bear in mind that when no gastric tumor is made out there may be metastatic growths which can be felt—for example, in the lymph-glands—and thus a diagnosis of cancer confirmed.

In case of symptoms which indicate stenosis of the pylorus, with subsequent gastrectasia, it is of great practical importance to determine whether they are due to cancer or to the scar of an ulcer. In this instance, even if we feel a tumor, that fact is not decisive, for the cicatricial hyperplasia of gastric ulcer may cause a distinct tumor; but, as a rule, it is not difficult to reach a decision. Suggesting a scar from ulcer would be: long duration of the disease (two years, sometimes four or five years, and longer); youth of the patient; comparatively healthy appearance; distinct history of ulcer; cardialgia; profuse hæmatemesis; and finally, as a decisive factor, the demonstration of an abundance of hydrochloric acid and hypersecretion in the stomach. Under these circumstances the differentiation of stenosis from ulcer is absolutely certain. On the other hand, there is complete certainty of a cancerous stenosis if the disease has developed in a comparatively brief time—in one or two years; if it has attacked an elderly person; if there is distinct cachexia; and, most important of all, if hydrochloric acid is never demonstrable in the gastric contents, while there is an abundance of lactic acid. Other cases occur which are not typical and of easy diagnosis. Age, duration of the disease, and previous symptoms may suggest a stenosis due to ulcer, and yet on washing out the stomach we find no hydrochloric acid at all, or, at most a faint trace. In these cases we are usually justified in assuming that carcinoma has developed secondarily on the floor of a former ulcer. It must be confessed that in simple stenosis following ulcer, when there is an excessive stagnation of the gastric contents, there may be temporarily a complete combination of all the hydrochloric acid with albumen, and consequently no reaction for free hydrochloric acid; but if the stomach is washed out a few times we soon find an abundance of free hydrochloric acid. On the other hand, when a stenosis due to ulcer has lasted for a long time, our suspicions should be aroused if the reaction for hydrochloric acid is persistently feeble or at times absent, and in its place or along with it lactic acid appears in the gastric contents. We have ourselves repeatedly seen this gradual transition of simple stenosis, due to ulcer with hypersecretion, into carcinomatous stenosis with achlorhydria. Finally, it should be remarked that there is also a stenosis of the pylorus due to simple hypertrophy of its walls, without any ulcer or carcinoma. We have ourselves seen a few cases of this sort, which presented the ordinary symptoms of pyloric stenosis with great dilatation, and ended fatally. The pathogenesis of this rare condition has not yet been explained. We venture to suggest that perhaps it may be due to congenital anomalies. As yet, unfortunately, no particulars are known as to the constituents of the gastric juice in these cases, and for the present it is scarcely possible to make a diagnosis of them with certainty.

Treatment.—If once the diagnosis of gastric carcinoma has been settled, the question arises whether there is any definite prospect of success from surgical treatment. If not, our treatment can be merely symptomatic with the object of alleviating the patient's suffering, and preserving his strength as long as possible.

The first point in symptomatic treatment is a regulation of the diet. In general, we advise mainly liquids and soft foods, including milk, gruel, rice, sago, delicate vegetables, potato purée, softened zwieback, toast, or water-crackers, and delicate puddings. We may also permit finely chopped or scraped meat, or, still better, fowl; and fish, such as trout, pike, and perch-pike. Such artificial foods as somatose, neutrose, beef peptone and beef juice, and infants' foods, do good service for a time. If the appetite fails we order stomachics—for example, compound tincture of cinchona. The fluid extract of condurango deserves a special mention, and has been a favorite prescription in gastric carcinoma since condurango bark was recommended by Friedreich. We usually prescribe hydrochloric acid after meals (15 to 20 drops of the dilute acid in water) in the hope of making up for the achlorhydria. Pain is alleviated by narcotics, such as morphine, opium, codeine, belladonna, and chloroform, and by moist warm or cold applications. Warm poultices on the epigastrium for several hours a day are usually beneficial. To combat obstinate vomiting we may give the narcotics just named or bits of ice, but the best remedy is rinsing out the stomach, provided the patient is not too feeble. If there are sour or offensive eructations, we prescribe bicarbonate of soda, magnesia, or powdered charcoal. If the pyloric cancer is leading to stenosis with stagnation of food in the stomach, the daily washing out of that organ is an excellent means of relieving the patient for a time, at least, of a great part of his discomfort. If this is practiced regularly with either pure water or a 1- or 2-per-cent. solution of hydrochloric acid, and if the diet is suitable, there may be a great temporary improvement in the condition and even a not inconsiderable gain in weight. But, despite all our efforts, the symptoms may become worse and worse till, finally, the subcutaneous injection of morphine may be indispensable. Then our whole object is to alleviate the patient's sufferings, and to give him moral support and encouragement.

When the vigor of the patient is such as to permit the consideration of surgical interference, the more promptly the operation is undertaken the more chance is there of eradicating the disease and obtaining permanent recovery. It is, therefore, permissible when there is incipient disease and the diagnosis is not yet fully established, although somewhat probable, to propose to the patient an exploratory laparotomy, to be immediately followed by a regular operation, provided the case turns out to be a suitable one. The results of operations on carcinoma of the stomach have not been very brilliant. Still, in many cases the surgeon is fully justified in endeavoring to combat an otherwise absolutely hopeless disease, since there is some prospect of recovery. Although there have been many failures, surgeons have obtained a number of extremely satisfactory recoveries under these circumstances. If there is a stenosis of the pylorus and yet it is no longer possible, or at least no longer wise, to extirpate the tumor, gastro-enterostomy may give very great relief. If the patient survives the operation, the suffering is much diminished and nutrition may decidedly improve. For the particulars we refer to surgical treatises.

CHAPTER VII

ANOMALIES OF THE SECRETION OF GASTRIC JUICE

(Achylia Gastrica and Hypersecretion of the Gastric Juice)

IN the present chapter are to be considered those dyspeptic conditions which are associated with a change—that is, either a diminution or an increase—in the secretion of the gastric juice, independently, at least in most cases, of any definite anatomical lesion. We have here to consider, therefore, no marked catarrhal condition or new growth of the stomach, nor gastric ulcer with hypersecretion. The cause of the change in secretion in the cases here considered must rather be sought in the disturbances of the activity of the cells, the special occasion of which is as yet almost entirely unknown. Many clinical observations, it is true, indicate that abnormal nervous influences are important in this connection, but, on the other hand, it is possible that the explanation lies in primary changes in the minute structure and chemistry of the secreting cells themselves. Besides this uncertainty of ætiology, there is also an absolute lack of thorough anatomical investigations in this regard, so that it is at present a very difficult matter to map out and depict accurately these abnormal conditions. We must, therefore, limit ourselves preliminarily to a simple statement of the clinical facts thus far known, and present, as well as we can, the leading points of view for the interpretation and treatment of these groups of symptoms.

1. **Anacidity of the Stomach (Achlorhydria).—Achylia Gastrica.**—Fenwick was the first to observe that there are cases of chronic and complete absence of any demonstrable secretion of gastric juice, independently of chronic gastritis or gastric carcinoma, but associated with an almost complete atrophy of the glandular apparatus of the gastric mucous membrane. The clinical picture in these cases displayed scarcely any marked gastric symptoms. Anorexia and moderate oppression after eating were almost the only indications of dyspepsia; but there were observed gradually progressive anæmia and emaciation, so that finally the general weakness, independent of any other local symptoms, resulted in death. Since then we have learned, particularly from the investigations of Martius, that the grave symptoms of these cases can not possibly be caused by an atrophy and cessation of function of the gastric glands alone, but occur only when there is also present an atrophy of the intestinal mucous membrane.

It has been shown, and we have ourselves repeatedly found, that it is possible to have an achylia gastrica—that is, persistent absence of hydrochloric acid (and pepsine, *vide infra*), without any noticeable disturbance of nutrition, and with only slight and fugitive dyspeptic symptoms, or even with no gastric symptoms whatever. In all these cases it is wholly probable that the digestion and absorption by the intestines are perfectly normal, and completely replace the digestive function of the stomach. It is only requisite that the intestines should preserve their normal function, and that the motor power of the stomach should be unimpaired. In such cases the stomach serves to a certain degree merely as a reservoir for the ingested food, which it duly passes on to the intestines; in them are present in abundance all the reagents, bile, pancreatic juice, and intestinal juice, requisite for the further absorption of the ingesta. There is only one defect in these persons with achylia gastrica: there is no disinfection of the gastric contents by means of the hydrochloric acid of the gastric juice. Ordinarily this lack is unimportant, but if any hurtful material reaches the stomach it is more likely to lead to disturbance than under normal conditions. This explains why those persons with achylia gastrica have a "sensitive" stomach and are prone to suffer from temporary dyspepsia. They very often also have a striking tendency to diarrhœa. But if such individuals live cautiously and are not exposed to any especial injurious influences, they feel perfectly well and may be extremely well nourished.

Is there an atrophy of the gastric mucous membrane as a foundation for these benign cases of achylia gastrica? It is possible, and in many cases even probable, that there is; but it is not certain. This is certain: that sometimes a long persistent absence of hydrochloric acid has been observed clinically, and later an autopsy has shown no anatomical explanation of the condition. Thus, for example, a short time ago the author had a patient under observation in his wards for a considerable time with diabetes mellitus, in whose gastric juice no trace of hydrochloric acid could be demonstrated, although repeated examinations were made. The patient died, comatose, and the stomach seemed to be perfectly normal, even upon microscopic examination. Here there may perhaps have been some toxic inhibition of the secretion. At any rate, observations like this prove that when there is no gastric juice secreted, we can not assert that any marked anatomical change will be found in the gastric mucous membrane.

From what has been said it follows that, according to the present state of our knowledge, we must distinguish several forms of achylia gastrica (or of long persistent gastric anacidity). We should mention incidentally that in a strict sense the name *achylia* should be employed only for that condition in which there is a constant absence not only of hydrochloric acid, but also of pepsine, indicating a complete drying up of the secretion of gastric juice. Those cases, in which free hydrochloric acid is never demonstrable but pepsine is found, should, in strictness, be termed anacidity, or, better, *achlorhydria*. In practice it is true that we can not always make these fine distinctions, and that even such cases as present not an absolute absence but a slight or occasional trace of hydrochloric acid are put in this latter category. We distinguish the three following forms of achylia:

1. Achylia or achlorhydria may appear as a symptom in certain other diseases of the stomach, particularly, as we have already seen, in severe acute and chronic gastritis (when there is genuine catarrh of the stomach), and also in cancer of the stomach. These cases are to be termed "symptomatic achylia."

2. Achylia or achlorhydria may appear as a necessary result of marked atrophy ("anadenia") of the gastric mucous membrane. Here the further point is to determine the causes of this distinctly characterized anatomical change in the gastric mucous membrane. Sometimes it seems to be the result of a previous catarrhal inflammation (atrophic catarrh, analogous to similar conditions of other mucous membranes—*vide supra*, page 426). How far this assumption agrees with the facts must be determined by further careful investigation.

Apart from this secondary or inflammatory atrophy there is also a simple primary atrophy of the mucous membrane. The cause of this condition is most likely to be found in congenital peculiarities, of which we as yet possess no intimate knowledge. We might see a certain analogy in progressive muscular atrophy. In these cases of atrophy of the gastric mucous membrane there is no demonstrable secretion of hydrochloric acid, nor of pepsine. The gastric contents obtained in the ordinary way are absolutely devoid of peptic activity, but at the same time the muscular efficiency of the stomach is in many cases perfectly normal, and this explains why, although there is no secretion of gastric juice, there are no marked symptoms of gastric discomfort. In many cases, however, the atrophy of the gastric mucous membrane is coincident with an atrophy, or at least with marked changes of the intestinal mucous membrane. Then it is inevitable that severe disturbances of nutrition should gradually develop, for the food can not be sufficiently digested and absorbed. Such patients grow progressively thin and feeble. Often they suffer from marked intestinal symptoms, having persistent diarrhœa, alternating with constipation. By suitable treatment and nursing such patients may improve, or at least their symptoms may be checked. In a few rare cases death finally occurs. The clinical picture of pernicious anæmia (*q. v.*) has been repeatedly presented in association with this sort of atrophy of the stomach and intestines. In our opinion, however, the nature of these cases is still unsettled, since the atrophy of the intestinal mucous membrane of itself might well lead to extreme inanition, but not to a specific anæmia. For this there must be present some special circumstances with which we are as yet unacquainted.

3. The third form is simple functional achylia. Here belong the not infrequent cases in which the symptoms are not usually particularly severe, consisting of anorexia, gastric oppression, nausea, and rarely vomiting. The complaint may have been of recent origin. On careful examination of the gastric contents, we find complete absence of hydrochloric acid, while pepsine may be present, or, again, in other cases entirely absent. The motility of the stomach is normal, as a rule, but it may be somewhat impaired, probably because of a coincident weakness of the muscular coat (see the following chapter). With suitable treatment the symptoms diminish or vanish, but there is no change with regard to the absence, or approach to absence, of hydrochloric acid and pepsine. It can not be determined how long this condition has existed. Sometimes such patients say, as we have already mentioned, that they have been rather subject to mild dyspepsia or to temporary attacks of diarrhœa. Often, however, their previous health has been good. Whether we should invariably assume that there is a marked atrophy of the gastric mucous membrane in all such cases is very questionable. Probably we have rather to do with an impairment of the secretory capacity of the individual stomach from causes not at present accurately determinable. Apparently there are not a few persons with achylia of this sort. Inasmuch as the condition runs along without any special symptoms, it is not diagnosticated by the physician, and the patient is not subjected to an accurate examination until digestive disturbance arises because of some special cause (*vide supra*), and then the anomaly is detected. If such patients do suffer from persistent dyspepsia there will usually be found some special reason for it, such as motor incapacity, possibly dislocation of the stomach, or genuine "nervous" dyspepsia, meaning by that term dyspepsia of psychical origin. It would be an interesting question to investigate whether individuals with persistent achylia do not perhaps have a predisposition to the development of carcinoma.

The **diagnosis** of achylia, whether essential or secondary, can always be easily made by means of a careful examination of the gastric contents (see the first chapter of this section). Of course, the diagnosis is not established until repeated and painstaking examinations have been made at various times and under various circumstances, and have invariably given the same negative result. If achylia is demonstrated, the next question is whether it is merely symptomatic or essential. The presence of chronic gastritis is excluded by the absence of all ætiological factors and of excessive mucous secretion, and by the general course of the disease. The suspicion of cancer will often likewise be rendered untenable by the general course of the disease, and the absence of a tumor. In cases of atrophy of the gastric and intestinal mucous membrane, with consequent emaciation, there may be very great difficulty in excluding cancer. On the whole, however, the course of gastro-intestinal atrophy is much slower than that of carcinoma. Accompanying signs of pyloric stenosis invariably indicate the presence of carcinoma; and if we are enabled by the absence of any severe disturbance of nutrition to exclude any marked atrophy of the gastric and intestinal mucous membrane, we reach at last a diagnosis of simple functional achylia by exclusion. Whether in such cases there is a demonstrable anatomical change in the gastric mucous membrane, the present state of our knowledge does not enable us to determine.

We do not need to enter upon a special discussion of the **treatment** of symptomatic achylia. The severe cases of achylia which occasion extreme disturbance of nutrition need the most careful dietetic management. They should have such food as milk, soups, eggs, and meat-peptones. Practical experience will usually be a better guide than any theoretical considerations. Internally we may try hydrochloric acid and preparations of pepsine and pancreatine, also all sorts of stomachics, including nux vomica and condurango. The stomach may be rinsed out with a 2-per-mille solution of hydrochloric acid, and galvanic electricity may be employed. It is self-evident that when there is actual atrophy of the mucous membrane, the results of treatment must be very limited. The milder cases of achylia do not need treatment except when special dyspeptic symptoms appear, or when there is intestinal disturbance, such as diarrhœa; then the treatment is the same as in ordinary disturbances of the stomach or bowels, including a suitable diet, hydrochloric acid, perhaps rinsing out the stomach a few times, and, in case of diarrhœa, opium. If the symptoms subside, and we now find there is a persistent achylia, of course we must earnestly recommend such individuals to exercise a certain caution with regard to diet. They should not take too great a quantity of food at one time, and in general should choose a largely vegetable diet—i. e., one containing an abundance of starches. There is this exception, that if the achylia is associated with a dread of eating and a tendency to hypochondriasis, we must encourage the patient to take a rather abundant amount of nourishment and employ hydrotherapy and other general tonics.

2. **Hypersecretion and Hyperacidity of the Gastric Juice** (**Acid Dyspepsia**). —We have seen in the first chapter of this section that it may be regarded as a rule, that when fasting the stomach is almost completely empty, and that an hour after a test breakfast (*vide supra*, page 418) the acidity of the gastric contents is about 55 to 65. The investigations of late years have shown, however, that this rule has numerous individual exceptions not necessarily pathological. There are many persons who secrete scarcely any hydrochloric acid (*vide supra*), and also there are apparently still more who always have hydrochloric acid in the stomach even when fasting, and whose hydrochloric-acid index after a test breakfast is as high as 80 to 100, or even higher. Many such individuals have no dyspeptic disturbances whatever, so that the unusual abundance of hydrochloric acid can be regarded only as an individual peculiarity of secretion, and not due to disease. In other such cases, however, there are decided gastric symptoms, and these symptoms are such as to indicate very strongly their direct dependence

upon the increased production of acid. This state is termed acid dyspepsia, by which we mean the abnormal excess of hydrochloric acid at the time of digestion—that is, hyperacidity as distinguished from hypersecretion, which latter term is applied to the continuous secretion of gastric juice even when digestion is not taking place, the stomach having received no food, or having discharged its contents into the pylorus. Hypersecretion is probably almost always associated with hyperacidity, but not *vice versa*. However, we can not maintain a strict clinical distinction between the two in practice.

Essential, acid dyspepsia should be sharply distinguished from symptomatic hyperacidity and hypersecretion, such as occur perhaps in certain forms of gastritis (page 428), and almost invariably in ulcer of the stomach. This distinction is very important, even from a practical point of view. It must be confessed that it may be scarcely possible to make a differential diagnosis, as we shall soon see, between ulcer and hypersecretion; but we can, at any rate, hold fast to the important theoretical distinction between the two conditions. The term "essential" hypersecretion ought to be applied only to cases in which there is an increase in the secretion of hydrochloric acid, without any ulcer formation. If we regard hypersecretion as a possible cause of ulcer (*vide supra*, page 433), then ulcer can be termed merely a complication of hypersecretion.

As to the causes of simple hyperacidity and hypersecretion we have as yet, unfortunately, scarcely any knowledge. Very few histological examinations of the mucous membrane in characteristic cases have thus far been made. It is, therefore, at present customary to assume that hypersecretion is a "neurosis," and this view is apparently confirmed by the frequent combination of hypersecretion with other nervous conditions. We must later discuss this point more fully. On the other hand, however, it is not impossible that functional disturbances of cellular activity may lead to increase in the secretion. We are ourselves inclined to class many cases of hypersecretion along with the well-known cases of increase in the secretion of sweat (hyperidrosis of the hands and feet), of saliva, and of similar products. For such it is not at all essential that there should be abnormal nervous conditions.

Having such imperfect insight into the true nature of these processes, we must provisionally take a purely clinical standpoint, and describe the phenomena seen in practice. It should be added that some of the conditions are by no means infrequent, but it is often difficult to interpret accurately what we observe. One important question is how far the symptoms which are present are actually referable to the hypersecretion as such, and how far they are due to the other anomalies which often accompany the hypersecretion, such as general nervousness, gastroptosis, and chlorosis. In order to get a better comprehension of the subject we shall distinguish in what follows several forms of acid dyspepsia, but we wish to repeat that a perfect differentiation is impossible, particularly between hyperacidity and hypersecretion.

1. *Dyspepsia with Hyperacidity* (*Hyperchlorhydria*).—The conditions which belong in this category are most often found in young females. They are very often conjoined with symptoms of chlorosis, general nervousness, etc. Certain errors in diet may also excite hyperchlorhydria, such as hot or highly spiced food, and particularly too strong coffee; and dyspeptic conditions with hyperchlorhydria are frequently observed in excessive smokers.

The symptoms are tolerably characteristic. They occur especially after eating, and sometimes only after certain kinds of food, and consist first of a sense of oppression in the stomach, often increasing into genuine cardialgia. Usually the pain does not appear until two or three hours after eating—i. e., at a time when the stomach is already empty, although the excessive secretion of acid is still going on. Many patients have noticed that if they then take some more food or drink their pain will abate. This is easy to understand. Often there will be acid eructations at the time of the excessive formation of acid, and a distinct feeling of heartburn. The acid gastric contents may be vomited, but this is not especially frequent. When the stomach is completely empty, the patient feels perfectly well again. Inasmuch as the appetite is usually unimpaired, nutrition remains fairly good. The general course of the disease is chronic, with marked variations. At many times the symptoms are aggravated, particularly under the influence of psychical disturbances, such as anger or excitement, or because of an unsuitable mode of life. At other times the symptoms may entirely vanish.

The diagnosis of hyperchlorhydria may be suggested by the symptoms just described, but it can not be established except by means of the stomach-tube. External examination of the stomach shows nothing special except that there may be a moderate diffuse tenderness on pressure. It is not surprising that often there is gastroptosis. If we examine the stomach when fasting, it is empty or almost empty, provided the case is one of pure hyperchlorhydria. The acid index after a test breakfast is very high, 70 to 100 or over. The digestive power of the gastric juice is greater than normal. It is therefore natural to find, as we do find, that the stomach is entirely empty as a rule as soon as three or four hours after a test meal; which indicates also a good degree of motor power. At that time hydrochloric acid is still present. On the other hand, digestion of starch in the stomach is impeded by the abundance of hydrochloric acid.

If we find things as above depicted, the diagnosis of hyperchlorhydria as such is established. But a difficult question remains to be answered—whether the case is one of simple functional increase of the secretion, or whether the hyperacidity is symptomatic of an ulcer. If there are no characteristic symptoms of ulcer, such as gastric hæmorrhage and localized tenderness, it is often impossible to answer this question with more than a certain degree of probability. It is characteristic of simple hyperchlorhydria that the pain is often improved upon taking food, while in ulcer the pain is aggravated. If there are well-marked symptoms present of general nervousness, this is usually decidedly in favor of "nervous hyperacidity." Finally, the results of treatment may aid us in diagnosis (*vide infra*).

The mode of treatment will be considered with that of hypersecretion.

2. *Dyspepsia with Hypersecretion of the Gastric Juice* ("*Continuous Flow of Gastric Juice*").—Here belong those cases of dyspepsia in which the examination of the stomach shows the presence, even when fasting, of considerable amounts of fluid containing an excess of hydrochloric acid, and in which this hypersecretion can not be regarded as the result of an ulcer.

The disease is much more exceptional than simple hyperchlorhydria. It appears in men somewhat oftener than in women. No special ætiological factors can be named, except long-continued mental strain and excitement; often no cause can be made out.

The **symptoms**, as in hyperchlorhydria, consist of cardialgia, sour eructations, heartburn, and not infrequently vomiting. Pain often appears when no food has been taken. When vomiting occurs there may be little or no food in the vomitus, but merely a cloudy, often bile-stained fluid, strongly acid, with a very sharp taste and so irritating that it causes burning pain in the pharynx. The appetite is generally good, and the patients have usually found out by experience that if they drink some tea so as to dilute the gastric juice, or take some sort of food, they get relief. Often there is abnormal thirst; the bowels are usually constipated; the urine faintly acid and turbid with deposits of phosphates; the pulse is often slow; in many cases there is a distinct general neurasthenic condition, associated with the dyspepsia. Here, again, the only method of arriving at a certain diagnosis is by examining the gastric contents. The decisive point is that in the morning before breakfast we should invariably find a considerable amount of fluid, containing practically no portions of food but a large percentage of hydrochloric acid. At the same time it should be particularly pointed out that it is not abnormal, now and then, to find a teaspoonful or two of fluid in the stomach, containing hydrochloric acid, while fasting. It is not proper to speak of the case as one of hypersecretion unless we can obtain from the fasting stomach, without special effort, some two or three ounces (50–100 cubic centimetres) of strongly acid fluid. Along with the hypersecretion there is usually hyperchlorhydria, so that we find a high acid index an hour after the test breakfast. If we give a test meal, and examine three hours later, we shall find that the meat has vanished, while the digestion of starches has been decidedly delayed by the hyperacidity. Some seven hours after the test meal the stomach is empty of food, but probably contains an abundance of acid secretion.

If we inflate the stomach, we may find the organ dislocated downward (gastroptosis). This, however, is merely a chance complication. There is never great dilatation. If we find the stomach much enlarged, our suspicions are at once directed to pyloric stenosis, and this condition is usually easily diagnosticated, one important indication being delay in the emptying of the stomach. Whenever the stomach in a fasting condition, or seven or eight hours after a test meal, is found to contain not only an abundance of hydrochloric acid, but in addition considerable amounts of partially digested food, the condition is not one of simple hypersecretion, but of hypersecretion combined with stenosis of the pylorus due to ulcer. This puts an entirely different aspect on the case.

It is a much more difficult matter to distinguish hypersecretion from a gastric ulcer without stenosis. Indeed, sometimes an absolute decision is almost impossible. In such instances we may be enabled to form an opinion by considering the general course of the disease, the associated nervous symptoms, and all the special symptoms (*vide supra*).

In general, the course of acid dyspepsia with hypersecretion is chronic, but variable. The prognosis is not unfavorable, and correct management, based, as it must be, upon an accurate examination, may bring about very satisfactory results.

In the **treatment** of acid dyspepsia, including hypersecretion as well as simple hyperchlorhydria, we meet with one difficulty in the fact that we can seldom absolutely exclude ulcer of the stomach. The physician must, therefore, determine whether he shall begin with a course of treatment for ulcer or not. We believe that in all cases in which the symptoms have existed for a considerable time and are severe, and in which there has been no systematic treatment as yet, it is wise to treat the patient as if there were an ulcer. If the case is one of simple acid dyspepsia this can do no harm, and if it is one of ulcer it is the only method that can do good. In either case, the quiet, the unirritating diet, and the administration of alkalies are certainly appropriate. Moreover, cases are not infrequent in which the patients are anxious and hypochondriacal, and upon them a methodically conducted " cure " for ulcer often has a favorable subjective influence. Still, this just-mentioned combination of acid dyspepsia with decided nervous and hypochondriacal disturbances may, on the other hand, render the effect of a strict ulcer treatment quite unfavorable. This applies to patients who have long been tortured by a dread of gastric ulcer, have consequently gone through the appropriate treatment many times already, and have grown thin and feeble as the result of an excessively cautious dietary. For such unfortunates there may be no use whatever in repeating a strict course of treatment suitable to ulcer. Such a procedure confirms the patient in his apprehensions, and merely makes him more wretched.

We see, then, how carefully we should consider the individual in the treatment of acid dyspepsia, and, above all, how much weight we should assign to the general condition as well as to the dyspeptic symptoms. It is often advisable to begin with a course of treatment for ulcer in the ordinary manner, but upon the prompt cessation of symptoms to make a more rapid transition than usual to nourishing food and other therapeutic methods. The more convinced the physician becomes that he has to do with simple "nervous" hypersecretion and not with an ulcer, the less need has he to pursue a strict dietetic treatment. In such cases it is best to prescribe for the patient a diet list, containing an abundance of albuminoids, including meat, fish, eggs, and milk; and with this, rye bread and butter, and easily digested vegetables. There should be as great a limitation as possible of alcoholic beverages, spices, strong coffee, and sour articles, as well as of puddings and starchy vegetables. Yet in such cases, also, one must be guided by the individual experiences of the patient as well as by merely theoretical considerations. As to drugs, of course alkalies come first. We should be guided by the circumstances of the particular case in prescribing, several times a day, particularly after meals, a knife-point full of bicarbonate of soda, or some similar powder, such as bicarbonate of soda and calcined magnesia, equal parts; or bicarbonate of soda, 30 parts; subnitrate of bismuth, 5 parts; and similar drugs. If there is hypersecretion we like to give the patient in the morning, before breakfast, a half litre (pint) of warm Carlsbad water, or the artificial Carlsbad salt, dissolved in water. In severe cases of hypersecretion regular washing out of the stomach is advantageous. The best time to do this is in the early morning. We may employ a one-per-cent. solution of bicarbonate of soda.

Atropine and the narcotics, such as morphine and codeine, have been recommended for the purpose of diminishing the secretion of the gastric glands. We have ceased to expect any benefit from their use, and we would warn the physician against their unnecessary employment.

The simultaneous constitutional treatment is very important, particularly in all cases associated with neurasthenic symptoms (*vide infra*, the chapter on nervous dyspepsia). From this point of view there is much benefit in drinking the waters at Carlsbad, Tarasp, and similar watering places, in connection with the hope of constitutional improvement from the outdoor life, the baths, cold sponging and rubbing, and similar agencies. In treatment at home these last factors must always be duly considered.

3. *Periodical* (*Intermittent*) *Hypersecretion* (*Gastroxynsis*).—In the condition here to be described, persons who at other times feel perfectly well, without any digestive disturbance, are rather suddenly attacked by violent gastric pain, sour eructations, and vomiting. The intervals between attacks may be longer or shorter, and there may be either some exciting cause or no apparent reason for the disturbance. The vomitus consists of large amounts of watery fluid containing an excess of hydrochloric acid. During these attacks the general condition of the patient is wretched; he looks pale, his pulse is small and frequent. Often there is extremely violent headache at the same time. An attack of this sort lasts a few hours, or even two or three days. Often it ends with considerable suddenness, to be followed by a period of good health which may last a week or even months, or longer.

The causes of this strange disease are entirely unknown, but it certainly seems as if there were conditions of irritation, originating in the nervous system. It seems to us indubitable that there is a close relation between gastroxynsis and migraine (see the chapter on migraine). It is a very important fact that almost precisely the same group of symptoms occurs in tabes dorsalis (*q. v*), under the name of gastric crisis. In every case, therefore, the physician should examine the tendon reflexes and the pupils; but attacks of this sort undoubtedly occur when tabes is absent. It may be added that in its well-marked form the condition is quite rare.

If possible we should examine the gastric secretion in the time between attacks, as well as during the attack. It appears that many cases of periodical gastroxynsis are merely acute exacerbations of a constant hypersecretion. In such cases there are likely to be mild dyspeptic symptoms in the intervals, and a careful treatment of these by lavage, alkalies, and Carlsbad water has an excellent effect upon the paroxysms. If there are coincident neurasthenic symptoms, of course the greatest stress must be laid upon constitutional treatment. The treatment of the paroxysm itself is seldom very successful. The best remedy is bromide of sodium in large doses. We may also try chloral, chloroform, belladonna, codeine, and also antipyrine. Externally, warm compresses or hot poultices may be applied over the stomach. If the pain is very violent a subcutaneous injection of morphine may be almost indispensable, although we should avoid it if possible.

CHAPTER VIII

ABNORMALITIES IN THE SIZE AND POSITION OF THE STOMACH—MOTOR DISTURBANCES OF THE STOMACH

1. **Abnormalities in the Size of the Stomach.—Atony of the Stomach.**—Like any other organ, the stomach may present considerable differences in its size without being pathological. These differences partly depend upon congenital conditions, and partly are associated with the mode of life. It has long been known that certain classes of people who eat very bulky articles of diet (e. g., a great proportion of potatoes), and other large eaters, including many diabetics, have noticeably large stomachs; but a change in size of this sort can not be regarded as pathological, provided the function of the stomach is not impaired. The excessive distention of the organ, or the excessive demands upon the functional activity of the muscular coat of the stomach, may finally render it unequal to its task. Then we reach a pathological disturbance, a sort of overstrain of the stomach, a muscular insufficiency analogous to the myopathic diseases of the heart, which have been so much studied. Obviously one important factor here would be the original quality of the muscular fiber: if the muscles of the stomach are weak from the start, they will become insufficient all the sooner.

We must state that in our experience well-marked cases of primary idiopathic dilatation of the stomach, as an expression of pure muscular insufficiency, seem to be far from frequent, although slight degrees of muscular weakness occur quite often.

From what has been said, it is evident that the physician should lay little stress upon the mere estimation of the size of the stomach. And, indeed, its size varies greatly according to the amount ingested. We can form an opinion of the size of the organ by percussion of the empty stomach, or by percussing it alternately empty and filled with water by means of the stomach-tube, or, what is much simpler and more certain, by distending it with air, as above mentioned (page 420). In general, we can say there is dilatation of the stomach if the lower border of the fundus extends below the level of the navel while the lesser curvature is in a normal position; but, as we have said, the important question is not the size, but the motor ability of the organ, and it is far more important to determine the functional activity by means of a test meal, in the manner already explained (page 420), than to find out the mere size of the stomach. If the organ empties itself in a normal way, any dilatation that there may be has no significance. If there is some tendency to stagnation of the gastric contents because of muscular insufficiency, we may have mild dyspeptic symptoms, such as gastric oppression, eructations, or even occasional vomiting.

If, however, we find that the stomach is dilated and that the motor efficiency is decidedly impaired, with actual stagnation and accumulation of ingesta in the stomach, we ourselves think it is always justifiable to assume that there is a pyloric stenosis. In such case the disease with which we have to deal is not a dilatation of the stomach, but an ulcer or cancer of the pylorus, which leads to stenosis and its results, as we have already described at length (pages 438 and 447). It has often been maintained that in cases of primary muscular dilatation there may occur mechanical kinks in the pylorus and sacculations in the stomach, so as to cause a considerable hindrance to the emptying of the viscus. This may be possible, but we have never seen such a case, nor read a conclusive report of one.

In this place a symptom may be mentioned which is often regarded as a sign of dilatation. We refer to splashing in the stomach when palpation is practiced by a sort of pushing or thrusting motion. Marked splashing is indeed very often audible in cases of actual dilatation due to stenosis of the pylorus, but it is very frequently heard in persons who have dyspepsia, and even in persons who are perfectly well. Children sometimes play at producing splashing sounds in their own stomachs, and by practice acquire great technical dexterity! And in precisely the same way the sound is produced by nervous and hypochondriacal patients, to whom the splashing is a source of anxiety. Splashing occurs when air and fluid are both present in the stomach, provided the abdominal walls as well as the gastric walls are not too tense. In this sense it is, therefore, correct to associate so-called atony of the stomach with the splashing, because it may often be due to a certain laxity of the muscular coat of the stomach, but we should never lay any special stress upon the sound, unless upon examination we find at the same time a distinct disturbance of the motor efficiency of the stomach.

It follows, therefore, that mere dilatation of the stomach is not an independent disease. For example, in practice we may unhesitatingly refer every case of well-marked dilatation with decided stagnation of the gastric contents to stenosis of the pylorus, and of course this latter is to be regarded as the true disease. At the same time, however, there are slight degrees of gastric dilatation, or perhaps a better expression would be idiopathic motor insufficiency of the stomach (muscular atony of the stomach), which occur when there is no reason for believing that there is any anatomical change at the pylorus. We may assume that such motor insufficiency exists when there is a slight but distinct tendency for food to accumulate in the stomach.

In treating this simple muscular atony, we should first regulate the diet. The separate meals must not be too large, but they should be nutritious, and perhaps even somewhat stimulating. Rye bread, small portions of vegetables, and nourishing meat dishes are by no means interdicted. Large amounts of liquid are to be avoided, as they needlessly dilate the stomach. In simple atony, lavage is seldom necessary. At the most, we might at the beginning of treatment completely empty the stomach a few times. Other suitable measures are cold sponging and rubbing of the epigastrium, the application of electricity (particularly the galvanic current) to the stomach, and cautious massage of that region. Among internal remedies, nux vomica deserves to be employed.

Whether there is such a thing as abnormal smallness of the stomach, giving rise to symptoms, is not yet known. Perhaps there may be such a condition in cases in which feeble and ill-nourished individuals, usually of the female sex, can take but a few morsels before they have a feeling of fullness and satiety, and so never become well nourished despite every effort. We may find the stomach very small as a secondary result in many forms of inanition, particularly in association with stenosis of the œsophagus and of the cardia.

2. **Abnormal Position of the Stomach.—Gastroptosis.**—Kussmaul was the first to call attention to the great frequency of the downward dislocation of the stomach. Glénard has written much about it. Either the entire organ may sink down, or, what is more common, the right half alone, so that the organ takes a vertical position. The cause of this anomaly may lie partly in congenital conditions, but in the main it is certainly the purely mechanical result of the pressure of clothing. Gastroptosis is an extremely common phenomenon in grown-up girls and women who wear tightly-laced corsets, and fasten the bands of their clothing snugly about them. It also occurs in men, but far less often. Usually, with the gastroptosis is associated a change in the position of other organs, occurring in the same way. In particular, the right kidney becomes abnormally movable, and consequently readily palpable (see the chapter on movable kidney), and the transverse colon sinks down either in its right half or in its middle portion. Glénard has termed this condition enteroptosis, and ascribed to it an important rôle in the ætiology of nervous dyspepsia and neurasthenia. Well-marked cases of enteroptosis are most frequent in women who have lax abdominal walls because of repeated confinements.

The experienced eye may often recognize, or at least surmise, gastroptosis upon external inspection of the abdomen. One is struck by the collapsed appearance of the epigastrium, where one often feels marked pulsation of the abdominal aorta. Below the navel, on the other hand, is seen a protuberance, the shape of which may quite distinctly suggest the contours of the stomach. At this spot, below the navel, distinct splashing may not infrequently be obtained by proper palpation. An absolute diagnosis of gastroptosis, however, requires the artificial inflation of the stomach (*vide supra*, page 420). When this is done the abnormal position of the stomach is immediately recognized. Of course, the most important point is the demonstration of a downward displacement of the lesser curvature. This is found halfway between the navel and the epigastric angle, or often even at the level of the navel. The downward displacement of the greater curvature alone does not establish gastroptosis, because it may be due to dilatation of the stomach.

Inflation of the stomach is such a simple and reliable method of examination that there scarcely seems any real necessity for other methods. For this reason Einhorn's recommendation of electric transillumination of the stomach, by means of an incandescent light introduced into the organ ("gastro-diaphanoscopy"), is scarcely likely to become generally adopted by physicians. It gives some indications of the position of the stomach, but the conclusions drawn from its use are by no means infallible. The displacement of the kidney, which is often associated with gastroptosis, can be easily recognized by means of palpation. The position of the colon (coloptosis) can be demonstrated by distending the large intestine with air or water.

The question of the clinical significance of gastroptosis is not very easy to answer. In the first place, it should be remarked that, if we pay attention to the subject, we shall find that well-developed gastroptosis is very frequent in persons who have not the slightest symptoms. On the other hand, there are many patients, especially women, who complain of such annoyances as pressure and a sense of fullness, attacks of gastric pain, eructations, and occasional vomiting, and who are found to have a distinct gastroptosis. In such a case we should invariably proceed to test the secretory and motor functions of the stomach. Very often these are perfectly normal, and such a condition scarcely justifies us in assuming offhand that there is a causative relation between the gastroptosis and the patient's symptoms. Gastroptosis is so common that the relation is more likely one of simple coincidence. If we make a searching examination into the patient's condition we shall usually discover that the gastric symptoms are associated with many other symptoms of a neurasthenic kind, and hence are very likely, for the most part, of purely psychical origin (see the following chapter).

Still, it can not be absolutely denied that certain disagreeable sensations in the abdomen and along the digestive tract may be directly dependent upon gastroptosis; and, furthermore, gastroptosis in some few cases seems to occasion a mechanical hindrance to the discharge of the stomach contents, especially because of the vertical position of the organ, so that we have a sense of oppression in the stomach, eructations, and other symptoms; but this motor insufficiency should be proved, not assumed. As a rule, even well-marked gastroptosis is not associated with any disturbance of the motor function of the stomach. There is more likelihood that the habitual constipation, which often accompanies the dyspeptic symptoms of gastroptosis, is at least in part the result of an associated coloptosis.

For the **treatment** of the dyspeptic symptoms which may be present in a case of gastroptosis, we have a most desirable basis in the gastroptosis itself, although the author at least is convinced that its main efficacy is subjective. If gastroptosis has been made out, we must absolutely forbid the wearing of tightly-laced corsets, or too snugly fitting gowns. It is often useful to apply a wide abdominal band above the symphysis. This in many cases fully replaces the more complicated "abdominal corsets" and similar apparatus. With regard to diet, we forbid the taking of large amounts of liquid, and prescribe nourishing food that is not bulky. Particularly when patients are emaciated and apprehensive, we often do the greatest good by ordering a highly nutritious diet. The region of the stomach should be rubbed briskly once or twice a day with cold water or brandy. The employment of faradic and galvanic electricity or massage is particularly indicated when constipation is present.

Of course we must pay attention, as we have said, to the general neurasthenic condition, which is usually also present. In this regard great importance attaches to a suitable mental treatment, in order to free the patient from her exaggerated anxiety and apprehension.

CHAPTER IX

NERVOUS DYSPEPSIA

(Gastric Neurasthenia)

We have repeatedly indicated in the preceding chapter that many disturbances of the gastric functions may be due to changes in the innervation of the stomach, or to actual disease of its nerves; thus some have been inclined to refer the disturbances of the secretion of the gastric juice, such as hyperacidity and hypersecretion, to abnormal conditions of the secretory nerves. Many cases of "cardialgia" have been regarded as pure "neuralgia" of the sensory nerves of the stomach; and also in the domain of the muscular activity of the stomach, authors have distinguished conditions of nervous weakness (nervous atony of the stomach) and of nervous irritability of the muscular coat (so-called "peristaltic unrest" of the stomach; constant rumbling in the stomach and similar symptoms). There is no doubt that in cases of severe organic disease of the nervous system there may be well-marked gastric symptoms as direct consequences of nervous irritation—for example, we have nervous vomiting in cerebral tumor and meningitis, and gastric crises in tabes. It is, therefore, not impossible that there may also be primary diseases of the sensory, motor, and secretory nerves of the stomach with corresponding symptoms, but this is by no means proved, and it is certain that such actually "nervous" gastric diseases are very rare, if they occur at all. At present, clinical experience does not justify us in putting in this category, with any degree of certainty, any disorder except the state above described (page 459), in which there is periodical vomiting with gastric pain and hypersecretion.

There are numerous cases which are at present habitually termed nervous dyspepsia, but in our opinion the overwhelming majority of these have an origin different from the one implied. Such patients assert that after every meal they have pressure and pain in the stomach, that after a few mouthfuls the stomach feels full and distended, and consequently they often have palpitation and a troublesome feeling of constraint in the chest. The patients frequently complain of eructations also, and occasionally of vomiting. These symptoms are all such as occur in the same way in actual diseases of the stomach, but in these patients there are usually certain other symptoms present, which indicate the nervous character of the disease. In the first place, it is noticeable that the above symptoms display striking variations in their severity. The same patient who to-day complains of great distress in his stomach after a few spoonfuls of soup, at another time, when he is in a happy and lively frame of mind, will eat a large dinner without being reminded that he has a stomach; but as soon as he gets angry or irritated, or, in brief, in a "sensitive" condition, the gastric symptoms appear at once in an exaggerated form. On all such occasions, therefore, we see most clearly the influence of the imagination and of general psychical irritation on the behavior of the stomach. Every one knows that a violent rage or any experience arousing anxiety or hope—any excitement, sad or joyful—may immediately destroy the appetite; and very great mental disturbance not infrequently also causes vomiting or similar symptoms. In the same way in sensitive individuals even the mildest psychical influences produce similar results; but in this regard there is nothing which has so unfavorable an influence as that state of psychical disturbance which is associated with great concern for one's own health—that is, a condition of hypochondriacal anxiety—anxiety lest something which has been eaten may do damage; constant dread that a grave disorder of the stomach may be developing. These mental disturbances are especially influential in maintaining and progressively aggravating the abnormal condition. This is the explanation of that peculiar psychical hyperæsthesia which feels the acutest "pain" in the stomach, when there is really nothing but a perfectly normal general sensation. And the same explanation applies to certain muscular contractions—half unconscious, half voluntary—which occasion eructations, vomiting, and similar phenomena. The patient has some symptoms which he feels subjectively, and some which seem to him to be actually objective, and they are in part the result of pure imagination (or "auto-suggestion") and in part the physiologically necessary consequence of the great psychical disturbance.

What we wish to emphasize is our conviction that in the great majority of cases of nervous dyspepsia we are not dealing with any functional disturbance of the gastric nerves, but with abnormal "psychogenic" irritations of the central nervous system, the results of which are apparent mainly in the domain of the gastric functions. Nervous dyspepsia is merely one example of that great group of nervous diseases which owe their origin mainly to hypochondriacal disturbances of mind, and which may appear in the most diverse organs. It is properly nothing but a symptom of the constitutional "nervousness" or neurasthenia, and is therefore termed by many physicians, with absolute correctness, gastric neurasthenia. By careful clinical observation it is easy to demonstrate that almost all the ordinary gastric symptoms may, under favorable circumstances, be excited by purely psychical influences. It is evident that anorexia is often the result of simple mental disturbance, especially when of a hypochondriacal character; but excessive craving for food (bulimia) is certainly in most cases also of purely psychical origin, arising from special imperative conceptions. There is no doubt that the most varied abnormal sensations, ranging from simple oppression to a most acute pain in the stomach, may have a purely subjective origin. The best proof of this lies in the often apparently miraculous effect of remedies which have a purely suggestive and psychical influence (simple reassurance of the patient, hypnotism, and various external and internal remedies). It is very important to know that in many cases also eructations and vomiting have a merely psychical and central origin. They are due to the influence of certain ideas which make the eructations or vomiting appear unavoidable, and the action at first is due to unconscious volition, and finally becomes a sort of diseased habit. Many nervous and hypochondriacal patients who regard themselves as dyspeptics become true *virtuosi* in eructations and vomiting, the reflex process becoming easier and easier because of repetition, and being excited by unconscious volition. General psychical disturbance may also cause eructations and vomiting by direct stimulation. We know persons who are attacked with nausea and vomiting upon any great excitement. Even in children it is by no means rare to observe persistent vomiting after almost every meal, and that this is of a purely "nervous" character appears from the rapid beneficial effect of exclusively psychical treatment—e. g., if the child is strictly forbidden to vomit. We often see just the same condition in adolescent girls, frequently associated with other hysterical symptoms. In this connection it should be briefly mentioned that there may be a "nervous"—that is, hysterical—hæmatemesis. (For particulars in regard to this, the reader is referred to the chapter on hysteria; and, in general, the chapters on hysteria and neurasthenia should be referred to as supplementing what has been said here.)

Very often other nervous symptoms are observed besides those referred to the stomach. There are signs of excessive mental irritability, symptoms referable to the head, such as headache, pressure in the head, vertigo; abnormal sensations in the extremities, of pain, coldness, numbness, and the like. There are almost invariably, also, certain attendant intestinal symptoms. The patient complains of bloating of the abdomen, and of irregular and sluggish bowels.

Nervous dyspepsia becomes a more serious matter when the patient is prevented from taking sufficient nourishment, by fear of committing errors in diet, and because of the loss of appetite which his mental uneasiness occasions. There are sometimes complete nervous anorexia, a constant dread of eating or an actual aversion toward any food, and a considerable or excessive emaciation, usually associated with great bodily weakness. Such patients finally take to their beds and give the impression of severe illness.

Diagnosis.—In many cases the experienced physician can make an extremely probable diagnosis of nervous dyspepsia merely from the symptoms which the patient enumerates. The true condition is suggested by the general nervousness, by the prominence of anxiety and of hypochondriacal notions, the variability of the symptoms and their relation to psychical irritation, and the other attendant symptoms of a nervous character, such as headache, vertigo, palpitation, and a sense of oppression. Still, it is easy to be deceived, because the symptoms seem to be so objective that the physician feels constrained to believe that there is some organic disease of the stomach, although none exists, and because, on the other hand, an actual disease of the stomach may be present in a very nervous individual and fail to be recognized, being wholly obscured by the general nervous phenomena. For this reason, even in cases which seem to be the most simple, we would do well to make a careful objective examination, and this is of course an absolute necessity in all cases which are obstinate and severe.

Often the objective examination shows clearly normal conditions in every respect: the external examination is normal, and the position, secretion, and motility of the stomach are normal. In such a case the diagnosis is established, and the mere result of examination will often have a most beneficial effect upon the patient. For this reason a painstaking examination is often the best remedy for many patients with nervous dyspepsia. It is more difficult to reach a conclusion when certain abnormalities are found, although there are no signs of indubitable organic disease—that is, there is no tumor nor evidence of stenosis of the pylorus nor genuine hæmorrhage—but we do find, for example, distinct hyperacidity or hypersecretion; less often achlorhydria; very frequently gastroptosis; and often, it is said (although, according to the author's experience, this is rare), some slight disturbance of motility, so-called atony of the muscular coat of the stomach. How shall such cases be interpreted? Shall we always lay stress exclusively upon the objective anomalies which are discovered, and refer to them every one of the many symptoms the patient enumerates? In the author's opinion, this would often be erroneous. It has been especially emphasized in the preceding chapters that the conditions mentioned are frequent and are apt to cause no symptoms at all, so that if we find associated with well-marked nervous dyspepsia a gastroptosis, or a moderate hypersecretion, or achlorhydria, in many cases the association is a mere chance coincidence without significance. We think that such conditions should not be wholly disregarded, but yet their clinical importance should not be overestimated; and from a therapeutic point of view, also, they should be borne in mind, but never to the neglect of that general psychical treatment which is usually much more important. Such conditions also are very useful as a handle for mental therapeutics (as we have already pointed out), because of the favorable effect which treatment of them has upon the mind of the patient.

A most difficult question in diagnosis, in our opinion, is to distinguish between ulcer and nervous dyspepsia when there is distinct hypersecretion without evident signs of ulcer (see page 458). In such a case all the separate symptoms must be duly considered, and yet we may be obliged to await the further development of the disease and the results of treatment before arriving at a conclusion. Patients with nervous dyspepsia are apt to grow worse and worse under strict treatment for ulcer, while an opposite mode of treatment (*vide infra*) often has the most brilliant results.

Prognosis.—The prognosis depends mainly on the outward circumstances of the patient. If the harmful mental influences or other ætiological factors are persistently active, actual and permanent recovery is hardly to be expected; but if the cause can be removed, complete recovery is not rare, even in what seem to be grave cases. A liability to relapses is, of course, almost always left behind, as most patients are constitutionally nervous.

Treatment.—If nervous dyspepsia has once been diagnosticated, the proper aim of our therapeutic efforts becomes perfectly definite. We must, in the first place, convince the patient that he has no incurable or even dangerous gastric disease, but that, on the contrary, his stomach is perfectly capable of performing its functions in a normal manner. Nothing could be more harmful to a sufferer from nervous dyspepsia than to have his physician manifest great anxiety about treatment, and prescribe a very strict diet. The patient must rather be gradually led to use an abundance of nutritious food. It is in this way alone that he regains a confidence in himself, when he sees that the hearty food does him no harm, that he is gaining flesh, and that the bowels are becoming regular.

Internal remedies are best omitted altogether if the patient has already taken a good deal of medicine. If they are successful, it is usually because of their influence on the mind. It is very important to make sure that there is no abuse of purgatives. If we must prescribe some medicine, the bitter tonics are most suitable, particularly the tincture of nux vomica. For anæmic patients, we may order iron, quinine, or arsenic. For nervous vomiting and eructations, bromide of potassium is to be given, or antipyrine and similar remedies. Treatment with cold water and with electricity are often extremely beneficial, although probably their effect is mainly subjective, for in cases of this sort psychical treatment is of the greatest importance. Even if we gravely command the patient not to yield to the impulse to vomit, but to resist it as much as possible, such an injunction will often do away with the symptom, and it is not rare to find in cases of nervous vomiting that it will cease as soon as nourishing diet is allowed, although previously it has occurred invariably after the most delicate kinds of food. It is often very advantageous in nervous vomiting to limit, as far as possible, the amount of liquids, such as soup and milk, and to prescribe a dry substantial diet. In many cases the vomiting will then cease very promptly.

If there are certain objective abnormalities present as well as the purely nervous symptoms, we must pay a certain amount of attention to these (*vide supra*). If there is gastroptosis, we should forbid constriction of the waist by the clothing, and should order a suitable abdominal bandage. If there is hypersecretion, we should employ alkalies, or possibly lavage; if achlorhydria, hydrochloric acid.

Those methods of treatment are of great value which are directed to the toning up of the body in general and the nervous system in particular. The patient may go into the country or to the mountains or the seashore. Methodical treatment with cold water is good; in particular, sponging combined with rubbing of the trunk and the whole body almost always gives good results. On the other hand, those health-resorts which often do good in the case of organic gastric disease are but seldom beneficial in nervous dyspepsia. Thus, we have frequently seen such patients, who had been sent by their physicians to Carlsbad, return worse rather than better. We have repeatedly found electricity valuable, although we surmise that its subjective effect may be of chief importance. Galvanism is applied along the spinal column, and also through the stomach horizontally, one large electrode being placed upon the epigastrium and the other on the back. It is well to reverse the current frequently. Faradization and massage of the abdominal walls are indicated, especially when there is constipation.

We need hardly add that the ætiological factors must not be overlooked. The patient must be warned against mental or bodily overexertion, emotional excitement, etc. The main point is to aim at a methodical moral training of the patient. He should learn to feel and to behave like a healthy person. He should regain his self-control, and not allow himself to be upset by every slight psychical shock, whether subjective or coming from without. It is self-evident that this goal is to be attained not by baths and prescriptions, but by correct moral guidance. The best proof of the correctness of our views, with regard to the true nature of this disease, lies in the therapeutic successes which can be obtained in this manner alone, although it is a pity that they are more often achieved by empirics and quacks than by scientifically educated physicians. It is therefore true of nervous dyspepsia, as of all other similar neurasthenic conditions, that it is to be cured not by physic but by the physician.

SECTION V

DISEASES OF THE INTESTINES

CHAPTER I

INTESTINAL CATARRH

(*Catarrhal Enteritis*)

Ætiology.—The majority of cases of intestinal catarrh, like gastric catarrh, are due to an abnormal irritation of the mucous membrane of the intestine by its contents. In many cases the irritants are of a mechanical or a chemical nature, and depend upon the quantity and quality of the food taken, which explains why catarrh of the stomach and catarrh of the intestine are so often combined with each other. Noxious substances, taken into the system by the ingestion of spoiled food, such as spoiled meat, fish, beer, etc., very often play a part in the origin of intestinal catarrh.

To the intestinal catarrhs caused by improper food we may add the toxic catarrhs which are produced by the direct ingestion of poisonous substances into the digestive tract. Severe inflammations of the intestinal mucous membrane arise from poisoning by mineral acids and corrosive alkalies, arsenic, corrosive sublimate, etc. Intestinal catarrh may also arise from the imprudent use of certain drugs, especially active cathartics.

A great many cases of intestinal catarrh are due to infectious influences; these include most of the apparently spontaneous catarrhs, and also many, if not all, of the catarrhs attributed to taking cold or getting wet, and, finally, those affections which often develop epidemically or endemically in hot weather, and which we term summer complaint, cholera morbus, etc. Cholera morbus is an especially severe form, and it will be described more fully later on. We must also mention here that intestinal catarrh is very often one symptom of other general infectious diseases, such as typhoid, dysentery, septic diseases, or severe malaria.

In a final class of cases intestinal catarrh develops from disturbances of the circulation, which cause a passive hyperæmia of the intestinal mucous membrane. Diseases of the liver and portal vein, and also chronic diseases of the heart, kidneys, and lungs, are the chief affections which produce a stasis in the portal system, and thus an intestinal catarrh; but here the stasis is probably, in most cases, only a predisposing factor in the development of the catarrh, since the action of all other irritants is made easier by the disturbance of the circulation.

The great frequency of intestinal catarrh in both sexes, and at every age, is well known. Children, above all, have a pronounced tendency to diseases of the intestine, so that, by a probable estimate, almost one third of the illnesses of children are to be referred to the intestinal canal. We will give a special description of intestinal catarrh in children on account of this fact.

Pathological Anatomy.—The pathological changes in catarrhal inflammation of the intestines are essentially the same as are met with in the inflammation of any other mucous membrane. Redness and swelling of the mucous coat, increased secretion of mucus, and in severe cases purulent products on the surface of the membrane, and a cellular infiltration of the tissue itself, are the well-known processes characteristic of all catarrhal inflammations. The solitary and agminated follicles often swell in follicular catarrh, and they may finally become the seat of superficial follicular ulcers. We often find superficial erosions on the rest of the mucous membrane, and in severe cases the so-called catarrhal ulcers.

If the catarrh has lasted a long time, we sometimes find a considerable thickening of the mucous membrane, which is due to hyperplasia of the connective tissue, and gives an uneven, puffy appearance to the internal surface of the intestine. Circumscribed hyperplasia of the connective tissue may actually lead to the formation of polypi. If the orifices of Lieberkühn's follicles are stopped, we have a cystic degeneration of the follicles from the retention of the intestinal juice.

We very often find, however, a considerable atrophy of the mucous membrane, especially in the chronic intestinal catarrh of children. This atrophy, which has lately been carefully investigated, especially by Nothnagel, affects chiefly the glandular layer of the mucous coat. In place of the glands, which in many parts may wholly disappear, we find connective tissue more or less rich in cells. The atrophy is usually most pronounced in the colon and the lower part of the ileum. The muscular coat may also take part in the atrophy.

Certain peculiarities of catarrh affecting single portions of the intestine will be mentioned later on.

Symptomatology.—The symptom by which chiefly we determine an affection of the intestinal canal, and which in the milder cases is often almost the only sign of an intestinal catarrh, is diarrhœa—that is, abnormally frequent stools of a looser consistency than usual; yet, strictly speaking, we should not attribute every diarrhœa to a catarrh of the intestinal mucous membrane, since a large number of influences may directly produce an increased peristalsis and a consequent diarrhœa. Thus, for instance, it is a well-known fact that sudden terror or great anxiety may sometimes cause an obstinate diarrhœa in a very short time. In general nervous and neurasthenic conditions, we sometimes have a chronic diarrhœa which can be due only to abnormal processes of innervation—"nervous diarrhœa." The diarrhœa which may arise immediately after taking cold is also merely the result of abnormally great peristaltic movements excited in a reflex manner. Probably a number of chemical and infectious irritants may also stimulate the movements of the intestines, and thus set up a diarrhœa, without causing at the same time a catarrh of the mucous membrane. Practically, however, we can not make a sharp distinction between diarrhœa and intestinal catarrh; and, in most of the diarrhœas which have lasted for some time, we are certainly right in supposing that there are actual anatomical lesions of the intestine, as well as functional disturbances.

There are two chief factors which cause diarrhœa in intestinal catarrh. In the first place, as has already been intimated, the same injurious substances which cause the catarrh also excite peristalsis. The many products of the abnormal processes of decomposition in the intestine also exert a like influence. Besides the abnormal irritants, however, we ought also to consider an abnormally great irritability of the intestinal walls in catarrh. Thus it happens that the fluid contents of the intestine are expelled by the vigorous peristaltic movements (which the patient himself often feels as a "rumbling in the abdomen"), before the normal consolidation of the fæces is completed by the absorption of water. The food, under normal conditions, passes through the small intestine in two or three hours, and thus the consolidation of the fæces takes place, as is well known, almost exclusively in the colon. We see, therefore, why the diarrhœa owes its origin chiefly to the increased peristalsis of the large intestine; although in many cases the peristaltic action of the small intestine is also increased.

Besides increased peristalsis, another circumstance may perhaps contribute to the diarrhœa, viz., the greater fluidity of the contents of the intestine due to the increased secretion of mucus and the exudation caused by the catarrhal inflammation.

In the intestinal catarrh due to passive congestion we must consider still another factor, to explain the thin and watery stools—namely, the diminished absorption of water by the intestine from disturbance of the circulation. In other catarrhs this factor is quite subordinate to increased peristalsis.

The diarrhœal dejections show a considerable difference in regard to their minor characteristics. Their number varies very much. There are sometimes two or three, and sometimes ten or more, evacuations in the twenty-four hours. The consistency of the stools is pap-like, or almost wholly watery. This is due to the abnormal amount of water in them, amounting to ninety or ninety-five per cent., while the amount in normal stools is about seventy-five per cent. The color of the thin stools in intestinal catarrh is usually bright yellow, but they are sometimes greenish from the admixture of bile pigment, and sometimes slimy (*vide infra*).

In only a part of the cases does microscopic examination give us information as to the extent and intensity of the catarrh. We usually find the remains of the food, muscular fibers, starch-granules, and fat, and also countless bacteria, and often triple phosphates, occasional pus-corpuscles, and cylindrical epithelium—chiefly the constituents which are found in normal stools. Further peculiarities will be mentioned below.

Besides the diarrhœa, there is often, but by no means always, abdominal pain in intestinal catarrh, either continuous, or having the character of paroxysmal, so-called colicky pains. In catarrh of the rectum there is that constant painful desire to go to stool which we term tenesmus.

Physical examination of the abdomen gives, on the whole, few important results. Sometimes the abdomen is flat, and sometimes there is meteorism. Marked peristaltic action of the intestines often causes gurgling and rumbling noises—*borborygmi*. On palpation, the abdomen is often somewhat sensitive. The peculiar colicky pains, however, are, as a rule, alleviated by external pressure. In rare cases we may detect a fluctuation on palpation, if the intestine contains much fluid. The results of percussion depend largely upon the contents of the intestines. There is dullness on percussion if the intestines are full, and also if they are contracted and devoid of air.

In many cases of simple diarrhœa the general health is practically unaffected, but in other cases of acute intestinal catarrh, especially in the severe infectious forms, the disturbance of the general health may be considerable. The patient feels so dull and weak that he stays in bed. We often see a moderate rise of temperature, between 100° and 102° (38°–39° C.). There are very often gastric symptoms also, especially loss of appetite and vomiting. Other organs are quite rarely affected, except in duodenal catarrh, when the liver is involved (*vide infra*). In acute infectious intestinal catarrhs, there is sometimes an eruption of herpes on the lips. We have repeatedly seen, in severe cases of acute enteritis, marked muscular and articular pains, and even slight but manifest swelling of the joints. There may also be albuminuria, casts, and even the signs of acute nephritis, as a sequel of enteritis.

Different Forms of Intestinal Catarrh.—Since the intestine is an organ which is only slightly accessible to physical examination during life, and since we can only rarely make a post-mortem examination in the mild diseases of the intestine, our knowledge as to the different forms of enteritis is defective in many respects. In practice we content ourselves in most cases with diagnosticating an intestinal catarrh simply from the existence of diarrhœa, without laying much stress upon the special variety; but in many cases some points can be obtained which give more accurate information as to the seat of the catarrh. The distinction between acute and chronic intestinal catarrh is also of practical significance.

Duodenal catarrh can be diagnosticated only if it is combined with jaundice. The details regarding it may be found in the chapter on catarrhal jaundice.

Isolated catarrh of the small intestines, of the jejunum and ileum, is probably only of rare occurrence, except when the upper portions of the colon are involved. We can very rarely diagnosticate it with certainty, but there are a number of factors which permit us to decide that the small intestine is chiefly affected, or at least that it is involved in the disease. In the first place, we may assume an affection of the small intestine, from obvious reasons, in all those cases in which there are also gastric disturbances. It is evident that, in the frequent combination of gastric and intestinal catarrh, the portions of the intestine nearest the stomach will be chiefly affected. Physical examination of the abdomen also gives some indications, since the slight sensitiveness and swelling of the abdomen, as well as the visible abnormal peristaltic action, affect chiefly the middle and lower portions of the abdomen in catarrh of the small intestines, while the analogous symptoms in catarrh of the large intestine affect the lateral and upper portions of the abdomen, corresponding to the anatomical course of the colon. We can not make a sharp distinction, however, in this respect. The results which auscultation and percussion over the abdomen give in regard to the point of origin of the gurgling sounds and the fullness of the loops of intestine are very rarely unequivocal, and hence are of little value in diagnosis.

Careful examination of the stools gives us more information. As has already been said, we need not have diarrhœa in a catarrh confined to the small intestines, since diarrhœa is due only to the increased peristalsis of the large intestine; hence diarrhœa is absent, for example, in most cases of duodenal catarrh (catarrhal jaundice). In more extensive catarrh of the small intestines the firm stools passed may, however, be regarded as pathological, because, on microscopic examination, they appear intimately mixed with little lumps of hyaline mucus (Nothnagel). As a rule, of course, catarrh of the small intestines is combined with a catarrh of the upper portion of the large intestine. Then we have a diarrhœa, but the thin stools show some peculiarities which point to an implication of the small intestines. As a result of the increased peristalsis of the small intestines, we find certain constituents in the stools which are normally contained in the small intestines, but which under normal conditions are no longer to be met with in the fæces in the large intestine. We find here, in the first place, undigested constituents of the food, large masses of muscular fiber, or even fragments of meat which may be recognized by the naked eye, and also starch and fat. Of course the opposite hypothesis does not hold good, that, if we find a large amount of the undigested portions of the food in the stools, it must necessarily always point to a catarrh of the small intestines, since the digestion may be impaired by other circumstances, and increased peristalsis of the intestines, from any cause, must result in the same symptoms. A diarrhœa, in which the thin stools contain a very large amount of undigested particles of food which can be recognized by the naked eye, was formerly called lientery, and the term is still occasionally used.

If the stools contain bile in addition to some portions of the food, it is to a certain degree characteristic of catarrh of the small intestines. Under normal conditions the contents of the small intestines alone show Gmelin's test for bile-pigment, while the contents of the large intestine, and also the normal stools, do not. In intestinal catarrh, with increased peristalsis of the small and large intestines, there is, however, often quite a large admixture of still undecomposed bile-pigment. The green stools which are so often seen in the diarrhœa of children, and more rarely in that of adults, are also well known. Such stools usually show a marked color reaction with nitric acid. In other cases we find only certain constituents of the stools stained with bile—a fact to which Nothnagel has called special attention. Yellow pigmented bits of mucus, and cylindrical epithelium and round cells stained with bile, are especially characteristic of the diarrhœa of catarrh of the small intestines.

Catarrh of the large intestine is probably present in every diarrhœa, as has been repeatedly stated, inasmuch as the thin stools can be explained only by an increased peristalsis of the large intestine; but in a number of cases we have symptoms which point especially to a disease of the large intestine, particularly of its lower portion.

Physical examination of the abdomen should show changes, such as swelling, sensitiveness to pressure, etc., chiefly in the lateral portions, corresponding to the course of the colon; but this is rather a theoretical hypothesis than a sign of practical value. We can not definitely affirm, either, that "colicky pains" are characteristic of catarrh of the large intestine alone. The condition of the stools, however, is of importance. In the first place, we may note that, if the stools contain many masses of mucus which may be recognized by the naked eye, it is of diagnostic significance. As we have seen above, the stools in catarrh of the small intestines also contain mucus, but it is intimately mixed with the other constituents of the fæces, and hence it can usually be recognized only by the microscope. In catarrh of the large intestine, however, the mucus rather adheres to the outside of the other constituents, and is often present in large masses visible to the naked eye. If the catarrh affects the lower part of the large intestine chiefly, it may be that the intestinal contents are already formed into firm lumps, which may sometimes be wholly or partly inclosed in a layer of mucus. In acute catarrh of the lowest part of the large intestine the evacuations are sometimes composed chiefly of pure mucus, with a greater or less admixture of pus, as is seen especially in the "catarrhal flux" (see the chapter on dysentery). The more the rectum is involved in the inflammation, the worse is that painful feeling of tension and pressure at the anus during and after the evacuation, which we term tenesmus.

Isolated inflammation of the rectum (proctitis) is, at least in part, directly accessible to examination by the finger or by the speculum. Painful tenesmus and an admixture of mucus, and especially of pus in the stools, are the chief symptoms of the disease. In most cases, however, we have to do, not with a primary disease, but with a secondary catarrh of the rectal mucous membrane, as a result of different morbid conditions in the vicinity of the rectum, or of new growths, syphilitic processes, etc., in the rectum itself. Periproctitis (ischiorectal abscess) belongs to the domain of surgery, and can not be described here.

Intestinal catarrh is divided into an acute and a chronic form.

In the acute intestinal catarrhs, excluding the toxic inflammations, we class simple diarrhœa, which usually passes off in a few days, and the severe enteritis, which is probably usually infectious, and is attended by a marked disturbance of the general health, by fever, and sometimes by gastric symptoms also, as well as by herpes, by occasional slight albuminuria, by articular pains, etc. It lasts from three to ten days. Cholera morbus (*vide infra*) is to be regarded as a special form of acute infectious inflammation of the gastric and intestinal mucous membranes.

Chronic intestinal catarrh either comes from an acute disease of the intestinal mucous membrane, or gradually develops independently. In adults it is by no means a frequent disease, at least as regards pronounced cases, and is much rarer, for example, than chronic gastric catarrh; but we have already mentioned that it is very common in practice among children.

In regard to the ætiology and symptomatology, much the same may be said of chronic catarrh which we have learned to recognize in considering acute catarrh. In regard to ætiology we must note, in adults, chiefly the intestinal affections remaining after an attack of some acute disease—e. g., dysentery, severe malaria, or typhoid. Among the most prominent symptoms are the abnormal evacuations, usually alternating between diarrhœa and constipation, due chiefly to the atrophy of the muscular coat and the disturbance of the nervous apparatus. We must also mention, as a prominent symptom, the secondary disturbances of the general nutrition, such as emaciation and anæmia. In regard to peculiarities in the character of the stools, we must refer to what has been said above. As chronic catarrh of the large intestine is far commoner than chronic catarrh of the small intestines, we very often find large amounts of mucus in the stools.

Treatment.—Most of the milder cases of acute intestinal catarrh need only a dietetic treatment. If the patient avoids all injurious substances for a few days, he recovers completely. The different gruels, such as barley and oatmeal gruel, and also weak broths, milk, and thoroughly toasted bread, or the German *zwieback*, are generally regarded as the most suitable food. The coarser vegetables and fruits, fat meat and brown bread, are to be avoided as much as possible. The best beverage is tea, or claret diluted with water. In other respects we may refer to the dietetic rules laid down under the treatment of chronic gastric catarrh.

It is also an important rule, confirmed by much experience, to keep the abdomen warm. Children should always stay in bed, and adults should do so, at least in all severe cases. It is a good plan, particularly in children, to protect the abdomen from cold by a flannel band.

In many of the mild cases it is scarcely necessary to use internal remedies. Gum mixture (P. G.) or almond mixture is a good prescription if there is no other special indication, but in severe cases further medication may be proper. If we have reason to suspect some irritating ingesta or a collection of fæces as a cause of the intestinal catarrh, a cathartic acts favorably at the beginning of the treatment, in spite of the existence of diarrhœa. Our best cathartic in such cases is castor-oil or calomel. In all those cases in which many thin dejections point to a greatly increased peristalsis of the intestine, we use astringents, especially opium, which we give in the form of the simple tincture or the wine in doses of ten or fifteen drops, one to three times a day; or as a powder, half a grain to a grain (gramme 0.03 to 0.05) of opium with a grain (gramme 0.05) of sugar, two or three times a day. It is also well to combine the opium with some mucilaginous vehicle, as 2 parts of laudanum to 150 of gum mixture or decoction of salep (P. G.), a tablespoonful every two or three hours. Tannic acid and the like are seldom employed in acute enteritis.

If there is severe colic, opium, or, under some circumstances, an injection of morphine is the best remedy. In milder cases it is sufficient to apply warmth to the abdomen, by warm poultices or hot towels. The colic, however, often depends upon the presence of old fæcal masses in the intestine, when it is necessary to prescribe a cathartic, such as castor-oil.

In all cases in which the symptoms point to a more intense disease of the large intestine, local treatment may be employed. This is chiefly of importance in the treatment of chronic intestinal catarrh situated mainly in the large intestine. We irrigate the large intestine daily with weak astringents, and sometimes with disinfectants. The necessary apparatus is very simple. It consists of an ordinary irrigator, to which a rubber tube, about half a metre long and with a proper tip, is attached. Instead of the irrigator we can use a large glass funnel, a "Hegar's funnel." We may very well use, for an end-piece to be introduced into the rectum, a long, soft, elastic œsophageal tube, which can easily be pushed quite high up. The fluids used for irrigation must always be warmed to about 85° (30° C.), and should be allowed to run in gradually and slowly. The amount of fluid used for one irrigation should be two or three pints (1–1½ litre), or sometimes more. The patient keeps on his back during the irrigation. The knee-elbow position, which is much more uncomfortable than the dorsal, is only occasionally necessary. The fluids most used are a one- or two-per-cent. solution of salicylic acid, solutions of salicylic and boracic acids combined, a one-per-cent. tannin solution, or a solution of acetate of lead (1 to 1,000).

If there is painful tenesmus, it is usually relieved by suppositories of cacao butter containing extract of opium.

In chronic intestinal catarrh a careful regulation of the diet is of the greatest importance. To be avoided are fruits and sour or greasy dishes, indigestible vegetables and puddings, coarse bread, and above all, beer; and to be recommended are tender lean meat (sirloin), sweetbread, fish that are not oily (such as trout and pike), potato purée, and particularly soft-boiled rice and sago. In regard to eggs and milk, individual experience must decide. Beyond this the main point is overcoming the chronic diarrhœa. Besides the cautious use of opium, the astringents are to be particularly borne in mind, either alone or combined with opium. Those most often prescribed are tannic acid, acetate of lead, columbo, and logwood. We have also seen excellent results in mild cases from the steady use of bilberry wine. The preparations of bismuth also deserve to be recommended, particularly the salicylate (8 to 15 grains several times a day, gramme 0.5–1), and the newly introduced tannigen (acetyl-tannin) and tannalbine (albuminate of tannin) in doses of eight grains or more (gramme 0.5) several times a day. In case the offensive odor of the discharges indicates abnormal decomposition in the intestinal canal, we employ naphthaline in doses of 1½ to 5 grains (gramme 0.1–0.3) several times a day. We often have to try various remedies in a particular case before we find one that is efficient. Great care should be exercised that there be no long periods of constipation; if necessary, we may use injections, saline laxatives, or castor-oil.

Good results are often obtained in chronic intestinal catarrh by drinking the waters at Carlsbad, Tarasp, Kissingen, Marienbad, or Homburg, particularly in cases with occasional constipation.

It is of practical importance to distinguish the genuine cases of chronic intestinal catarrh with an anatomical basis, from the common nervous disturbances of the digestive tract. Not infrequently the most diverse symptoms on the part of the intestine, including colic and irregularity of the bowels, appear as part of a general neurasthenia or nervousness (see page 1193). In such cases there is little benefit from internal remedies and strict diet, while appropriate general treatment with cold water, electricity, and massage is often attended with the best results. With regard to this compare also the chapters on nervous dyspepsia and on habitual constipation.

APPENDIX

Membranous Enteritis and Mucous Colic.—The name of membranous enteritis or mucous colic is applied to a condition which is not very common, but yet of great practical importance. It is characterized chiefly by the habitual discharge with the fæces of a great abundance of mucus, in the form of membranes. The disease is observed especially in women, but, in rare cases, also in men. It is noticeable that the patient is almost always a nervous, hysterical, or hypochondriacal individual.

In many cases a discharge of the membranes takes place in separate attacks, which occur either daily or at longer intervals, and are associated with severe colic ("mucous colic"). The membranes are gray or reddish gray, and often cylindrical or rolled up in a ball. They consist mainly of mucine—sometimes, it is said, of albuminoid matters also—and examined under a microscope they are usually found to contain an unusual amount of desquamated cylindrical epithelium, with a very small number of leucocytes, and perhaps also a few crystals of triple phosphate and cholesterine. In other cases there are scarcely any attacks of colic, but merely a persistent discharge of the above-described membranes and bits of mucus. The true discharges of the bowels are almost always very sluggish and very hard.

There is still much uncertainty as to the true nature of this diseased condition. Whether there is a genuine "enteritis" is doubtful, at least with regard to many cases. The noticeably frequent association of the disease with hysteria and neurasthenia seems to indicate nervous causes for the exaggerated production of mucus. The formation of the membranes probably takes place through the rolling up of the mucus in the depths of the longitudinal folds of the colon during its cramp-like contractions.

The **course** of the disease is usually tedious, but complete recovery may occur; there is no danger. The nutrition of the patient may remain perfectly normal, but in some cases it is greatly impaired. This depends chiefly upon the character of the other nervous symptoms present.

The **treatment** is partly local, by means of irrigations with alkaline solutions, or diluted lime-water, and the injection of oil and similar measures; and partly, or rather chiefly, general, for the cure of the associated neurasthenia. The most common modes of treatment are by means of hydrotherapy, electricity, and massage. In addition, we should combat the habitual constipation in the ordinary ways (*vide infra*).

CHAPTER II

CHOLERA MORBUS

(*Cholera Nostras. Cholera Infantum*)

By the name "cholera morbus" we mean an acute disease of the stomach and intestinal canal of a definite form, whose symptoms in severe cases greatly resemble those of genuine Asiatic cholera. It is in the highest degree probable, from the whole course of the disease, that cholera morbus also depends upon an acute infection of the body by a specific germ; but this germ has not yet been identified.

Cholera morbus comes on usually as an epidemic, and almost exclusively in the hot summer months—June to August. Hence it is often termed summer cholera. Children in the first two years of life are chiefly attacked, especially those who are artificially fed or who have recently been weaned. The disease also attacks older children and adults, but much more rarely.

[Special opportunities are afforded in this country for the study of cholera infantum. The causative conditions are, briefly, unsuitable food, a high temperature, and bad hygiene—conditions which are all combined and attain their maximum intensity in large cities. That a high thermometric range alone is not sufficient is shown by the comparative immunity of all country districts. Those who live in the country, or who have never been busied among the city poor, have no idea of the atmosphere breathed by the children of the poorer classes, especially during the heated term, nor of the extreme difficulty—impossibility we can almost say—of getting really good milk. Even if the milk was good at the start and it has not been tampered with, the time which necessarily elapses after it is drawn from the cow and before it reaches the consumer permits marked fermentative changes during hot weather. And milk is and must remain the main article of diet for children under two years of age.]

The symptoms of cholera morbus are those of a severe acute gastro-enteritis. The disease begins suddenly, or after some slight warning, with violent vomiting and severe diarrhœa. In some cases one of these symptoms predominates, and in others the other. The vomitus consists partly of the food taken, and partly of a slimy, watery substance. The stools at first retain their fæcal character, but they soon become more colorless and more watery, so that they sometimes approach the well-known rice-water appearance of the stools in genuine cholera. Abdominal pain is usually absent, but a feeling of pressure and constraint in the epigastrium is often present. The diminished secretion of urine and the frequent muscular pains cause the whole type of the disease to resemble genuine cholera still more closely. There is sometimes a cutaneous eruption resembling roseola.

The severe constitutional disturbance is especially characteristic. The patient becomes extremely dull and has a wasted look, the voice is weak and hoarse, an unquenchable thirst sets in, the pulse is very small, the skin of the face and the extremities is cool and livid; in short, we have the pronounced picture of a general collapse. The body heat also falls, although at the first stage of the disease there is often a rise of temperature.

[The temperature is always high, even during the stage of collapse, when the skin and extremities are cool to the touch; if the thermometer is introduced into the rectum—generally the best place, by the way, to take the temperature in young children—it will rise to 101° to 102°, and is more apt to reach 104° to 107°. This shows that inflammation plays a large part in the pathology of the disease.]

The picture of a severe general disease is especially prominent in cholera infantum. In severe cases of this form of the disease the general restlessness, which at first exists, rapidly passes into somnolence. The child lies with sunken, half-closed eyes, the conjunctivæ are slightly injected, the corneæ are cloudy, the face is pale and cyanotic, the fontanelles are depressed, the skin is cool, and the pulse is small and frequent and it can scarcely be counted. Amid these symptoms, which are usually termed "hydrocephaloid" by specialists in children's diseases [Marshall Hall], death comes on in coma or with slight convulsions. There can scarcely remain room for doubt that these severe cases of gastro-enteritis are of infectious origin, and that the constitutional symptoms are the result of toxic matters generated in the intestine under the influence of the micro-organisms (*vide supra*, the chapter on acute gastritis). Another peculiar symptom which occurs in severe cholera infantum is the so-called sclerema adiposum. While the temperature keeps sinking the extremities become peculiarly stiff and rigid, and the skin grows pale and firm. This phenomenon is caused by the fat in the fat cells of the subcutaneous connective tissue stiffening as a result of the low [superficial] temperature.

The mortality of children with cholera infantum is very marked, especially in large cities, and among the poorer classes of society. Severe cases usually end fatally in a few days, but, on the other hand, many cases recover, either because the course of the disease from the first is not so severe, or because cases apparently hopeless take a favorable turn. In adults it is extremely rare to see cholera morbus terminate unfavorably. Patients also recover quite rapidly from apparently severe conditions, although the stomach and intestines often remain rather sensitive for a long time.

The anatomical appearances in children who die of cholera infantum usually contrast, from their insignificance, with the severe symptoms observed during life. The catarrhal affection of the gastric and intestinal mucous membranes is not at all prominent in the cadaver, and the solitary follicles and Peyer's patches show only a slight swelling. A careful microscopic examination of the intestinal mucous membrane shows, however, quite severe inflammation. The other lesions which are most frequently seen are lobular atelectases in the lungs, venous hyperæmia and œdema of the pia mater, and slight lesions of the kidneys.

The diagnosis of cholera morbus presents no difficulty if the characteristic symptoms of the disease are present. The distinction between it and genuine Asiatic cholera used to be occasionally quite difficult, and it was rendered possible only by considering the ætiological factors, and the evident connection between the individual case and other cases of undoubted cholera. By Koch's discovery of the comma-bacillus in Asiatic cholera the distinction between the two diseases has now become absolutely certain. In all suspicious cases, therefore, we must examine the dejections for comma-bacilli, and upon the result of this examination depends the determination of the proper means of prophylaxis.

The treatment of cholera morbus in adults must be first to take special care to limit the diet. The food should be only gruels, or at most broth, soft-boiled eggs, and milk. It is a good plan to give the milk iced, and in small amounts. The distressing thirst is best relieved by cracked ice. Wine (iced champagne) is to be given if the general weakness becomes marked.

Among drugs, opium is the most effective remedy, and, whether in powder, as the extract, or in liquid form, as laudanum, it is the first thing to use to relieve the diarrhœa and vomiting. All other remedies which are recommended in cholera morbus in adults, such as nitrate of silver, are quite subordinate to opium. We may combine small doses of calomel with the opium. If there is a severe collapse which threatens life, it is advisable to make a subcutaneous injection of water, containing 0.6 per cent. of salt, or 5 per cent. of grape sugar.

We are more cautious in prescribing opiates for children, although here small doses of opium, one or two drops of laudanum according to the age of the child, may often be indispensable. In fresh cases calomel has obtained a great reputation, a sixth of a grain (gramme 0.01) two or three times a day. Ice-cold cow's milk, given in teaspoonfuls, serves best as food, if the child can not be fed naturally by breast-milk.

Many specialists recommend that no food at all should be given for a few days, but merely a sufficient amount of cold water or chamomile tea. If nothing will stay on the stomach, we may try a subcutaneous injection of 0.6 per cent. saline solution. If there are symptoms of severe collapse, we should immediately employ hot baths at 100° F. (30° R.), with the addition of 10 to 15 ounces of mustard (300–500 grammes) and hot packs, as well as such stimulants as camphor or alcohol subcutaneously. To guard against desiccation of the sclera and cornea, the eyes should be covered with compresses moistened with solutions of acetate of lead, corrosive sublimate, etc.

CHAPTER III

INTESTINAL CATARRH OF CHILDREN

(Chronic Dyspepsia of Children. Pedatrophy)

THE great frequency and the practical importance of the "dyspeptic conditions" in children in the first years of life, which conditions are associated with severe disturbances of nutrition, justify a short description of them, but we must refer to the special manuals on children's diseases for a detailed account.

That diseases of the digestive organs play so large a part in children's troubles is owing, on the one hand, to the great sensitiveness which the digestive apparatus in children shows to the irritants which are brought in contact with it, and, on the other, to the too common foolishness and carelessness which the child's parents and nurses show in its feeding. Of course it is not always ignorance and neglect, but often, unfortunately, poverty and want which cause children to suffer, and explain the terrible mortality in the first years of life.

The simple fact that by far the larger number of children who suffer from dyspeptic and atrophic conditions are fed artificially, leads us to the belief that the cause of most of the intestinal diseases in children is to be found in faulty and injudicious feeding. The food, which is not suited to the child's digestive powers, is only imperfectly absorbed; it undergoes many processes of decomposition, whose products irritate the intestinal mucous membrane and give rise to increased peristaltic action. Thus the imperfect digestion, or "dyspepsia," excites a catarrh of the gastric and intestinal mucous membrane, by which again, in a vicious circle, the digestive power is still further reduced. Hence the boundary between "dyspepsia" and catarrh can be drawn only artificially. It is certain that infectious influences also play an important part in the intestinal diseases of children. One proof of this is that indigestion is much more frequent during the hot weather than during the winter months, but although numerous bacteriological investigations have been made, observers have not yet been successful in determining special pathogenic organisms for the several varieties of intestinal catarrh.

The anatomical changes of the intestinal mucous membrane in children who die of "chronic intestinal catarrh" are, as a rule, only slightly marked, and contrast, in their apparent insignificance, with the severe intestinal symptoms observed during life. Probably here also the toxic influences of the matters abnormally generated in the intestine, play a more important part than do the anatomical changes. We must remember, however, that most catarrhal conditions are hard to recognize in the cadaver because of the disappearance of the hyperæmia. The changes present are quite important, but they require microscopic examination for their detection. Sometimes the swelling of the follicles is especially marked—follicular catarrh. Follicular ulcers are also seen. In other cases the atrophy of the mucous membrane, which is often seen after chronic catarrhs, is the chief lesion. Chronic thickening and swelling of the mucous membrane is of rarer occurrence. In most of the severe cases the large intestine, and also the lower portion of the ileum, are the chief seat of the changes. We often find a swelling of the mesenteric lymph-glands, and also a fatty liver. In the lungs extensive atelectases or nodules of catarrhal pneumonia often develop as a result of the imperfect respiration.

The symptoms of chronic intestinal catarrh are, in the first place, those due directly to the intestinal trouble, and, secondly, the quite rapid disturbance of the child's general nutrition.

The condition of the stools is the most important intestinal symptom. The normal dejection in children until they are weaned is of the color of the yolk of an egg, of a rather pasty consistency, and of a faintly sour smell. In intestinal catarrh the stools are more frequent, six or seven, and even more, a day. They are thinner, more watery, contain large flakes and lumps of undigested bits of caseine and other remains of food, and smell badly. They very often have a green color,* or acquire it on standing. We may find admixtures of mucus, sometimes in the form of the so-called "sago grains," especially in catarrh of the large intestine. With the microscope we find in severe cases, besides particles of food, leucocytes and epithelial cells, threads and clumps of mucus, and innumerable bacteria of all shapes. There are also crystals of triple phosphate, when the stools have an alkaline reaction, and needle-shaped crystals of the fatty acids, as well as plates of cholesterine, when the discharges are acid.

There is no definite distinction in regard to the dejections in catarrh of the large and of the small intestines. On the whole, the rule holds that, in catarrh of the small intestines chiefly, the stools are larger, they are passed with more wind or gas, and show a more uniform consistency; while in catarrh of the large intestine they are smaller but more frequent, ten or twenty a day, are passed noiselessly, are associated with tenesmus, and show a different consistency in their various parts, partly normal, partly thin, partly slimy, etc. Examination of the abdomen has some importance, for, as a rule, in catarrh of the small intestines the abdomen is much swollen, while in catarrh of the large intestine it is often deeply sunken. The liver is often enlarged and distinctly palpable, and sometimes the spleen also.

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* The statement that this green color is due to a special variety of bacillus lacks confirmation.

We often find disturbances in the stomach, vomiting, eructations, etc., as well as trouble in the intestines. There may be thrush in the mouth, or the development of aphthous ulcers.

Among other complications, we see diseases of the respiratory tract, including bronchitis, atelectasis, and catarrhal pneumonia; also albuminuria, eczema, furunculosis, and nervous disturbances.

In almost all long-continued cases, however, the general disturbance of nutrition, the atrophy (athrepsia) of the child, takes the first place in the picture of the disease. The muscles become shriveled and flabby, and the whole body finally becomes so much emaciated that the pale, dry skin hangs in broad folds and wrinkles about the bones, whose prominences are everywhere visible. The face is sharp, and has an aged expression ("Voltaire-face") from the many little folds of the skin. The eyes are dull, lusterless, and wide open; the voice is merely a low, hoarse whimper. The abdomen is deeply sunken, or in some cases it is swollen by meteorism, in peculiar contrast to the emaciation elsewhere; and its surface is traversed by bluish veins.

From this sad picture, unfortunately so frequently seen in practice among children, we can usually recognize the condition of things at the first glance, for by far the larger part of the cases called "pedatrophy" are due to chronic digestive disturbances. Very often it is combined with rachitic changes in the bones, of whose occurrence we shall speak further in the description of rachitis. Tubercular changes, too, are often found in the cadaver, especially in the lungs and the bronchial and mesenteric lymph-glands. In such cases, of course, the tuberculosis is usually to be regarded as the main disease, upon which the intestinal affection, which may be simple or even tubercular, has developed secondarily. During life tuberculosis in little atrophic children may very easily be overlooked.

If we would give a full account of the treatment of the atrophic conditions in children due to digestive disturbances, we must include in our consideration the entire hygiene and care of children in health and disease, for all children's physicians are united in the opinion that, as the cause of most intestinal diseases in children is to be found in improper feeding, so recovery from existing digestive disturbances can take place primarily only by a corresponding proper and judicious feeding. In what follows we can refer only to the most important principles and general points which are here to be considered.

The only proper and natural food for a child in its first year is breast-milk. All dyspeptic conditions are much rarer in children who are nursed than in bottle-fed children, and, when they do occur in children at the breast, they often are only of brief duration. They are then to be referred usually to certain disturbances in the mother, such as disease, improper food, or severe mental excitement. The return of menstruation or a new pregnancy has sometimes an unfavorable influence on the character of the milk. Finally, we may mention that in spite of the best of milk, if the breast is given irregularly and too frequently, it may cause anomalies of digestion in nursing children.

Most of these slight disturbances are easily quieted. Sometimes the fault lies in the unsuitable diet of the wet nurse. If this is too rich in fat or in albumen, the breast-milk may not be well borne. Exceptionally it happens that, without any discoverable reason, the milk of a wet-nurse "does not agree" with the child. Then we must change the nurse. The atrophic conditions which develop and progress in children, in spite of plenty of normal food, are usually due, not to simple digestive disturbances, but to deep-seated, general, constitutional diseases like tuberculosis, or syphilis.

The great majority of cases of chronic intestinal catarrh and chronic atrophy are found, as we have said, in bottle-fed children. The first question which every physician should ask a mother who brings him such a child for treatment, must therefore refer to the sort of feeding which the child has. If the mother, for any reason, can not nurse it herself, and if the bottle-fed child has digestive disturbances, we must invariably consider, in the first place, the possibility of procuring a wet-nurse. Feeding by the milk of a wet-nurse is a remedy which, at least in many cases, by saving the child's life, repays the many annoyances and quite large expense which a wet-nurse causes. We must tell the parents this, and represent to them, without reserve, the great dangers which threaten the life of every bottle-fed baby. Complete, and sometimes even quite rapid, recovery may be obtained through a wet-nurse, even in cases of severe chronic intestinal catarrh, when atrophy and weakness are already very far advanced.

Often, however, it is impossible to hire a wet-nurse, especially in the poorer classes of society. We must continue bottle-feeding, and these are the cases among which chronic intestinal catarrh demands the greatest number of victims; yet even here the physician can always do much good by instructing the parents.

The best substitute for mother's milk is cow's milk. This must be as fresh as possible, and is given boiled. A great and most beneficial advance in the artificial nourishment of infants was made by the introduction of Soxhlet's apparatus for boiling milk. This accomplishes an almost complete sterilization of the milk and the vessels which contain it. One part of the milk must be diluted, according to its quality, with two or three parts of boiled water in the first months, in children from four to six months old with equal parts of water, and in older children with about half as much water. At the age of about nine to twelve months, the child may have undiluted milk. In general we give the milk warmed to about 85° (28° C.), but children with gastro-intestinal catarrh often bear cold milk, given in small amounts, better than warm. There are special additions made to the milk, with the intent of rendering the cow's milk a better substitute for mother's milk. Of these, the first to be mentioned is milk sugar. In feeble children experience shows that it is often very advantageous to dilute the milk with oatmeal water or rice water. Sometimes, also, the addition of veal broth is most satisfactory.

Cow's milk, properly diluted, is better for children with chronic intestinal catarrh, in many cases, than any other food. In severe acute digestive disturbances, however, it is sometimes advisable to omit the milk entirely for a few days, and give instead of it only a little chamomile tea or some mucilaginous drink such as oatmeal gruel made with water and strained, or decoction of salep. In chronic dyspepsia we must first try good cow's milk. If the milk is not well borne, if the diarrhœa increases, and if the child becomes still more emaciated, we may try to get milk from another and better source; but it often happens either that we can not procure good milk, or that the child can not bear even the best cow's milk. We are then obliged to have recourse to one of the many "artificial foods" and "substitutes for mothers' milk" in the market. We can not here go into particulars concerning these. Each of these preparations has occasional good results to show, but none of them has an uncontested pre-eminence over the rest. We will mention the preparations most in use at present, of whose value in individual cases we have convinced ourselves: Swiss condensed milk and the infants' foods of Nestlé, Kufeke, Löfflund, and Theinhardt, as well as many others. Gärtner's cream-milk (*Fettmilch*) is sometimes advantageous. It is artificially prepared and contains little caseine but much fat.

If we keep fast to the principle that every intestinal catarrh in children is to be treated in the first place by a judicious regulation of the diet, in many cases we shall not have to use any drugs. These may be of service only when we have also carried out the dietetic measures which are specially necessary.

Calomel has obtained the greatest reputation in the treatment of intestinal catarrh in children. It deserves to be tried, particularly in rather early cases, in doses of $\frac{1}{12}$ to $\frac{1}{6}$ of a grain (gramme 0.005–0.01) in powder. If the diarrhœa lasts a long time, we may very well use opiates, although with great caution. The combination of calomel and opium often does good service.

℞ Calomel .... gr. $\frac{1}{6}$ (grm. 0.01);
Extracti opii .... gr. $\frac{1}{30}$ (" 0.002);
Pulv. acaciæ .... gr. ss (" 0.03).
M. et ft. pulv.
Sig.: One such powder, three or four times a day.

With little children we may put two to four drops of laudanum in three ounces (grammes 100) of liquid, such as gum mixture, salep decoction, muriatic-acid mixture, etc., and give a dessertspoonful of this every two or three hours.

Many attempts have been made to check the abnormal processes of decomposition in the intestine by prescribing remedies which possess antiseptic and antizymotic properties. Creasote has been warmly recommended by many, four to six drops in two ounces (grammes 50) of water with half an ounce (grammes 15) of syrup, a teaspoonful every two hours. Other drugs used for the same purpose are naphthaline, one-half- to one-per-cent. solution of dilute muriatic acid in water, and one-per-cent. solution of chloral in water. A prescription for naphthaline is as follows:

℞ Naphthaline .... 0.5 to 1.0;
Mucilag. acaciæ,
Aquæ destillat ....āā 40.0;
Ol. menthæ piperitæ .... gt. j.
M. Sig.: Shake. Teaspoonful every two hours.

If the stools have a green color, it is said that lactic acid is especially beneficial. A teaspoonful of a two-per-cent. solution may be given fifteen minutes after every meal.

A number of other remedies, "astringents," are given to act directly on the diseased mucous membrane. Those most to be recommended in chronic diarrhœa are subnitrate of bismuth, one or two grains (gramme 0.05–0.1) four to six times a day, which may be combined with opium, nitrate of silver (1 to 2,000 solution), alum (1 to 200 solution), guarana, five to fifteen grains (gramme 0.3–1.0), three times a day, and many others. Of late, tannalbine has been much recommended for diarrhœa in children; smaller children should receive four grains (gramme 0.25) several times a day, and larger children twice as much, in milk or water.

If a large amount of mucus in the stools points to a catarrh of the large intestine, we may sometimes employ irrigation of the colon with excellent results. We inject the fluid, one-per-cent. solution of tannin or alum, or solution of acetate of lead (1 to 3 to 1,000), once or twice a day. The amount of fluid to be introduced at once, by a Hegar's funnel, with a gum-elastic catheter, may reach one or two pints (half a litre to a litre).

In many cases of chronic diarrhœa it is said that the continued use of the water of the Carlsbad Mühlbrunnen proves wonderfully beneficial even for little children. If there are at the same time marked gastric symptoms, such as frequent vomiting, we may advantageously employ lavage. This is performed by means of a soft-rubber catheter, about six millimetres in diameter, connected with a rubber tube attached to a small glass funnel. For liquid we take a weak solution of hydrochloric acid, 1 or 2 parts per mille, or a 0.5-per-cent. solution of boracic acid.

[In view of the great importance of this malady, it seems desirable to remark on one or two points.

In the first place, prophylactic measures are deducible directly from the ætiology. No child should be kept in a large city in summer, if it can be provided for at the sea-shore or in the country. The vast number who must perforce remain are to be kept under the best general hygienic conditions possible. Mothers should be encouraged to take their children to the relatively pure air of the public parks as much as they can. The relation between diet and the diarrhœa of children should be dwelt upon whenever there is opportunity.

The establishment of boards of health has done much, and will do more, to check the ravages of summer diarrhœa.

Great advances have been made in the artificial digestion of cow's milk within a few years. By the aid of the preparations of Fairchild Bros. & Foster the caseine is digested in part, and the remaining portions coagulate in light flocculi; at the same time no appreciable taste is imparted to the milk, provided that a moderate amount of care is exercised. The importance of preventing the formation of large, firm curds has long been recognized, and an older means of attaining this end was mechanically to separate the curd by adding to the milk a barley or other similar water-gruel. Mellin's, Horlick's, and Ridge's foods also deserve mention in this connection.]

CHAPTER IV

TYPHLITIS AND PERITYPHLITIS

(*Appendicitis. Typhlitis Stercoralis. Inflammation of the Cæcum*)

Ætiology and Pathological Anatomy.—Inflammation of the cæcum and its vicinity has a special place among the diseases of single portions of the intestines. The reason why circumscribed inflammation so often develops here is to be found in the peculiar anatomical arrangement of the cæcum and its appendix, the vermiform process. It is evident that the conformation of the parts makes it particularly easy for inflammatory germs to settle in this place.

Formerly it was believed that the inflammation might often be limited to the cæcum itself. It was asserted that impacted fæcal matter occasioned mechanical irritation of the intestinal wall, and thus facilitated the attack of chemical and infectious agents. In this way arose the doctrine of "typhlitis stercoralis," but no such simple typhlitis has ever been demonstrated post-mortem, and although it can not be absolutely denied that under certain circumstances there may occur a genuine typhlitis, as a result of the accumulation of fæces, yet its frequency has certainly been much overestimated (Sahli). It is years since the author has made a diagnosis of "typhlitis stercoralis."

By far the greatest number of cases of acute inflammation in the ileo-cæcal region are perityphlitis; that is, inflammation of the peritoneum and the connective tissue surrounding the cæcum. From the point of view of pathological anatomy, we might term the inflammation of the retro-cæcal tissue paratyphlitis, inasmuch as it is extra-peritoneal, in distinction from the true intra-peritoneal perityphlitis; but clinically we can not maintain this distinction, or at most, imperfectly. The starting point for perityphlitis is the vermiform appendix. To this there are rare exceptions in which the inflammation arises from perforation of the cæcum itself. The appendix, although of so little physiological importance and a merely rudimentary portion of the intestine, plays an important part in pathology. Small fæcal masses from the cæcum often enter the vermiform appendix, and, under some circumstances, they may remain there. The fluid in them is absorbed, they are very often incrusted with lime-salts, and thus the little so-called "fæcal calculi" are formed. In many cases the return of fæcal masses into the cæcum is probably hindered by the valve at the orifice of the vermiform appendix, Gerlach's valve. Foreign bodies, such as little seeds of fruit, often enter the vermiform appendix and perhaps give rise to the formation of a fæcal calculus; but one must be cautious in assuming that any small hard mass is a foreign body, for these calculi often have such a rounded shape that they were formerly considered, very erroneously, to be retained cherry-stones.

In many cases fæcal calculi may remain in the vermiform appendix for a long time without producing any further injurious results, but as a rule they cause a mechanical irritation of the mucous membrane which leads to inflammation, and often, in some circumscribed spots, to a pressure necrosis, and later to ulceration of the vermiform appendix. If the ulcer does not cicatrize—it always may—the ulceration gradually deepens. The pressure necrosis of the wall allows ingress to the ordinary pathogenic agents of inflammation and suppuration, among which the colon bacillus probably plays the chief rôle. The inflammation, once started, rapidly spreads to the surrounding tissues. In rare cases a diffuse general peritonitis is immediately developed. In by far the greatest number of cases there are timely adhesions around the appendix, so that the inflammation is limited and encapsulated.

It is improbable that fæcal calculi occasion the development of all cases of perityphlitis, but they certainly do of the majority. It may be that the inflammatory germs sometimes reach and attack the appendix unaided. One circumstance that would favor such an occurrence is the abundance of adenoid tissue in the appendix, which has suggested a comparison with the follicles of the tonsils. Sonnenburg has insisted that in many cases the inflammation may be entirely limited to the vermiform appendix. This is possible, but in our opinion it is out of the question to make at the bedside an absolute diagnosis of "appendicitis simplex."

With regard to the nature of the inflammation in perityphlitis, there can be no doubt that it is almost always purulent, but this does not mean that there is always a marked destruction of tissue with the formation of an extensive abscess. Such results are found only in the severe and long-continued cases. In the ordinary milder cases there are usually merely an infiltration of the tissue with pus cells, and an inflammatory œdema of the tissue, or perhaps also a limited fibrinous exudation. There is an inflammatory tumor which consists essentially of the thickened intestinal walls and the coils of intestine glued together, and perhaps sometimes also of impacted fæcal matter; and inside of this there is an actual abscess, small and localized, and lying usually in the immediate neighborhood of the appendix.

Perityphlitic inflammation can very well be compared with the exudate of parametritis, for in this also the cellular infiltration predominates over the formation of pus. In rare cases there may be an encapsulated exudation of a more seropurulent character.

The further pathological changes in perityphlitis will be best considered in connection with the clinical symptoms.

Symptoms and Clinical Course.—We have already said that there is great doubt whether simple "typhlitis stercoralis" ever occurs, and therefore we shall refrain from giving a description of its symptoms, which we should have to invent, and turn at once to those clinical phenomena which we observe in acute inflammation of the ileo-cæcal region, and which are now almost universally termed "perityphlitis" [appendicitis]. This disease has great clinical importance, for it is very frequent both in children and adults, and involves great danger to life, especially if there is not prompt surgical interference.

The symptoms of perityphlitis have usually a rather acute development. The patient has felt perfectly well, and, as a rule, has not even suffered from constipation, when he has a rather sudden pain in the ileo-cæcal region. There may be no occasion for this, or there may be some such cause as stooping, lifting, or a long walk. Not infrequently there is slight vomiting, and the patient feels languid and feverish. The bowels may be constipated, but sometimes they continue regular. Many patients are obliged to go to bed at once, but others keep about for some days till they are forced to give up because of the aggravation of their symptoms, particularly the pain. It is a point of some practical importance that the pain is by no means invariably referred to the region of the cæcum at first, particularly as the position of the appendix is very variable. Not infrequently the pain is localized more in the middle of the abdomen or toward the left, or upward. It is not until subsequent days of the attack that the pain gradually settles in the true ileo-cæcal region.

Upon objective examination we usually find distinct local changes in the ileo-cæcal region, even in the first days of the disease. Our manipulations should be extremely cautious. It may be possible by careful inspection alone to recognize a limited swelling. In that case it is very important to determine whether tenderness is present. This may be very considerable, or comparatively slight. The typical painful spot (McBurney's point) is said to be an inch to an inch and a half (three or four centimetres) inward from the anterior superior spinous process, on the line joining this process with the navel. This localization does not possess great importance, because the painful spot varies in different cases. Sometimes the greatest pain is felt posteriorly, in the lumbar region, suggesting a retro-cæcal paratyphlitic abscess (*vide supra*). Lastly, there is decisive importance in the discovery of abnormal resistance, or of a more or less sharply-defined tumor due to the inflammatory exudation (*vide supra*). This tumor can usually be made out. For this purpose we recommend particularly the employment of the palm of the hand in gentle pushing movements, beginning on the left side and gradually proceeding toward the ileo-cæcal region. This is the best way of determining the size of the inflammatory mass. In milder cases it is also permissible to make deeper palpation, if we are cautious. The examination *per anum* may be very important: we have seen a number of cases in which no exudation could be demonstrated by external palpation, while a large inflammatory tumor could be distinctly felt through the rectum. The results of palpation are confirmed by percussion, which gives either a muffled tympanitic resonance, or marked dullness over the seat of the disease. In general, however, the results of percussion should not be interpreted too strictly, for they are ambiguous, because of the variations in the degree of intestinal distention.

There are other symptoms to be considered besides the local ones. Of great importance are the aspect and general condition of the patient. If the patient has no suggestion of the "abdominal facies" (*vide infra*, chapter on peritonitis), it is very probable that the inflammation remains circumscribed. It is also important to study the chart of the temperature and pulse. Elevation of the bodily temperature invariably suggests aggravation of the local process. Marked acceleration of the pulse almost invariably indicates active suppuration, and the advent of sepsis. In most cases the fever at first varies from 101° to 103° (38.5°–39.5° C.), and if the patient keeps quiet it falls in the next few days. The pulse varies from 80 to 100. Not infrequently there is a decided remission in all the symptoms after the first two or three days. In the most favorable cases, convalescence may ensue upon this change, but very often the fever rises again, and in such cases there is great need of caution. The bowels are almost always constipated from the start. Vomiting occurs only temporarily, if at all. Frequent eructations and a return of vomiting later on in the attack are unfavorable symptoms. In many cases there is temporary difficulty in micturition, so that a catheter must be employed. There is apt to be a distinct increase in the amount of indican in the urine early in the attack.

A question of the greatest clinical importance is whether the perityphlitis will lead to the formation of a large abscess or not. We have already said that in a pathological sense almost all cases of perityphlitis are suppurative, but the formation of a large collection of pus is of practical importance. This is recognized by an increase in the fever, or by the mere persistence of the fever. If the fever lasts more than a week or a week and a half, suppuration is highly probable. If there is a temperature of 104° to 105° (40°–40.5°) we may suspect suppuration, even at an early period, particularly if the rate of the pulse corresponds to the height of the fever. Rigors are not frequent at all, but, if they do occur, they indicate abscess formation. The local signs are not very valuable in the diagnosis of an abscess. Early in the attack it is scarcely ever possible to detect fluctuation, for the abscess is deeply situated and surrounded by infiltrated tissue. For an absolute decision it is usually necessary to resort to exploratory puncture. This should be done with a long, slender needle, carefully disinfected, and it need cause no apprehension; but still it should never be employed unless there is an important reason for it.

The further course of perityphlitis is favorable in the overwhelming majority of cases. After a few days, or a week or so, if the treatment is appropriate, the symptoms abate and the fever disappears; it may fall gradually or defervescence may be quite rapid, almost like a crisis. The inflammatory tumor shrinks, the pain abates, the bowels become regular, and the patient gradually regains complete health. Often there are manifold variations in the disease, which lengthen its duration. With regard to the pathological changes in these cases that recover, of course we have no accurate knowledge. Sometimes it is probable that there never is a well-marked abscess formation, but only an inflammatory infiltration of the tissue, which subsides again. It is probable that, in many cases, there is a small abscess which discharges spontaneously into the intestine, usually into the cæcum, but exceptionally into other portions of the intestine. One thing which seems to us to suggest such a spontaneous discharge of an abscess is the not infrequent occurrence of a crisis, with rapid fall of temperature and simultaneous cessation of all the other symptoms. Still, we scarcely ever detect pus in the stools, for the pus-corpuscles are changed during their passage from the colon to the rectum. Perhaps in some cases small localized abscesses are reabsorbed.

If there is a larger abscess, and it does not discharge into the intestine or receive surgical attention, the pus seeks other paths. The worst occurrence is perforation into the general abdominal cavity, with consequent diffuse peritonitis and speedy death. Or the abscess may continue to grow, and finally break through the abdominal wall; or it may extend downward into the iliac fossa or the groins, or perforate into the rectum, bladder, or other parts. It has also happened that the suppuration has involved the ileo-cæcal vein, with consequent pylephlebitis and the formation of abscesses in the liver. In most of these chronic cases the symptoms are those of a tedious septico-pyæmia such as is nowadays, thanks to the progress of surgery, very rarely seen. As the inflammation originates from perforation of the intestine, the pus is apt to be extremely offensive, whenever there is any extensive abscess formation.

The greatest caution is demanded even in cases in which the course of the disease seems at first favorable. The improvement of the second or third day after the sudden onset of the illness is often deceptive, as we have already mentioned, but later on there may be manifold vicissitudes in its course, so that it is necessary to watch the patient constantly and closely. Often there will be a fresh elevation of temperature and the formation of an abscess late in the attack. If the bowels are obstinately constipated the symptoms of obstruction may appear, such as tympanites, frequent eructations, or even fæcal vomiting. Complications in other organs are exceptional.

Diagnosis.—In general, there is no difficulty in the diagnosis of perityphlitis, but it is difficult and to a certain degree impossible to determine accurately about the anatomical changes, the exact location and size of the inflammatory tumor, or the formation of an abscess. Here a careful examination, including palpation *per anum*, is indispensable. In so far as this relates to the extremely important question of surgical interference, we shall revert to the matter when considering treatment.

In those rare cases of perityphlitis which have a chronic course, the disease may be confounded with new growths, particularly carcinoma originating in the cæcum or the appendix. Mistakes in diagnosis have also been occasioned by tumors of the right kidney and the right ovary, and by psoas abscess due to Pott's disease. In this connection it may be mentioned that in rare cases the lumen of the appendix may become occluded. The portion of the appendix thus cut off is then gradually distended by the secretion of its mucous membrane, giving rise to so-called dropsy of the vermiform appendix. This may occasion a tumor which can be felt in the ileo-cæcal region.

Prognosis.—If we take all the cases of acute inflammation of the ileo-cæcal region, we shall find that some 80 or 90 per cent. get well without operation, but still it should be emphatically stated that perityphlitis is a dangerous disease, and every year brings no small number of individuals, both children and adults, to sudden death. The main danger lies in the rapid onset of general peritonitis, or of severe sepsis. Even after the patient has survived the attack he may be troubled in various ways. For instance, the resultant adhesions and chronic exudates may impede the action of the bowels for a long while. The frequency of recurrent attacks is also to be considered. Many persons have repeated attacks of perityphlitis to the number of four or six, or even more during their lives. This is probably the result of peculiar anatomical conditions.

Treatment.—Formerly, when the opinion was still held that many of the cases of acute inflammation of the ileo-cæcal region were to be regarded as simple typhlitis stercoralis due to constipation, the first question in treatment was whether to give laxatives or opium. If there was typhlitis, laxatives ought to be given; if perityphlitis, opium. The aim of diagnosis, therefore, was to distinguish between these two conditions. To-day the conception of simple typhlitis has been almost completely abandoned, or, at any rate, it has become very limited, and consequently the question of treatment has become simpler, for now laxatives are scarcely ever given at the onset of the disease. Perhaps we have gone too far in this direction, for most of the older physicians will certainly, like the author, recall many cases of what was supposed to be typhlitis stercoralis in which rapid recovery followed the immediate use of castor-oil. Still, it can not be disputed that the premature employment of laxatives may do harm, while the administration of opium in a proper dose probably never leads to unfavorable consequences. For this reason we now make it a rule in any case of inflammation in the ileo-cæcal region, with acute onset, to begin with opium; but it is important, above all, that upon the first appearance of pain the patient should remain perfectly quiet in bed, and should have for nourishment merely milk or soup, but no solids. An ice-bag should be applied at first over the seat of the disease, and this may be replaced later, or at once, in case cold is not well borne, by wet compresses or poultices. Opium is administered in the form of laudanum, giving fifteen to twenty drops several times a day, or one half grain to a grain of the extract (gramme 0.3–0.5). The amount of opium to be administered is determined by the pain. In severe cases one must not hesitate to use large doses, but, on the other hand, the dangerousness of the remedy, particularly in children, should never be forgotten. The local application of eight to ten leeches has become entirely out of fashion and may be dispensed with, but, nevertheless, it is undeniable that leeches sometimes give the patient decided relief. With this treatment of rest, diet, opium, and ice, the pains are usually soon relieved. There is no necessity at first to be anxious about opening the bowels. Usually there is no harm if the patient goes without a movement for five to eight days, providing that his condition is satisfactory as regards his general health, pulse, and temperature. It is indeed not unusual to have a natural operation of the bowels while rather large doses of opium are being administered, but if the abdomen becomes more and more distended and there is rather long-continued constipation, we need not hesitate to make a cautious use of injections. For this we usually choose cool soapsuds, and let it run through an ordinary fountain syringe into the colon, while the patient is lying on his back. If necessary, the injections must be repeated, and of their beneficial effect we have often been convinced.

We have already insisted that every patient with perityphlitis should be most carefully watched, however favorable the course of the disease seems to be. The physician must ask himself every minute whether further symptomatic treatment is permissible, or whether it may not be necessary to have the abscess opened by surgical interference; for, although we are by no means of the opinion that every case of perityphlitis without exception demands surgical treatment, yet there is no doubt that prompt surgical interference can save many lives. The question when operation is required is consequently prominent at present in all discussions of this disease. Our answer can only be, in general, that immediate operation is invariably indicated as soon as we infer that perityphlitic suppuration is present. We dare not wait for its absorption or its spontaneous discharge into the intestine, because of the possibility of its breaking through into the peritoneal cavity, or arousing a constitutional sepsis, or some chronic suppurating process. The most important signs of the imminence of these dangers may be again summed up as follows: persistence or fresh exacerbation of the fever, after the first three to five days; increase in the frequency of the pulse beyond 100 to 120, and diminution in the tension of the pulse (both pulse signs are important, because they almost invariably suggest a large abscess); increase in the local phenomena; and, finally, above all, unfavorable appearance of the patient, a beginning "abdominal facies." If these symptoms appear there must be no hesitation about the operation. If we are still in doubt, we may first make an exploratory puncture. If even a drop of pus is obtained, operation is absolutely demanded.

It is impossible to give more detailed rules, of general application, with regard to the question of operating. The individual circumstances of each particular case and the personal experience of the physician must always be controlling factors. No physician can avoid unfortunate occurrences in perityphlitis, and the most experienced practitioner will never escape a certain feeling of uneasiness in any case of the disease; still, it would certainly be better to operate too early than too late. It is true that when the general condition of the patient is favorable, waiting is advisable, but, as Sahli has well said, it must always be an "armed peace." For particulars as to laparotomy and further treatment, as well as concerning the question of the necessity of removing the appendix, the reader is referred to text-books on surgery.

CHAPTER V

PERFORATING ULCER OF THE DUODENUM

THERE is a form of ulcer of the duodenum, especially of its upper, horizontal portion, which is precisely analogous to the round gastric ulcer in regard to ætiology, pathological anatomy, and, very largely, symptomatology. The ulcer is probably also due in most cases to the action of the acid gastric juice on the duodenal mucous membrane, under conditions which have been detailed more fully in the ætiology of gastric ulcer. We must mention here the noteworthy fact that, after extensive burns of the external skin, ulceration of the duodenum, rarely of the stomach also, has been repeatedly observed. This is probably due to the thrombosis of a duodenal vessel, occasioned by the setting free of fibrine ferment.

Ulcer of the duodenum is much rarer than the round gastric ulcer, and, in distinction from the latter, it has been found decidedly oftener in men than in women.

Many cases of ulcer of the duodenum run their course entirely without symptoms, or they cause no symptoms until sudden hæmorrhage appears (from erosion of the pancreatico-duodenalis, gastro-duodenalis, etc.) with hæmatemesis and bloody stools (quite often bloody stools alone without hæmatemesis), or until there are suddenly signs of peritonitis from perforation. In many cases a type of disease exists for a long time whose symptoms, as we have said, are so like the clinical symptoms of gastric ulcer that we can very rarely distinguish the two forms with certainty during life. We notice especially continuous or neuralgic pain, which, in ulcer of the duodenum, has its chief seat in the right hypochondrium. Severe gastric symptoms, especially vomiting, are not as common as in gastric ulcer. The general health and general nutrition may remain quite undisturbed for a long time.

Ulcer of the duodenum ends by cicatrization and recovery, or by cicatrization and the formation of stenosis, with secondary dilatation of the upper portion of the duodenum and of the stomach. In regard to the different adhesions and perforations of the ulcer into neighboring organs, we may refer to what has been said of gastric ulcer.

The treatment must be governed by the same principles which were laid down in the treatment of gastric ulcer, especially as the diagnosis is usually doubtful.

CHAPTER VI

TUBERCULOSIS OF THE INTESTINES

TUBERCULOSIS of the intestines is in most cases a secondary disease, and is one symptom of a more extensive general tuberculosis. It develops most frequently in the course of chronic pulmonary tuberculosis, and depends here, as we have seen (compare page 249), upon an infection of the intestines by the tubercular sputum that has been swallowed.

Intestinal tuberculosis, however, may also be a primary disease, and the source of further extension of tuberculosis over the body. "Tuberculosis of the abdominal organs," which usually comes from the intestines, has a clinical significance, especially in children. It is not improbable that in such cases the first infection of the intestine comes from without, and that the tubercular poison is taken into the body with the food. Here we must suspect especially the milk from cows with pearly distemper—that is, with tubercular disease.

The anatomical changes in intestinal tuberculosis are precisely analogous to the tubercular changes in other mucous membranes. The tubercular new growth has its origin usually in the lymph-apparatus of the intestine, in the solitary follicles, and in Peyer's patches. The first miliary tubercles form beneath the epithelium, and soon fuse with one another into a diffuse infiltration. In its further course the infiltration on one side extends deeper into the surrounding tissue, so that it attacks the submucous and muscular coats and even extends to the serous coat, and on the other side, by the destruction of the new growth which begins at the surface and constantly spreads, tubercular ulcers are formed. We can often make out with the naked eye single miliary tubercles or groups of them at the base or in the infiltrated edges of the ulcer. This is especially plain in deep-seated ulcers on the corresponding portion of the serous coat. The form of the larger tubercular ulcers is often irregular. In many cases the long diameter of the ulcer is parallel to the circumference of the intestine, so that the girdle-like ulcers, which are especially characteristic of tuberculosis, are formed.

Tubercular ulcers are situated both in the large and in the small intestines. They are usually most marked in the vicinity of the ileo-cæcal valve. Tubercular ulcers in the stomach are extremely rare. Besides the intestinal tuberculosis there is very often tuberculosis of the mesenteric lymph-glands, and also frequently tuberculosis of the peritoneum.

The symptoms of intestinal tuberculosis are usually quite subordinate to the symptoms caused by other co-existing tubercular affections. There may often be quite extensive tubercular ulcers without any marked symptoms, but, as a rule, the onset of diarrhœa turns the attention to the intestinal complication (see the chapter on pulmonary tuberculosis).

Tubercle bacilli have been repeatedly demonstrated in the fæcal discharges, but it is not very easy to find them, and, moreover, their diagnostic significance is impaired by the fact that they may be referable to sputum which has been swallowed.

Primary tuberculosis of the abdominal organs sometimes presents quite a characteristic type of disease, especially in children. This was termed by the older physicians *tabes mesenterica.* The chief feature of this type of disease consists in a progressive general emaciation and anæmia, which are usually associated with a persistent hectic fever, which obstinately resists all the remedies employed. The abdomen is usually swollen by meteorism, but it is sometimes flat and sunken. In some cases, but less frequently than was formerly believed, we can feel the swollen mesenteric lymph-glands through the abdominal wall during life. The involvement of the mesenteric lymph-glands obstructs the lymph-channels which serve for the absorption of fat, a circumstance which undoubtedly contributes, along with others, to the excessive emaciation frequently seen. The liver may be enlarged and its lower border can often be felt. The bowels are irregular, and there is usually a moderate diarrhœa, persisting in spite of all remedies. The invariably fatal termination is due to an increase of the general marasmus, or to a final acute tubercular affection, such as miliary tuberculosis or tubercular meningitis. The autopsy shows tuberculosis of the intestines, peritoneum, lymph-glands, liver, etc., to a greater or less extent. The lungs may be quite free from tuberculosis. We will return to this affection in the description of tuberculosis of the peritoneum.

The treatment of intestinal tuberculosis can be only symptomatic. Besides the general dietetic treatment which seeks to keep up the patient's strength as far as possible, medical interference is demanded by the abdominal pain and diarrhœa.

The chief remedy is opium, either alone or combined with tannin, tannigen, acetate of lead, or subnitrate of bismuth. Sometimes a decoction of logwood is beneficial. Talc also has lately been recommended; about six or seven ounces (200 grammes) are given daily, suspended in milk. We have seen some good results from it. The most serviceable local applications are poultices or cold compresses.

In other respects the treatment coincides with the general treatment of tuberculosis (*vide supra*).

CHAPTER VII

SYPHILIS OF THE RECTUM

In not very rare cases we see in the rectum, especially in its lower portions, extensive syphilitic ulcerations, which produce a severe and practically important type of disease. The more intimate relation between syphilis of the rectum and the general syphilitic process is not perfectly clear. According to quite a widespread opinion, the infection of the rectum comes from the secretion trickling down from the ulcers of the genitals. The facts seem to support this view, since syphilis of the rectum is seen much more frequently in women than in men. Some authors have even asserted that all the so-called "syphilitic" ulcers in the rectum have no connection at all with genuine syphilis, but are chancroids. It is in fact striking, even if it by no means proves such a hypothesis, that, at the autopsy of persons who have died of "syphilis of the rectum," we rarely find definite syphilitic changes in other internal organs—a fact which we also can confirm.

The most characteristic mark of syphilitic ulcers in the rectum is their tendency to form cicatrices and stenoses. This result of the ulcer is also important in its clinical relations, since the chief symptoms of the disease usually begin with the development of the stenosis. The seat of the stenosis is usually so low down that we can conveniently reach it with the finger, on a digital examination of the rectum during the patient's life. The rectum narrows like a funnel upward, and we can feel the quite sharp edge of the ring-like cicatrix with the point of the finger. This funnel-shaped stenosis of the rectum is so characteristic of syphilis of that organ that, in almost all cases, we can make the diagnosis with perfect certainty from this alone.

The rectum and the descending colon are usually dilated above the stenosis, and here extensive, irregular ulcerations, with undermined edges, are usually found in the mucous membrane. These are partly of a specific nature, and partly diphtheritic ulcers caused by the pressure of the accumulated fæcal masses.

The symptoms of syphilis of the rectum usually develop quite gradually. At first the bowels are irregular, and there are disturbances of defæcation which stubbornly resist the ordinary remedies employed. There are sometimes, in the first stage of the disease, frequent and severe hæmorrhages with the dejections, as we have seen, and for a long time these may falsely be considered to be "bleeding from hæmorrhoids." The symptoms become more marked as cicatrization of the ulcer increases and as stenosis of the rectum develops. There is usually a decided catarrh of the rectum, so that the thin stools contain a large admixture of mucus and pus. The patient's condition is extremely distressing, from the pains with the frequent but always scanty dejections, and from the severe tenesmus. Nodular thickenings and prolapse of the mucous membrane, and sometimes true hæmorrhoids, form about the anus. The patient's strength constantly diminishes from the pain and the continual diarrhœa. He finally becomes emaciated, looks very pale and wretched, and has fever toward night. Death ensues from increasing general weakness, or rarely from a terminal peritonitis due to perforation, after the whole disease has lasted one and a half to two and a half years.

This unfavorable termination unfortunately seems to be the rule in all the cases described; hence the prognosis is to be regarded as very serious in all cases of syphilis of the rectum. Improvement worthy of mention, or even perhaps recovery, is possible only when the disease is recognized at the outset and properly treated.

At the outset of the disease the treatment, of course, must consist chiefly of an energetic general attack upon the syphilis by mercurial inunction and iodide of potassium; but, when once the characteristic funnel-shaped stenosis of the rectum has formed, we can not expect much from anti-syphilitic medication, since this can no longer exert any influence on the cicatrices and their results. Improvement is now to be obtained only by mechanical dilatation of the stenosis by bougies, or, if this is not enough, by a surgical operation. A suitable local treatment by irrigation is also of benefit to the catarrh and to the ulcers that still exist in the rectum. Internally, we may continue to use iodide of potassium.

CHAPTER VIII

CANCER OF THE INTESTINES

The development of cancer is far more rare in the intestine than in the stomach. Carcinoma is seen with any frequency only at the lower end of the intestine, in the rectum. Next in frequency is carcinoma of the colon, particularly the cæcum, the sigmoid flexure, and the hepatic and splenic flexures. Carcinoma is very much less common in the small intestine, in which its favorite seat is the duodenum, particularly the neighborhood of the common duct.

Most cancers of the intestine appear in the form of ring-like swellings that take in the whole circumference of the intestine. More rarely we find a more diffuse papillary proliferation, extending over a larger surface of the intestine. There is often quite an extensive destruction of the new growth on the surface of the cancer, from which deep ulcerations arise. Often metastases are found in the lymph-glands, liver, and other organs. Above a cancerous obstruction the intestine is usually dilated, with thickening of its muscular coat, and inflammation or ulceration of its mucous membrane due to the impacted fæcal matter. In its histological structure, cancer of the intestine is to be regarded as invariably a cylindrical-celled carcinoma, which sometimes shows a plainly glandular structure—adeno-carcinoma—and sometimes that of the other forms of cancer—scirrhus, medullary, or colloid.

Cancer of the intestines, like all cancers, occurs chiefly, if not invariably, in advanced life.

The clinical symptoms of cancer of the intestines are only in a part of the cases so pronounced that we can make a positive diagnosis of the disease. The symptoms vary with the location of the growth.

Cancer of the rectum begins usually with distress at stool (tenesmus) and pain in the rectum, which at first comes only with defæcation, but later becomes almost continuous. The pain often shoots into the neighboring parts—the sacrum, the thighs, the genitals, etc. The local symptoms gradually increase, the stools often contain some mucus and blood, and diarrhœa alternates with obstinate constipation. The patient also becomes emaciated, and constantly grows weaker and more miserable. Finally, we often find a complete paralysis of the sphincter ani, so that a mucous, bloody fluid constantly oozes from the half-open anus. An absolute diagnosis can usually be made with ease by digital examination of the rectum. This examination should never be omitted in any suspicious case, for otherwise it is only too easy to make a mistake. Many a fatal case might perhaps have been saved by a timely diagnosis. External piles are particularly common in cancer of the rectum, and their presence should by no means prevent the physician from an internal examination. If there is cancer of the rectum, the finger upon being introduced detects an irregular, firm growth, and we can usually make out with approximate accuracy its extent and its invasion of neighboring organs, such as the vagina and bladder. Examination with the rectal speculum sometimes makes the diagnosis more accurate. In some cases the destruction of the new growth may cause perforation into the organs mentioned, and we can easily understand the results of this, such as cystitis, purulent discharges from the vagina, etc. We may also have peritonitis from perforation. Secondary cancer appears with especial frequency in the liver, also in the peritoneum and elsewhere.

Carcinoma of the colon causes, as a rule, only very indefinite symptoms, which for a long time are hard to interpret. These symptoms consist chiefly of distress at stool, obstinate constipation, dull pains in the abdomen, and the signs of slowly increasing general weakness and emaciation. If the stenosis of the intestine increases there are usually attacks of colic, with the gradual or perhaps rapid development of all the symptoms of obstruction of the bowels (*vide infra*, Chapter XI). In many cases the shape of the fæces is peculiar. They are ribbon-shaped, or consist of queer flattened nodules which have a certain resemblance to sheep's dung (scybala). Not infrequently we find in the dejections an admixture of mucus, pus, and, what is still more important, blood; if blood is persistently present in the movements (to be determined by the guaiac-turpentine test, *vide supra*, page 422), or if there are bloody or muco-purulent masses in the discharges, cancer is suggested. Exceptionally there may be found in the stools small detached portions of the new growth. The examination of the abdomen is often negative, but sometimes it is possible to feel the new growth through the abdominal walls as a distinct tumor. One characteristic of the tumor occasioned by intestinal carcinoma is that it may be very movable, and change its position because of its own weight or of the collections of fæcal matter. As a rule, the growth does not move with respiration, except possibly in carcinoma of the transverse colon. It is easy to confound intestinal carcinoma with carcinoma of the stomach, the mesentery, or the mesenteric lymph-glands. It is very important to know that in case of intestinal carcinoma the tumor which is felt may be not the new growth at all, but the hardened fæcal masses collected above it. Such fæcal accumulations may cause all sorts of errors as to the situation and dimensions of the new growth. In such cases a correct conclusion may not be obtained until after continued observation and the use of laxatives and enemata. It is also true that ordinary fæcal masses may be mistaken for cancer of the intestine when there is no intestinal disease of any sort. In exceptional cases there may be perforation of cancer of the colon into the stomach, with the formation of a gastro-colic fistula; or perforation of cancer of the sigmoid flexure into the bladder or into the peritoneum, with a secondary peritonitis, which may be localized or diffuse. Carcinoma of the cæcum is often indistinguishable for a long while from tumors due to chronic typhlitis, or perityphlitis. Cancer is suggested by the age of the patient, the tedious course, and the progressive aggravation of the illness, and sometimes also by swelling of the inguinal lymph-glands. In the surgical wards in Leipsic we observed a case of cancer originating in the vermiform appendix, which penetrated through the skin.

Cancer of the small intestine is very rare. It is distinguished by the very great movability of the tumor, if one is found. The symptoms are those of gradually increasing stenosis (*vide infra*). Cancer of the duodenum is similar in many respects to cancer of the stomach, particularly of the pylorus. It leads to stenosis of the duodenum, and consequently to dilatation of that portion of the duodenum which is above the new growth, and of the stomach, and so occasions the well-known symptoms of gastrectasia (*vide supra*, page 438). Cancer which is seated in the neighborhood of the common duct usually causes excessive and persistent jaundice.

The course of all cases of intestinal cancer is absolutely unfavorable. The disease may last for a considerable time, about two or three years. On the other hand, the duration of the special symptoms may be brief—that is, a few months or weeks; undoubtedly because the condition existed long before it caused symptoms. The final result in intestinal carcinoma takes the form either of increasing general weakness, or of perforation with fatal suppurative peritonitis. Death may also be occasioned by extensive and foul suppuration in the surrounding connective tissue, or by phlebitis and pyæmia. Most cases run their course as a more or less rapid obstruction of the intestine, which may become almost complete. Sometimes, however, ulceration of the cancer occurs, and there is a temporary abatement of the symptoms of stenosis.

The only possibility of successful treatment in intestinal carcinoma is by surgical methods. The results of operation are very favorable in cases of cancer of the rectum if the disease is not situated too high, and has not extended too far. Cancer of the other parts of the intestine has a much less promising outlook. If surgical interference is impracticable, treatment must be merely symptomatic, with a view of alleviating suffering.

We will append a few remarks about *sarcoma of the intestine.* This is a very rare disease, which originates from the submucous layer or else, in the form of lympho-sarcoma, from the lymph-follicles of the intestine, particularly of the small intestine. Both varieties exhibit a tendency to extend cylindrically over considerable areas along an intestinal coil. They usually do not lead to stenosis, but they may even cause dilatation of the affected portion of the intestine. The tumors which are formed may attain to very considerable size. The most prominent symptoms are general weakness and anæmia, associated with slight fever. These constitutional symptoms are more prominent than the local ones. The course is usually rapid and ends, after six to twelve months, in death.

CHAPTER IX

HÆMORRHOIDS

By the name "hæmorrhoids" we mean diffuse or varicose dilatations of the hæmorrhoidal veins, especially of the venous plexuses at the lower end of the rectum. Hæmorrhoids are single large varices, which usually rise from the submucous layer, and push the mucous membrane out before them. If they are situated outside of the sphincter ani we speak of external hæmorrhoids, in distinction from internal hæmorrhoids, which lie above the sphincter. The size of the nodules varies with the fullness of the dilated veins; but hæmorrhoids, as a rule, do not consist exclusively of dilated vessels, for we often find, at the same time, considerable thickening of the surrounding connective tissue, so that the whole mucous membrane has a swollen appearance, with a polypoid proliferation in parts. The hæmorrhoids usually present themselves as bluish tumors, from the size of a pea to that of a walnut, which surround the anus like a garland. Most of them have a broad base, while others are apparently pedunculated.

The chief cause of hæmorrhoids is frequently-repeated stasis in the veins affected. The position of the hæmorrhoidal veins is such that their contents are constantly pressing downward by the action of gravity, and the development of varices is further promoted by their complete lack of valves. Special local conditions often contribute also to render the escape of blood difficult; thus hæmorrhoids are particularly frequent during pregnancy, also in persons with habitual constipation, and consequently in individuals who follow sedentary pursuits. Hæmorrhoids are sometimes found as a result of portal congestion (in hepatic cirrhosis and similar conditions), and finally in general circulatory disturbances due either to pulmonary or cardiac disease; but these cases are not so frequent as was formerly supposed. Quite often, however, we can discover no sufficient cause for the development of the disease, and we are then forced to the hypothesis of a local disease of the affected venous plexus, which is probably often connected with an individual, and apparently sometimes hereditary, predisposition. We most frequently see hæmorrhoids in men in middle life.

Hæmorrhoids sometimes cause only slight symptoms, or none at all, but in other cases they are a tedious, burdensome, and even distressing evil. The chief symptom is pain, which is felt as a constant burning at the anus, but which increases to great severity at each dejection. There is much pain when the hæmorrhoids and the surrounding tissue gradually get into an inflamed condition. In the skin about the anus, erythema, excoriations, and sometimes small but very painful fissures are formed. The mucous membrane at the lower end of the rectum is often found in a catarrhal state, which gives rise to the presence of pus and mucus in the dejections—"mucous hæmorrhoids." The worst symptoms are caused when by pressing and straining at stool an internal pile is forced outward, and constricted by the sphincter. If the constriction is not speedily relieved the extruded varix becomes much swollen and inflamed. This change is favored by a cramp-like contraction of the sphincter. Under such circumstances there may result a purulent phlebitis associated with very great pain, high fever, and severe constitutional disturbance. Sometimes we find a thrombus, thrombo-phlebitis, or suppurative periproctitis, even if there is no previous constriction.

Hæmorrhoidal bleeding is a frequent and familiar symptom, which rises from a rupture of the dilated veins, and usually comes on at stool. The hæmorrhage is seldom very large, so that the loss of blood is in itself very rarely dangerous. Still, the rectal hæmorrhage may be repeated so often as to cause severe and persistent anæmia. The swelling of the varices after the hæmorrhages have ceased explains why the hæmorrhoidal symptoms are apt to be less marked as long as there are hæmorrhages than when there are none. Hence the old term of the "golden vein" for hæmorrhoidal bleeding.

Besides the local symptoms mentioned, in the anus, there are sometimes other symptoms which are due to an implication of the neighboring venous plexuses, the vesical, prostatic, and sacral plexuses. There is often pain in the sacral region, difficulty in micturition, and in women vaginal catarrh, anomalies of menstruation, etc. Since the symptoms of some co-existing morbid conditions, such as abnormal corpulency, or chronic gastro-intestinal catarrh, may be added to the general picture, we can comprehend why medical superstition has found in hæmorrhoids an excuse for the strangest ideas, like that of "transposed hæmorrhoids"! The scientific physician should be careful not to refer such manifold symptoms to hæmorrhoids without due consideration, for otherwise important diseases may be readily overlooked.

Treatment.—The treatment of chronic piles consists mainly in caring for a regular and easy evacuation of the bowels. This is the best means to diminish the local discomfort (see the next chapter). The diet to be prescribed should be determined by the constitution of the patient. It is usually advisable to limit the ingestion of milk and to recommend in its stead a larger proportion of vegetables, in a broad sense, including fruit, such vegetables as grow below the surface of the ground, simple puddings, and rice. It is usually advantageous to prescribe a sufficient amount of physical exercise, also warm or cool sitz-baths, and cool sponging with rubbing. Moreover, laxatives are to be considered, particularly waters containing Epsom salts, also the regular use of injections of cold water or oil; compound licorice powder, or other laxative powders or pills; and perhaps a course of the waters at Carlsbad, Marienbad, Kissingen, or Tarasp. A laxative used with special frequency in the treatment of piles is sulphur. This is the chief constituent of most "pile powders," e. g.:

℞ Sulphuris loti,
Potassii bitartratis, } ....āā ℥ss. (grm. 15);
Sacchari albi,
Elæosacchari citri,* } ....āā ʒijss. (grm. 10). M.

The local treatment consists chiefly in protecting the piles from irritation. The parts are best cleaned after every movement of the bowels with cotton or soft linen, and the region of the anus is to be touched with oil, vaseline, or borated vaseline. If there are slight inflammatory symptoms we apply compresses wet with a solution of acetate of lead, or ice-water, or inject olive-oil into the rectum, or try suppositories containing morphine or cocaine. If the inflammation is more severe a few leeches are often beneficial. Constricted piles we should endeavor as soon as possible cautiously to replace with the finger. The pain attending this operation is often so severe that chloroform narcosis, or at least the local application of cocaine, is required. If there is suppurative phlebitis an incision is necessary. In some cases a radical operation for the piles may be performed at the same time.

The bleeding from piles is, as we have said, seldom severe enough to demand special treatment, such as the use of ice, chloride of iron, or packing the rectum. If the hæmorrhage is repeated and serious, or if the inflammation causes persistent and extreme discomfort, we should urgently recommend operative removal of the piles. Particulars as to the methods employed for this purpose, such as the use of the thermo-cautery or the knife, will be found in the text-books on surgery.

[Persistent and intelligent treatment will often bring about very great relief, or even complete cure, in chronic cases. There are many local applications, each of which has its warm advocates—so many that it is not possible here to go into details. Suffice it to say that an astringent with or without an anodyne may be used as an enema, in suppository or in ointment. The fluid extract of hamamelis, glycerine, and some other remedies given by the mouth, are reported to have afforded good results.]

CHAPTER X

HABITUAL CONSTIPATION

PERSISTENT tendency to constipation is a frequent symptom in many different acute and chronic cases. We often see persistent sluggishness of the bowels in all sorts of conditions of weakness, in general anæmia, in diseases of the stomach, intestines, liver, lungs, heart, and nervous system, and it is caused by a great variety of circumstances. Often the weakness of the peristalsis of the intestine, and particularly of the colon, is merely one manifestation of a general debility; in other cases the activity of the intestine is subnormal because of the insufficient and unstimulating diet, or because of the lack of physical exercise. In a third class of cases there is disease of the intestinal wall itself, such as catarrhal conditions, circulatory disturbance, atrophy of the muscular coat, or constriction of the lumen of the intestine. And finally, a fourth cause is disturbance of the innervation of the intestine, because of disease of the brain or spinal cord.

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* Powdered sugar, ʒj; volatile oil, gtt. ij. M.

The above are examples of symptomatic constipation, but in this chapter we shall not discuss these, but rather cases of habitual constipation occurring as an apparently independent disease. This may appear alone or associated with other disturbances likewise purely functional. The causes of this frequent and important condition are by no means invariable, and we are still far from a clear understanding of the nature of all the disturbances that belong in this category. Sometimes habitual costiveness seems to be associated with a congenital functional weakness of the muscular coat, or perhaps of the nervous apparatus of the intestines. At any rate, it is often possible to trace the symptoms back to the earliest childhood. In other cases it is apparently an improper mode of life which exercises a disturbing effect upon the intestinal activity; lack of bodily exercise often leads to disorders of the bowels, and, still more, irregularity in eating and an improper choice of food. Sometimes external mechanical influences promote constipation. In women unsuitable apparel, tight bands around the waist, tightly-laced corsets, and the like, certainly produce in many cases a permanent malposition of the intestinal organs ("enteroptosis," compare above the chapter on gastroptosis, page 460), and particularly of the large intestine. It is not improbable that these conditions are in part the cause of the chronic constipation which is so frequent in women and girls. In women frequent pregnancies may also exert a causative influence by inducing laxness of the abdominal walls and a tendency to enteroptosis.

Habitual constipation is associated still more frequently with general neurasthenia than with any of the conditions thus far enumerated (compare the chapter on neurasthenia, page 1193). The nature of this association is probably not the same in every case. Often neurasthenia and constipation seem to be co-ordinate symptoms; sometimes the existing constipation has an unfavorable effect upon the psychical condition of the patient, rendering him feeble, fretful, out of sorts, and nervous. As a rule, however, neurasthenia is the primary disease, and then the irregularity of the bowels appears as a result of abnormal nervous influences, or of secondary conditions (*vide infra*). Often the two states act in a vicious circle, each sustaining and aggravating the other. The most essential factor is often the hypochondriacal tendency of the patient. He ascribes an excessive importance to irregularity of the bowels, and regards every slight disturbance as of extreme gravity. Even when the movements are sufficient he can not free himself from a disordered apprehension that they are not really adequate. Such patients come at last to devote almost all their thoughts to their illness and make the most painstaking observations and investigations of their dejections, having energy for nothing else and no joy in life. They seek aid from all sorts of physicians and quacks, and yet they have no actual confidence in any one and do not follow the practitioner's directions with the requisite persistence. In many of these cases the constipation is only apparent, or imaginary. The patient fears to eat any hearty food and takes little nourishment, and consequently the excreta are scanty. In other instances, however, the abnormal psychical conditions have a direct inhibitory influence upon the intestinal activity. The intestine gets out of the habit of regular action. Moreover, the unwise use of internal laxatives may entail actual disease of the intestine.

Consequently, if the physician wishes to form a reliable opinion with regard to the intestinal function in a case of chronic constipation from actual observation, the first requisite is that he should himself see the dejections of the patient for some time, while the diet is normal and all laxatives are omitted. This is much easier in a hospital or sanitarium than in ordinary private practice. When a patient is under observation in this way it should be noticed whether the abdomen becomes much distended, and whether hardened fæcal matter can be felt in the colon or the sigmoid flexure. Furthermore, the dejections should be examined as to their amount and character. They may seem nubby, hard, or as if they were "burned," or of abnormally small diameter, or flattened, or covered with mucus. If we find that the evacuations are distinctly abnormal we infer that there is actual disturbance of the intestinal functions; while, on the other hand, our examinations often show that the complaints are exaggerated or imaginary.

The **treatment** of habitual constipation is a difficult and often a thankless task, and it demands patience and professional tact. It goes without saying that we must first of all look for the causal factors. If we succeed in improving the underlying disease—as, for instance, the chronic gastro-intestinal catarrh, the chronic affections of the heart or lungs, the anæmic conditions, or certain nervous troubles—a regulation of the bowels often follows of itself. In ordinary habitual constipation we must first attend to the patient's diet. Since most of these patients also suffer from symptoms of nervous dyspepsia, they are usually very careful in their diet, and take only a little, easily digestible, and chiefly liquid food. It is no wonder that no good dejections follow such food. Improvement can be obtained, in such cases, only by eating plenty of food which can mechanically stimulate the intestine. Hence we must try to bring the patient back to ordinary "household fare"—to take, besides plenty of meat, a sufficient amount of bread, vegetables, etc. It is a very good thing to recommend especially certain kinds of bread, such as Graham-bread or rye-bread, and also larger amounts of butter, besides fruit, prunes, grapes, apples, pears, dates, and figs either raw or boiled, almonds, nuts, and honey. It is often efficacious to have the patient drink a glass of cold water in the morning before breakfast. Of true laxatives we should be very sparing, for the patient readily becomes accustomed to them, and the dose has to be constantly increased. Among the milder laxatives the various waters which contain Epsom salts, such as Friedrichshall, usually one or two wineglassfuls, are prescribed. For more continued use we may also recommend tamarinds, rhubarb, aloes, jalap, cascara sagrada, and extract of frangula. It is not possible in this connection to give a minute description of all these remedies. They are prescribed in the most varied combinations in the shape of pills and powders. It is often necessary to make repeated changes in the medicine and its dose before the most effectual prescription is hit upon, for there are many individual peculiarities in this regard. There are patients with simple habitual constipation who for years take, every evening, some powdered rhubarb, or one or two laxative pills, with a good result and feel perfectly well. In most cases, however, the efficiency of the internal laxatives gradually fails, the patient is obliged to take more and more powerful remedies, or increasing doses, in order to get any effect. In such instances it is always best to abandon internal laxatives altogether, and in their stead to employ external physical agencies, or to stimulate the rectum by means of injections, irrigations, and the like.

The regular use of injections, if carried out in a proper manner, may be continued for a long time without harm. The practice may indeed be carried to an injurious excess, and occasion at last a catarrh of the rectal mucous membrane, because of the constant local irritation, or, at any rate, a needless dependence upon local stimulation. The simplest way of making an injection is by means of the fountain syringe. There are also various shapes of injection syringes used by many. For a fluid we choose simple warm water, or, what is often more efficient, cold water. We can strongly recommend the employment of soapsuds (a teaspoonful of powdered soap to a pint or two of water). Lately, again, injections of oil have been in great vogue. The patient is placed on his left side, and by the means of a fountain syringe, eight to sixteen ounces (250 to 500 grammes) of fresh poppy oil [or fresh cotton-seed oil] (olive-oil is much dearer) is slowly introduced into the rectum. Small injections of glycerine, one to two drachms (grammes 5–10) are also effectual. Still more convenient and often satisfactory is the employment of glycerine suppositories, which are dissolved in the rectum by the heat of the body.

The external physical remedies employed in habitual constipation are massage, electricity, and hydrotherapy. Regular massage is often attended with good results. The patient is stroked with the flat of the hands with moderate or even with considerable pressure, beginning at the cæcal region, and proceeding along the colon to the sigmoid flexure. There may also be cautious kneading of the abdomen. A ball may be used for massage. This is very convenient, and can be carried out by the patient himself. An iron ball, weighing about five pounds and sewed up in flannel, is rolled for several minutes in a spiral course over the abdomen, its general direction being that of the hands of a watch. The electrical treatment consists of faradization of the abdominal walls, or galvanization of the intestine, one electrode being placed upon the back. In the way of hydrotherapy, cold rubbing and sponging are employed, and are often associated with massage. In addition to these methods medical gymnastics may be simultaneously practiced, with a view to regular bodily exercise, and the invigoration of the abdominal muscles.

In the treatment of habitual constipation associated with hypochondriasis, the first rule is to treat the patient's mental condition. We should not make merry over his trouble, nor should we rudely let him feel that we do not consider his complaints so important as he himself imagines. The patient does not deserve to be scoffed at, since his subjective symptoms are to him of the most urgent nature; but it is extremely important to divert his thoughts from his trouble. As in many other reflex processes, so in defæcation, the voluntary attention abnormally directed to it has an inhibitory action. Hence we admonish the patient to think of his trouble as little as possible and to begin his regular activities again, and we try to convince him of the groundlessness of his fears. The cathartics, which most patients have already taken freely without the desired action, are usually of no advantage at all. On the contrary, it is almost always necessary to forbid the patient to use cathartics at all.

We should not only employ a suitable dietary (*vide supra*), but we should seek to stimulate the intestinal peristalsis by such external means as massage and electricity (*vide supra*). It is true that a considerable part of the success of these methods is due to their psychical effect upon the patient, but there is no doubt, also, of their direct invigorating influence upon the muscular coat of the intestine. We must not omit a proper general treatment: cold sponging, baths, a country residence, and sufficient physical exercise. Finally, it is often very useful to cause the patient to go to the closet at a fixed hour daily, even if there be no special desire for a stool, and to try to have a movement of the bowels. Thus a sort of training and education of the bowels is achieved, even when the patient at first believes it impossible.

By these means only do we succeed in giving the patient renewed courage, and sometimes finally in attaining recovery even in severe and persistent cases. In all cases of habitual constipation we should, however, not only endeavor to hit upon the proper means for making the bowels act, but if it is in any wise possible we should also strive to arrange the patient's life so sensibly and suitably that he will have regular and satisfactory evacuations without any special means. To accomplish this, it is often requisite that we should persuade the patient to give up all laxatives, no matter how constipated he may be. Such a course is naturally much more practicable in an institution than in a private house. The author has himself repeatedly treated patients with habitual constipation simply by regimen—i. e., by proper diet, cold sponging, and sufficient exercise or gymnastics, without any other remedies. In such patients the stool would often be delayed for five, eight, or ten days, and yet, what is remarkable, without any special inconvenience. Then at last the bowels would move spontaneously, or with the aid of a small injection of cold water, and would gradually assume complete normal regularity.

CHAPTER XI

STRICTURE AND OBSTRUCTION OF THE INTESTINES

Ætiology and Pathological Anatomy.—Different pathological processes may lead to stricture or complete obstruction of the intestinal tube in different parts. Since in this affection the purely mechanical effect of the intestinal stenosis is the chief cause of the clinical symptoms, the type of the disease is very similar in all the cases of this class, in spite of the manifold anatomical causes. Hence, after enumerating the individual affections which may lead to stricture of the intestines, we shall describe their symptoms in common.

The anatomical causes of stricture or obstruction of the intestines are as follows:

1. *Congenital closure* of the intestines is found at the anus, *atresia ani*, and much less frequently in the colon or small intestines. The form first mentioned is the only one of clinical interest, since it may be relieved, at least in some cases, by operation. All the other forms of congenital closure of the intestines are incompatible with a long duration of life.

2. *Tumors and Cicatricial Strictures.*—Cancer of the intestine is the only tumor that has any clinical significance. We have already described its most important anatomical relations and the possibility of intestinal stenosis as a result of it.

We see cicatricial strictures most frequently in the large intestine after recovery from dysenteric ulcers. The syphilitic stenosis of the rectum, which we have already described, is also of practical importance. Syphilitic ulcerations with cicatricial stenosis also occur in higher portions of the colon, perhaps even exceptionally in the small intestine. Typhoid ulcers scarcely ever lead to cicatricial stenosis. Strictures consequent upon tuberculous ulcers of the intestine are somewhat more frequent than typhoid strictures, but still they are very rare. Cases have been observed in which there were several tuberculous strictures of the intestine, while no sign of tuberculosis could be found elsewhere in the body. Stenosis of the duodenum after the healing of a duodenal ulcer (*vide supra*) resembles, in its clinical symptoms, stenosis of the pylorus, and not stenosis of the intestines.

3. *Intestinal Obstruction.*—The most frequent form of intestinal obstruction comes from the impaction of fæces. From the different conditions which cause

enfeeblement of the peristaltic movements, an accumulation of fæces (*coprostasis*) may arise, especially in the colon. This accumulation gradually but constantly increases until it leads to well-marked symptoms of intestinal obstruction, and in cases of obstruction from other anatomical causes, fæcal impaction not infrequently aggravates the condition.

We see obstruction of the intestines from other causes much less frequently than from impaction of fæces. In some cases impacted gall-stones have been found, especially in the lower part of the ileum and in the neighborhood of the ileo-cæcal valve, almost completely stopping up the lumen of the intestine. Genuine intestinal calculi (composed of phosphates) may exceptionally lead to obstruction. Likewise, occasionally conglomerations have been found, composed of indigestible portions of food, vegetable fibers, the seeds of fruit, potato skins, grape skins, and also clumps of round-worms. We must also mention here the very rare cases in which a large foreign body has been swallowed and wedged itself into some part of the intestine. Such a thing has been seen, especially in children and among the insane.

4. *Intestinal Constriction.*—Although the mechanism of intestinal constriction in external herniæ lies in the domain of surgery, we must mention here the chief causes of the so-called internal intestinal constriction, internal incarceration, or strangulation. In the abdominal cavity itself pouches and diverticula are found, as either normal or abnormal formations, in which single loops of intestine may be caught and constricted. The duodeno-jejunal hernia—the so-called Treitz's retro-peritoneal hernia—is worthy of special mention, and comes from the entrance of a loop of intestine into the duodeno-jejunal fossa. This hernia may become very large. It is sometimes found by accident in the cadaver, not having caused any symptoms during life, but in rare cases it may be the cause of acute internal constriction. We must also mention the hernia of the omental bursa—where a loop of intestine passes through the foramen of Winslow—the intersigmoid hernia, the subcæcal hernia, etc. Diaphragmatic hernia is of greater practical significance because it is somewhat commoner. By this name we designate both genuine protrusions into the diaphragm, and also the passage of abdominal viscera through congenital or acquired (traumatic) defects in the diaphragm. These herniæ may exist without symptoms, or at least without causing any signs of severe disease, but in some cases they cause obstruction by constricting or twisting a dislocated loop of intestine.

Those cases in which abnormal slits and holes in the omentum or mesentery give rise to internal constriction are to be added to the list of the internal herniæ. Finally, abnormal cords, membranes, and false ligaments in the abdominal cavity are a comparatively frequent cause of internal constriction. Such cords and bands are sometimes left as the results of a former peritonitis, and may cause constriction or kinks of single loops of intestine. Small diverticula of the colon sometimes form adhesions due to localized peritonitis, as we have ourselves seen, and thus occasion dangerous intestinal stenosis. One such false ligament, which must be specially mentioned, is found as a prolongation of Meckel's diverticulum. By this we mean that diverticulum which must be regarded as the remains of the omphalo-mesenteric duct, still persisting, which has its seat, corresponding to the duct, from half a metre to a metre above the ileo-cæcal valve. A firm cord sometimes arises from the free end of this diverticulum, the obliterated omphalo-mesenteric vein, which adheres to some part of the internal abdominal wall and may cause constriction of the intestine. Adhesion of the free end of the vermiform appendix has been the cause of internal constriction in some cases.

5. *Twists (volvulus) and Knots of the Intestine.*—Twists about the mesenteric axis, and complete constriction of a portion of intestine from this cause, are seen most frequently in the sigmoid flexure, especially if the mesentery of the flexure

is unusually narrow congenitally. The spontaneous correction of this abnormal condition is hindered by the weight of the loops of intestine filled with gas and masses of fæces, and by other portions of intestine lying on the place of twisting. Sometimes other portions of intestine wind themselves several times about the pedicle of the twisted loop so as to form a regular knot. Such twistings have been seen especially between the sigmoid flexure and a portion of the ileum. External injury sometimes gives rise to the formation of a knot. In some cases abnormally great peristalsis, severe diarrhœa, precedes the appearance of obstruction. We have observed a fatal case of volvulus in the highest part of the small intestine, consequent upon violent vomiting, excited by a remedy administered by a quack, for tapeworm. A most important factor in the development of the symptoms of twist is the mechanical strangulation of the blood-vessels of the intestine. This causes a great disturbance of the circulation, leading to paralysis, tympanitic distention, and final gangrene of the intestinal coils which are involved.

6. *Invagination of the Intestine (Intussusception).*—If a portion of intestine is pushed into the lumen of the portion that lies next below, we term the process invagination. The cause of this is usually assumed to be a diminution or a complete absence of peristalsis in a circumscribed portion of intestine. If now there are energetic movements in the portion immediately above, they push this into the paralyzed portion. According to Nothnagel, the beginning of the invagination is a spastic contraction of a limited portion of the intestine, in the shape of a ring. This furnishes a fixed point, and the contractions of the longitudinal muscles of the intestine below the constricted area are said to draw the lower portion of the intestine upward over this ring.

In the post-mortem examinations of children we frequently find intussusception of the ileum, which has occurred shortly before death and so is of no clinical importance. Sometimes intussusception has occasioned the most severe symptoms of intestinal obstruction. It occurs particularly in children up to the tenth year, and it may be of sudden onset without any apparent cause. Intussusception of this sort, which often involves a considerable extent of the intestine, may have its seat at almost any part of the bowels. The intussusception most frequently involves the cæcum and the lowest part of the ileum, pushing thence into the colon (*invaginatio ileocæcalis*); intussusception of this sort may be so extensive in children that the invaginated ileum reaches into the rectum, or even projects from the anus. There are cases in which the ileum alone is involved, and less frequently the colon alone. The invaginated portion of intestine usually suffers compression of its blood-vessels, particularly the veins. This is followed by inflammation of the constricted portion, and the inflammation often extends to the peritoneum. Not infrequently there is gangrene of the inner portion of the intestine, as a result of strangulation of the afferent vessels. The necrotic portions may slough off, and be discharged at stool. Such an occurrence has, in a few recorded cases, led to spontaneous recovery from the intussusception, and from the obstruction which it had caused.

We must mention intestinal polypi as a special cause of intussusception, as they gradually pull that portion of the intestine in which they are situated into the neighboring portion next below by their weight. This has been repeatedly observed.

7. *Compression of the Intestine from without*, by tumors of the uterus, ovarian cysts, pelvic abscesses, omental tumors, etc., has been met with in rare cases as a cause of intestinal stenosis. The symptoms of stricture in such cases develop either very gradually or sometimes rather suddenly.

We must now mention certain pathological changes which may follow every obstruction, from whatever causes it may arise.

The further changes in the intestine deserve the chief attention. Above the constricted point it is usually greatly swollen from gas and the accumulation of fæces. But it should be noted that the abundant fluid found above the narrow part of the intestine is by no means due exclusively to the ingesta, but probably is largely the result of transudation, and the secretion of intestinal juices. The intestinal contents are very apt to undergo putrid decomposition. This gives rise to a large amount of gas which may not be reabsorbed, so that above the obstruction there is apt to be a very marked tympanites. This may distend almost the entire intestine above the constriction, or the distention may be limited to the constricted coils in which circulation has been impaired, and in which consequently there is a greater tendency to decomposition and the formation of gas. The intestinal coils thus distended, being irritated by their decomposing contents, usually soon become inflamed; their walls become permeable for bacteria, and thus all forms of intestinal obstruction lead to enteritis, and usually promptly to peritonitis. A severe diphtheritic process often develops in the intestine, with ulceration above the stenosis. In the inflamed, softened intestinal wall, thin from its abnormal distention, a little tear easily occurs in some spot, or more rarely a genuine perforation following an ulcer. Some of the putrefying contents of the intestine thus enter the abdominal cavity, and an intense purulent or ichorous peritonitis is unavoidable. This is why acute peritonitis is so frequent a lesion in persons who die of intestinal obstruction. If the intestinal stenosis has lasted a long time, we usually find in the upper portion of the intestine, besides the signs of inflammation, a manifest hypertrophy of the muscular coat, the result of the abnormally active peristalsis by which the muscle has tried to overcome the obstacle. The intestine below the constriction, in contrast to the part just described, appears narrow, contracted, and empty.

The changes in the other organs correspond to the general inanition. The frequent development of inhalation-pneumonia is easily explained, if severe vomiting has preceded (*vide infra*).

Clinical History.—In regard to the clinical symptoms we must distinguish the cases with a rapid, complete obstruction of the intestine from those in which the condition develops gradually, and where there is, therefore, at least for a time, merely a constriction of the intestine.

1. **Intestinal Constriction.**—The first symptom of the partial intestinal constrictions, which arise from cicatricial strictures and new growths, from partial blocking up of the lumen, from intussusceptions, etc., is usually a disturbance in defæcation. The bowels are costive, they move only at long intervals, and their motion is often associated with pain and tenesmus. In the description of cancer of the intestines we have already mentioned that the fæces passed sometimes have a peculiar, flat, compressed, or scybalous form. Blood and mucus are often mixed with the dejections and are due to the character of the primary disease. In some cases there is no constipation, and there may be even constant diarrhœa. We can easily understand from the physiological conditions that in stenosis of the small intestines, whose contents have an approximately fluid consistency, disturbances of defæcation are less apt to take place than in stenosis of the large intestine, where the fæcal masses have already assumed a more firm consistency.

Physical examination of the abdomen often gives important and valuable information. The abdomen is usually swollen by meteorism (*vide supra*). The intensity of the meteorism varies very much in different cases and at different times in the same patient. Meteorism is sometimes absent, especially in stenosis at the beginning of the intestine. There may then be gastrectasis. The marked peristaltic movements, plainly visible through the abdominal walls, are very characteristic of most intestinal contractions. The contour of single loops of intestine is often marked, at times quite sharply, and then we can sometimes feel the thickened intestinal walls through the lax abdominal wall. As a rule, these vigorous contractions are associated with severe colic. We may often decide upon the seat of the stenosis from the location and course of the visible peristaltic movements. In general, it is true that peristalsis is much more noticeable when the stenosis is in the small intestine than when it is in the colon. We must finally state that we have been repeatedly struck by the great extent and strength with which we could feel the pulsation of the aorta through the swollen loops of intestine. If we put our ear to the anterior abdominal wall we can often hear many gurgling and splashing noises, which sometimes have a distinct metallic quality. Eructations are frequent, and in some cases there is occasional vomiting.

The duration of all these symptoms varies with the nature of the primary lesion. Of course the greatest importance attaches to the condition of the muscular layer of the intestine above the constricted spot. For a time the muscle may overcome the obstruction because of its gradual hypertrophy, but at last the muscular coat becomes insufficient, and consequently the symptoms become more severe. This explains why the symptoms of intestinal stenosis change gradually or suddenly into those of complete intestinal obstruction. With this change we have grave symptoms. Sometimes, in cases of chronic stenosis of the intestine, some temporary causes may occasion repeated attacks of intestinal obstruction, which, however, are relieved and give place again to the milder symptoms of mere stenosis.

The duration of all these symptoms differs with the form of the primary disease. Either gradually or sometimes quite suddenly the symptoms of intestinal constriction pass into those of obstruction. Then the same type of disease develops as is seen in all acute internal strangulations.

2. **Intestinal Obstruction.**—The symptoms of intestinal obstruction (ileus) form one of the severest and most frightful conditions known to pathology. The patient undergoes a rapid change for the worse. The signs of collapse quickly develop; the face sinks in and assumes a wasted and sharp expression, the extremities become cool and livid, the pulse is frequent and can scarcely be felt, the voice is weak and lifeless. Respiration is impeded by the upward dislocation of the diaphragm. The temperature usually falls, but it occasionally rises. As a rule, there is profuse perspiration. The abdomen is much swollen from meteorism, and is usually very tender on pressure from beginning peritonitis. The passage of fæces and the escape of flatus cease entirely. We often see the peristaltic motions of the intestines above the obstruction through the abdominal walls, but in some cases the muscular coat is so paretic that it is no longer capable of marked peristalsis.

The most characteristic symptom of intestinal obstruction is the vomiting of feculent-smelling masses, the so-called stercoraceous vomiting. There is often frequent eructation at the beginning of the attack. The vomitus at first is of the usual character, but it soon acquires a manifestly putrid, fæcal odor, and becomes finally precisely like the stools in diarrhœa. The old opinion is false that in this vomiting real fæcal masses were forced backward from the large intestine into the stomach by an antiperistaltic action of the intestine. Stercoraceous vomiting occurs, not only when the obstruction is in the large intestine, but also (and oftener) in obstruction of the small intestines. In this case we have to do with a putrid decomposition of the contents of the intestine stagnating above the obstruction (*vide supra*). Part of this putrid mass reaches the stomach because of accumulation and regurgitation, since the pylorus gradually yields to the increasing swelling of the small intestines. It is very likely that retrograde "antiperistaltic" movements of the intestinal muscles contribute to the passage upward of the intestinal contents. The vomiting itself is probably caused in large part by the irritation of the peritoneum, and perhaps by the stimulation of the abnormal matter which has entered the stomach. If death does not occur in a short time, the clinical picture exhibits distinct indications of secondary peritonitis; the temperature rises a little, the skin grows dry and hot, and the abdomen is very tender upon pressure.

We must mention, finally, the important changes in the urine which occur in the various forms of constriction. In the contents of the intestine, stagnating above the stenosis, large amounts of indol and phenol are formed from the decay of the albuminous substances, along with other products of decomposition; they are in part absorbed and excreted with the urine. Hence, in stenosis of the small intestines we often find that the urine contains an increased amount of indican * (Jaffé) and phenol (Brieger), but in stenosis of the large intestine the amount of indican in the urine is not increased, because the albuminous substances capable of decomposition are no longer present in the contents of the large intestine to cause it.

The course of intestinal obstruction differs according to the anatomical causes which exist in different cases. In many cases of acute internal strangulation the severe type of general disease above described develops in a very short time, and may lead to death in a day or two. In such cases death is caused by the poisonous action of the absorbed gases, generated by decomposition, or it may be due to reflex paralysis of the cardiac nerves. Usually, however, the course is somewhat longer, and occupies about a week. In intestinal obstruction which develops gradually from intestinal constriction, the disease may go on longer and show many variations in its intensity. In mere intestinal constriction we can make fewer definite statements as to the duration and course of the affection, since the symptoms of the disease depend entirely upon the form of the primary disorder.

In a great majority of cases intestinal obstruction terminates unfavorably. Death results either from increasing collapse or from secondary peritonitis (*vide supra*), or in rare cases from further complications, such as pyæmic conditions or pneumonia. Recovery (independent of operation) may occur even after the severest symptoms, but it is very rare. The obstructions from impaction are most hopeful for recovery. Impacted gall-stones, fæcal accumulations, etc., may be evacuated, after which the severe symptoms disappear. The possibility of recovery in intussusception, by throwing off the gangrenous internal portion of intestine, has been mentioned above. We can not wholly deny that internal strangulations are capable of restoration, although the prognosis must almost always remain doubtful on account of the uncertainty of the diagnosis in any individual case.

In the partial intestinal constrictions, too, the nature of the trouble causes an unfavorable termination in most cases, either from the primary disease itself or from the complete obstruction that finally follows, but the possibility of recovery can not be wholly excluded in certain conditions, such as impaction or external compression.

Varieties of Intestinal Obstruction.—Diagnosis.—To go into details as to the clinical symptoms of all the separate forms of intestinal constriction and obstruction would lead merely to repetitions. In most of the acute and many chronic cases the diagnosis can generally be made only as to the presence of a mechanical obstacle in the intestine, but the nature of the obstruction can at best be merely surmised. Yet in many cases we may reach a more exact diagnosis by careful questioning and examination. Above all, we should accurately examine all the known localities for external rupture, including the inguinal canal, the thighs, and the navel, and we should never omit to make rectal and vaginal examinations. Often valuable conclusions can be reached by inflation of the colon by means of a rectal tube attached to an ordinary Davidson's syringe, for in this way stenosis of the colon is sometimes rendered distinctly palpable. It is also very important to make a careful examination of the abdomen as early as possible, employing sight, touch, and percussion. We can often recognize the place of obstruction because of the localized protuberance, and the tenderness of certain intestinal coils. It is of the greatest importance to detect intestinal peristalsis, if present. With regard to distinguishing between stenosis of the colon and of the small intestine, stenosis of the small intestine is indicated by a special prominence of the central portion of the abdomen, by the visible and active peristaltic motion of many coils of intestine, by the occurrence of fæcal vomiting, and by the presence of a strong reaction of indican in the urine; while stenosis of the large intestine is characterized by a distention which corresponds more with the course of the colon, without much visible peristalsis, and a slow development of severe constitutional symptoms. We shall recur later to the differential diagnosis between intestinal obstruction and diffuse peritonitis, when considering the latter.

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* The indican test is performed in the following way: We mix equal volumes of urine and officinal hydrochloric acid (P. G.), and then add, drop by drop, a concentrated solution of chloride of lime shaking it after each drop. If now chloroform is added, upon shaking again, the chloroform will take on a striking blue color, if the urine contains any considerable amount of indican.

Of the special varieties of intestinal obstruction, we will call attention to two as of practical importance. Intussusception appears, as we have indicated, mainly in children. Its symptoms usually begin rather suddenly with violent abdominal pain. There soon appear, also, liquid dejections of bloody mucus or almost clear blood, originating from the constricted portion of the intestine. Often there is great tenesmus and protrusion of the anus. We have already mentioned that sometimes the invaginated portion of intestine may be felt in the rectum. The further course corresponds with the ordinary symptoms of intestinal obstruction, except that it should be said that fæcal vomiting does not occur in the ordinary ileo-cæcal intussusception, until comparatively late in the attack. The other form of intestinal obstruction which we wish to mention here is due to the accumulation of large amounts of fæcal matter in the rectum. We sometimes find monstrous accumulations of fæces in the rectum, especially in old women who have previously suffered from habitual constipation, or in whom constipation is due to some other affection. Severe symptoms usually come on quite suddenly, after long-continued mild prodromal symptoms, and these severe symptoms are much like the picture of internal strangulation—severe, sometimes colicky, abdominal pain, great tenderness of the abdomen, which is usually swollen, marked general collapse, loss of strength, a small pulse, an outbreak of cold sweat, vomiting, etc. If we try to give an enema in such cases, very little fluid runs into the rectum. On introducing the finger, it usually strikes solidly on old, hard, fæcal masses above the sphincter, and there is often nothing left but to undertake the dirty task of removing at least a part of the scybala with our own hands. We may then succeed, by repeated enemata and by giving cathartics internally, in removing sometimes quite an incredible amount of accumulated fæces, and in obtaining thus a rapid recovery from the condition.

[The diagnosis of the nature of an intestinal obstruction is so difficult in many cases, and yet so important with reference to treatment, that the editor ventures to introduce tables of differential diagnosis of the more common forms of the condition. These tables are based upon the masterly prize-essay of Treves, of London.

ACUTE INTESTINAL OBSTRUCTION

CHIEF COMMON SYMPTOMS.—Sudden *pain*, intermittent or constant, with exacerbations; tends to become constant with time. *Vomiting*, early, severe, becoming feculent. *Constipation*, more or less absolute. Abdominal *distention*. *Shock*.

	STRANGULATION BY BANDS OR THROUGH APERTURES (25 *per cent. of all cases of acute obstruction*).	VOLVULUS OF COLON.	ACUTE INTUSSUSCEPTION.
AGE AND SEX..	Young adults; rare after 40.	Males as 4 : 1 ; 40 to 60.	More than 50 per cent. under 10 years.
HISTORY.......	Previous peritonitis in 68 per cent.; previous attacks of obstruction in 12 per cent.	Previous constipation.	Usually negative.
ONSET.........	Sudden in 70 per cent.	Sudden.	Sudden in 75 per cent.
PAIN...........	Early, severe, continuous, with exacerbations.	Early, less severe, intermittent at first, becoming constant with exacerbations.	Early and severe: increasing and later subsiding; at first paroxysmal.
LOCAL TENDERNESS...	Absent at first, appears later.	Early over distended coil, and constant.	Common about a tumor.
VOMITING......	Early, marked; in 60 per cent. becomes feculent; affords no relief.	Less early, severe and constant; often affords relief.	Still less early and severe; in 25 per cent. becomes feculent.
CONSTIPATION..	Continuous and absolute; no blood.	Early and absolute; no blood.	Seldom absolute: diarrhœa not uncommon; blood in 80 per cent.
PROSTRATION..	Marked.	Rather less marked; may be dyspnœa.	Marked.
TENESMUS......	Absent.	In 15 per cent.	In 55 per cent., and often early.
ABDOMINAL WALL......	Flaccid unless peritonitis.	Rigid from early peritonitis.	Flaccid unless peritonitis.
TUMOR.........	Very rare.	Absent.	In 50 per cent.; invagination sometimes felt in rectum.
METEORISM....	Slight, appears about third day.	Early, rapid, increases, and is extreme.	Rare, unless marked constipation.

N. B.—No trustworthy conclusions can be drawn from the seat of the pain as to the seat of the obstruction unless local peritonitis comes on. The pain is usually referred in all forms to the region of the navel. In complete obstruction the pain is constant, though with exacerbations; intermittent pain shows that the obstruction is partial. Coils of intestine are not visible through the abdominal wall in acute cases.

CHRONIC INTESTINAL OBSTRUCTION

	STRICTURE OF THE SMALL GUT.	STRICTURE OF THE LARGE GUT.	FÆCAL ACCUMULATION.
AGE AND SEX..	Adults.	Adults.	Adults; more common in females, the hysterical, the insane, hypochondriacs.
HISTORY.......	Cancer, trauma, tuberculosis; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Cancer, trauma, tuberculosis, dysentery; disordered, imperfect, irregular action of bowels from time to time, with intervals of comparative ease.	Previous constipation.
ONSET.........	Gradual.	Gradual.	Gradual.
PAIN...........	Intermittent.	Intermittent.	Less prominent.
VOMITING......	Late, scanty, feculent only toward end of acute attack; may be provoked by food.	Less prominent, rarely feculent or provoked by food.	Late, scanty, rarely feculent, often absent.
CONSTIPATION..	May alternate with diarrhœa; blood points to cancer.	Form of fæces may be altered; blood points to cancer.	Gradually increasing: may be spurious diarrhœa; no blood.
TENESMUS......	Absent.	Often present.	Absent.
METEORISM.....	Not marked, unless acute attack.	Often marked.	Late; generally increases with obstruction.
TUMOR.........	Only in cancer, and then in 30 per cent.	Only in cancer, and then in 40 per cent.; may be felt in rectum.	Common and distinctive; most easily felt in cæcum; little or no tenderness; sometimes movable, and can be changed in shape.
COILS OF INTESTINE....	Marked in proportion to emaciation.	Marked in proportion to emaciation.	Rarely seen.

N. B.—In any form of chronic obstruction, the symptoms of acute occlusion may suddenly supervene.]

Treatment.—As soon as the dangerous signs of intestinal obstruction are recognized by the physician, his first duty is to make as careful an examination (*vide supra*) as possible, so as to determine whether the obstruction may not be within the reach of direct treatment. If there is an incarcerated external hernia, it demands such operative treatment as is taught by surgery. If there is an impaction of gall-stones, or intestinal calculi, or foreign bodies, we may in some cases furnish appropriate assistance by the cautious use of laxatives. The treatment of fæcal impaction is of special importance. We have described the most frequent form of this in detail above. As has already been said, it is usually necessary to remove at least a part of the fæces with the fingers, or some instrument like a dressing-forceps or a spoon. In the second place, we may use large enemata of pure water, or preferably soap-suds, which must often be repeated four or five times a day, until they have a satisfactory result. These are best given by a funnel and an œsophageal tube ("intestinal tube") introduced as high as possible into the intestine. Cathartics administered internally serve as aids, especially castor-oil and rhubarb.

In stenosis of the rectum from cicatrices and new growths we can also sometimes employ local surgical treatment. The treatment of fæcal accumulations usually plays an important part here. Finally, the cases of ileo-cæcal invagination, in which the lower end of the invaginated ileum reaches the rectum, may receive local treatment. We may try a partial replacement by a "sponge-sound" (an elastic œsophageal tube to the end of which a sponge is fastened). Blowing in air by the bellows was also recommended for this purpose by the old physicians. As a rule, however, we use here large enemata of warm water, which sometimes seem to exert a favorable mechanical action. Very often it is impossible to apply direct treatment to the cause of the obstruction, or even to determine with any approach to correctness, the anatomical cause and the seat of the obstruction. In these difficult cases the physician must resort at first to merely symptomatic treatment. It goes without saying that the patient should have complete physical rest. All food should, if possible, be entirely forbidden. The torturing thirst may be relieved by bits of ice or sips of cold bitter tea. Formerly when there was obstruction of the bowels it was the custom to make trial of laxatives, first employing milder remedies, then more vigorous drugs, and finally, as a "last resort," metallic mercury in single doses of from 5 to 10 ounces (150 to 300 grammes), which is sometimes claimed to act mechanically in "desperate cases" by its weight. Except among some champions of mercury, the present opinion among physicians tends far more to the belief that cathartics are usually of no service, but are often directly injurious by increasing the resistance. Hence we have at present gone over to the treatment of severe internal incarcerations with large doses of opium (20 drops of laudanum or 2 to 3 grains [gramme 0.1–0.2] of opium, several times a day). Opium acts favorably on the patient's pain, the vomiting is diminished, and, by quieting the peristalsis, the danger of increasing the stenosis and tearing the intestine is also lessened. In fact, the opium treatment has many favorable results on record. Sometimes the first dejection appears during the administration of opium. Morphine subcutaneously injected has much less effect upon the intestine than opium; its employment is therefore limited to cases in which pain is so violent as to demand relief.

Since, therefore, there are many objections to the internal use of cathartics, we may try large enemata in those cases in which the seat of the stenosis is not known to be in the large intestine. They must be given with caution, but persistently, and they must often be repeated; then they sometimes give good results, even in severe cases. Unpleasant effects have been reported by other observers, but we ourselves have never seen any. The introduction of air into the rectum, instead of water, has been recommended and has been useful, particularly when the obstruction was situated low down and was the result of a kink or some similar trouble. The employment of regular lavage (Kussmaul, Cahn, and others) is very beneficial in many cases of fæcal vomiting; if this is a prominent symptom, the stomach should be washed out, in every case of intestinal obstruction. Large amounts of feculent liquid are frequently removed through the stomach-tube; and it is easy to see that freeing the stomach of its accumulations may favor a more vigorous peristalsis. Even when the nature of the intestinal obstruction precludes definitive recovery, lavage usually gives no inconsiderable relief.

We need not enter into details as to the general treatment. It goes without saying that the patient's strength must be kept up as much as possible, and that in severe states of collapse all possible stimulants must be used, such as strophanthus, camphor, and ether. Local applications to the abdomen are usually ill borne on account of the tenderness, but still we may try ice poultices or wet compresses. Opium is the best remedy for pain and vomiting, as already stated, but it must often be replaced by subcutaneous injections of morphine. In cases of extreme gaseous distention the intestinal coils may be punctured with the needle of a subcutaneous syringe and the gas in part evacuated. This sometimes proves very beneficial.

Finally, the surgical treatment of intestinal obstruction is most important. It is true that we should not, as a rule, proceed immediately to laparotomy at the beginning of the characteristic symptoms, but if we produce no effect by means of opium, injections, lavage, and similar measures, and if the general condition and the pulse grow decidedly worse, then in many cases laparotomy is the only means of possible salvation. Sometimes the existing obstruction may be thus detected and removed. (For particulars, see text-books on surgery.)

[The safety with which laparotomy is now performed has stimulated the study of all affections on which the operation has any bearing. Internal strangulations and invaginations may be relieved, and the portion of intestine containing a non-cancerous stricture can be excised. An early operation offers much better chances, of course. In these days persons should not be allowed to die directly from intestinal occlusion without an attempt being made to restore the permeability of the canal by surgical means.]

CHAPTER XII

INTESTINAL PARASITES

(*Helminthiasis*)

1. Tape-worms

Natural History of the Tape-worm.—Three of the tape-worms (*cestodes*) which are found in the intestines have a clinical significance: the *tænia solium*, the *tænia mediocanellata*, and the *bothriocephalus latus*.

1. The *tænia solium* is, when fully developed, two or three metres long. Its head (Figs. 47 and 48) is about the size of that of a pin, and has four projecting cup-like suckers, and in front a beak with about twenty-six hooks. The top of the head is, as a rule, plainly pigmented. A small neck, about an inch long, is attached to the head, and then follow the single "joints" (*proglottides*) of the tape-worm, of which the youngest, lying near the head, are still very small and short. They gradually increase in length and breadth, and at about a metre from the head they have an approximately quadrilateral shape. The segments which lie farther down, and which have already reached puberty, have the form of pumpkin seeds, and are nine or ten millimetres long and six or seven wide. The matrix or uterus runs through the middle of each mature segment (see Fig. 49), and from it, on each side, go seven or eight side branches, which ramify like a tree. On

one side, a little below the middle, lies the sexual orifice (Fig. 49, *a*). The male sexual organs consist of a number of little clear vesicles in the anterior portion of the segments. The thick-shelled eggs (Fig. 50, 3) develop in the uterus, and contain an embryo with six hooklets.

The tænia solium inhabits the small intestines of man. Its head clings to the mucous membrane so tightly, usually at some point in the upper third of the small intestine, that the neck is often torn off in trying to loosen the worm from the intestinal wall. The rest of the worm, which is in part in many coils, extends to the lower part of the ileum, but only exceptionally into the cæcum. From the lower end long chains, or single mature segments, are often detached, mix with the contents of the intestine, and are passed with the fæces, together with some of the eggs from the uterus.

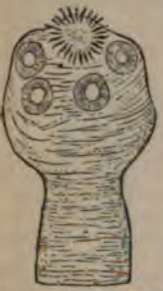


FIG. 47.—(From HELLER.) Head of tænia solium.

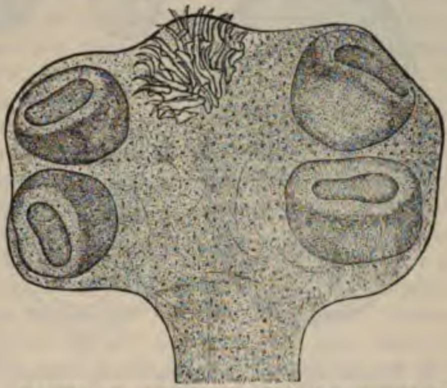


FIG. 48.—(From HELLER.) Head of cysticercus of the brain.

The further development of the eggs of the tænia solium takes place in another "host," almost always in the hog. Hogs are infected by eating fæces, offal, etc., containing tænia eggs. The thick shell of the eggs is dissolved in the hog's stomach, and the free embryos pierce the walls of the stomach and intestines and travel with the blood-current, or through the tissues, into the different organs, especially into the muscles. Here they develop, in two or three months, into cysts something larger than a pea, from whose walls a newly developed tænia-head arises, a so-called *scolex* (nurse). These cysts are termed worm-cysts, measles, or *cysticerci cellulosæ*. They live from three to six years; then they die and become calcified. If a cysticercus gets into a man's stomach from his eating raw or imperfectly cooked ham or pork, a new and complete tænia sprouts from the scolex, which forms mature segments in three or four months.

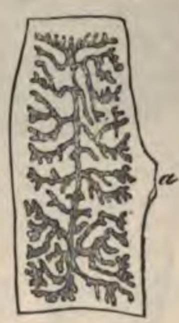


FIG. 49.—(From HELLER.) Tænia solium. Mature segment.

We usually find only one tape-worm in a man, but several specimens have been seen at the same time in the same intestine. The length of a tape-worm's life is not certainly known, but it has happened that some persons have lodged the same tape-worm for ten or fifteen years.

Although the fully developed tænia solium is seen only in man, as we have said, the cysticercus cellulosæ has been found, in rare cases, in dogs, rats, and monkeys, etc., as well as in hogs. It is a particularly important fact that the cysticercus cellulosæ itself may also occur, as such, in man. If tape-worms or mature segments get into a man's stomach in any way, probably by auto-infection by the finger, etc., the embryos travel into other organs. Cysticerci are often found in men, singly or in groups, especially in the skin, the brain, the eye, and the muscles. There is a special form of cysticercus of the brain, in which we find a whole chain of cysts, like a cluster of grapes, but sterile, the so-called *cysticercus racemosus.*

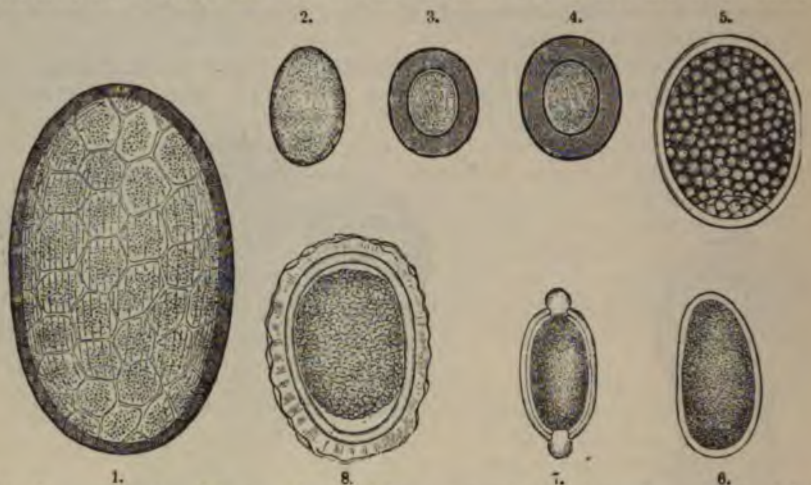


FIG. 50.—Comparative view of the eggs of some of the commoner intestinal parasites. 1. Egg of distoma hepaticum. 2. Distoma lanceolatum. 3. Tænia solium. 4. Tænia mediocanellata. 5. Bothriocephalus latus. 6. Oxyuris vermicularis. 7. Trichocephalus dispar. 8. Ascaris lumbricoides.

2. The *tænia mediocanellata,* or *tænia saginata* (from *saginare,* to fatten), is far more common than the tænia solium in many parts of Germany. It is longer than the tænia solium, being about three or four metres long, and its individual joints are, on the whole, broader and thicker. The head (Fig. 51) has also four prominent cup-like suckers, but it has no crown of hooklets. The mature segments differ from the proglottides of tænia solium, in that the central uterus sends off many more (twenty to thirty) side branches, which divide dichotomously, and not like a tree. The sexual opening is also on the side (Fig. 52, *a*).

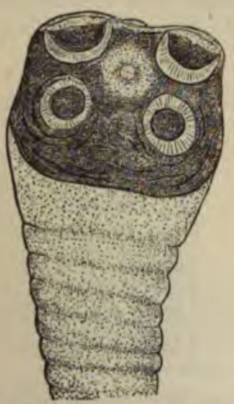


FIG. 51.—(From HELLER.) Head of tænia mediocanellata.

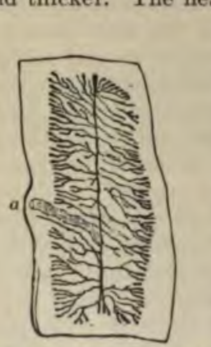
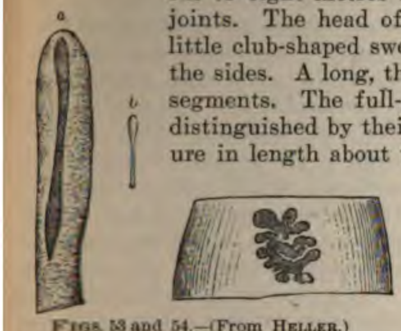


FIG. 52.—(From HELLER.) Tænia mediocanellata. Mature segment.

The life-history of the tænia mediocanellata is, on the whole, like that of the tænia solium. The tænia mediocanellata, however, throws off single mature segments much more frequently than the tænia solium. These segments are found in the fæces, and here they often exhibit a crawling motion. A kindred fact is that it is always easy to find large numbers of tape-worm eggs in the fæces of individuals who harbor the tænia mediocanellata. This is usually difficult in the case of tænia solium. The cysticercus of tænia mediocanellata does not inhabit pork, but beef, so that the infection of man by this tape-worm comes from eating raw beef. In man the cysticercus of tænia mediocanellata, which is somewhat smaller than the cysticercus cellulosæ, has never yet been observed.

3. The *bothriocephalus latus* occurs in Holland, Switzerland (Geneva), Pomerania, East Prussia, Hamburg, and Russia (the German Baltic provinces). It has not yet been observed in middle Germany. It is the largest tape-worm; it may be six or eight metres long, and sometimes has over four thousand joints. The head of the bothriocephalus (Fig. 53) consists of a little club-shaped swelling, with two slit-like depressed suckers on the sides. A long, thread-like neck joins the head to the youngest segments. The full-grown segments (Fig. 54) are short, but are distinguished by their great breadth. The largest segments measure in length about three or four millimetres, and in breadth ten or twelve, but the last joints are longer and are not so broad, so that they have an approximately quadrilateral form. The uterus consists of a very tortuous canal in the center. The sexual orifice does not lie on one side, as in the tænia, but in the middle of the abdominal surface, nearer the anterior border of the segment than the posterior. The eggs (*vide supra*, Fig. 50, 5) are of an oval form, and have a hood-shaped lid at one end. They are to be found in almost every dejection of persons affected with a bothriocephalus. Single joints of the tape-worm are not passed with the stools, but portions of the worm, several feet long, come away from time to time, especially in the spring and autumn.



FIGS. 53 and 54.—(From HELLER.)
FIG. 53.—Head of bothriocephalus latus. *a*, Lateral view, enlarged. *b*, Natural size.
FIG. 54.—Bothriocephalus latus. Mature segment.

The eggs develop only in fresh water. The embryo (Fig. 55), which is formed in them in a few months, and is provided with six hooklets and with vibrating cilia, is swallowed by fishes (chiefly pike and eel-pouts according to Braun, salmon according to Küchenmeister), and develops in their muscles and internal organs into cysticerci. The infection of man with bothriocephalus comes from eating such fish containing cysticerci.

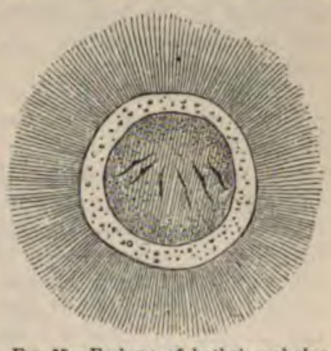


FIG. 55.—Embryo of bothriocephalus latus, with its ciliated coat. (LEUCKART.)

4. These three kinds of tape-worms that have been enumerated are the most important, but we will briefly mention the *tænia nana* and the *tænia cucumerina* (*tænia elliptica*). The first of these, the tænia nana [dwarf], is the smallest tape-worm, being only 1.0 to 1.5 centimetre long. The long head is provided with four suckers and a circlet of hooks which can be extended far out. The anterior joints are very narrow and short, those farther back broadening not inconsiderably. The tænia nana is very frequent in Italy and also in Sicily. In Germany it has been seen only exceptionally. Its cysticercus is said to inhabit snails. It is seen especially in children, and in them sometimes in very great numbers. Single joints are difficult to find in the dejecta, but the discovery of its eggs with the microscope is very easy. The expulsion of the worm by means of santonine and thymol (*vide infra*) is usually a simple matter.

The tænia cucumerina is twenty to twenty-five centimetres long. Its posterior joints are six to eight millimetres long, but have a width of only one millimetre. The head is provided with many hooklets; the beak can be extended and retracted. The tænia cucumerina occurs especially in children. Its cysticercus is found in the dog.

Symptoms and Diagnosis.—In many cases tape-worms are lodged in the intestines without causing any morbid symptoms. We can recognize their presence only by occasionally finding the joints in the dejections.

In other cases, however, tape-worms cause a list of disturbances which are often exaggerated by anxious, hypochondriacal, and nervous persons, but which ought not to be too little regarded. The symptoms are referred chiefly to the intestinal canal. Sometimes there is quite severe abdominal pain, which may assume a colicky character. The patient also frequently complains of irregularity of the bowels, and of occasional diarrhœa, which alternates with constipation. Many general symptoms are also added to those mentioned—loss of appetite, or at times marked voracity, general languor, disinclination to work, mental disturbance, depression, etc. Often the general nutrition is considerably impaired.

There are also certain symptoms to be mentioned which were formerly explained as "reflex." Still, it is very possible that poisonous matter may be produced by the tape-worms under certain circumstances, about which we do not yet have accurate knowledge, and that these poisons occasion some of the symptoms of tape-worm (*vide infra*). Among these we sometimes see marked salivation, tickling in the nose, dilatation of the pupils, palpitation, vomiting, headache (migraine), etc. In some cases even severe spasms and choreic conditions have been referred to the presence of tape-worms (particularly the tænia nana) in the intestinal canal, but it is hard to decide how far such a supposed connection can really be regarded as justified.

Thus in most cases tape-worms cause comparatively little disturbance, but they may occasion severe illness. Of late years it has been observed that when the bothriocephalus latus, or exceptionally a tænia, is present in the intestines there will often be an extremely severe anæmia, quite like pernicious anæmia (*q. v.*). The patient becomes excessively pale and feeble, the blood shows marked oligocythæmia and poikilocytosis; there are anæmic cardiac murmurs and similar symptoms. This condition also is probably referable to poisonous matter produced by the bothriocephalus latus, absorbed by the intestines, and fatal to the red blood-corpuscles. If the tape-worm is promptly expelled there is rapid and complete recovery.

Although many of the symptoms mentioned may arouse suspicion as to the presence of a tape-worm, the **diagnosis** can be made only by finding the joint or eggs of the tape-worm in the dejections. In many cases the patient himsel brings some of the segments found by him in the dejections to the physician, bu in judging of them a certain caution is always necessary, since shreds of mucu remains of food, etc., are quite frequently presented to the physician, under th idea that they are segments of tape-worm. But, on the other hand, it is also ver important that the physician himself should not forget the possibility of a tap worm and should himself direct attention in suspicious cases to the dejections o the patient. If there is a tænia mediocanellata or a bothriocephalus latus, it i usually easy to find the eggs in the fæces, but in the case of tænia solium this i more difficult. With tænia solium we usually have to search for segments of th tape-worm in the stools. Many patients with all sorts of indefinite, mild, ga tric and intestinal symptoms, or such general symptoms as headache or languo or even graver disturbances, such as apparent pernicious anæmia, are finall cured when the true cause of the disease is found to be a tape-worm.

If we obtain a few joints of the tape-worm we should endeavor to determine from them the species. If we spread out the pieces of tape-worm between two microscopic slides, the thicker, fatter segments of the tænia mediocanellata, with its many-branched uterus, may usually be distinguished without difficulty from the more tender and more translucent segments of the tænia solium, with a smaller number of lateral branches to its sexual apparatus. The statement of many patients that single segments of tape-worm come from them at other times than when at stool, and that they find them on their underclothing, almost always points to the presence of a tænia mediocanellata in the intestine. The eggs of tænia solium and tænia saginata can be distinguished from one another with extreme difficulty in the fæces, while it is easy to diagnosticate the bothriocephalus latus by its eggs, as they are so characteristic (*vide supra*, Fig. 50).

If we suspect a tape-worm, without having secured the certain evidence of segments or eggs in the dejections, it is a good plan to give the patient a mild cathartic, such as castor-oil, or a dose of boiled pumpkin-seeds, since after this, if the intestine harbors a tape-worm, single portions of it often come away.

Treatment.—The "tape-worm cures," which are recommended in so great a number that we can by no means mention all of them here, but only the most important and the most serviceable, aim at killing or benumbing the worm, and then at removing it from the intestine *in toto* by cathartics.

We usually begin with a so-called "preparatory treatment." This is to cleanse the intestine, especially the large intestine, from old fæcal masses, in order to prepare as free a passage as possible for the worm. For this purpose we give the patient a mild laxative, or, better still, a large enema of cold water. Many physicians recommend that there should first be given some active purge, such as calomel or castor-oil, but this is exhausting to the patient, and we believe it is not usually necessary. We also forbid for a day or two the use of vegetables, black bread, etc., and prescribe instead a limited diet of white bread, some meat, milk, and coffee. It is a wide-spread practice to take during the preparatory treatment certain articles of food to "make the worm ill." Among these a salad of finely chopped and very salt herring with onions and garlic is especially recommended. A similar action is also ascribed to strawberries, cranberries, and bilberries. Hence, on the day, and especially on the afternoon, before treatment, we have the patient take a large amount of the articles of food mentioned, such as herring salad.

On the next morning, after everything has been prepared, after the bowels have moved the night before, etc., the patient takes no breakfast, or only some strong sweet *café noir*. Then he takes the special anthelmintic, and in one or two hours, if he feels a great pressure in the abdomen, he also takes a few spoonfuls of castor-oil or rhubarb.

The number of tænicides recommended is, as we have said, very great. At present the following are most in use:

Many experienced physicians and we ourselves now employ almost exclusively the ethereal extract of male fern (oleoresina aspidii). It is true that the reliability of this drug as dispensed by different apothecaries varies, but in most cases, if we can obtain a good fresh preparation, the result is perfectly satisfactory. It should be said, however, that the drug is not entirely free from danger; in exceptional cases large doses have caused symptoms of poisoning, particularly amaurosis, also jaundice; and even death has occurred. We should, therefore, hold firmly to the rule of never exceeding a dose of two and a half to three drachms (grammes 10–12). We have never seen unfortunate results from this amount. The best way of administering the drug is in the gelatine capsules which are for sale, of which each contains thirty grains (two grammes) of the extract.

The bark of pomegranate-root (*cortex radicis Punicæ granati*) is one of the most efficient remedies. We formerly prescribed it in combination with the ethereal extract of male fern, in the following prescription:

℞ Granati radicis corticis .... ℥iv–v (grm. 120–150);
Aquæ .... O ij (grm. 1,000).
Macerate for twenty-four hours, and boil until it is reduced to ℥v (grm. 150).
Add: Oleoresinæ filicis .... gr. lxxv (grm. 5).

The whole amount is to be taken in three or four doses as near together as possible. In order to obviate the bad taste of the remedy and to increase the action by administering a larger amount at once, it has been recommended to introduce the whole amount of a still stronger decoction of pomegranate-root directly into the stomach by means of an œsophageal tube. As a rule, it is well to avoid this procedure.—The tannate of pelletierine, prepared from the pomegranate-root, is also to be recommended. It is almost tasteless, and in doses of eight to twenty-five grains (gramme 0.5–1.5) is said to be a very certain tænicide.

Another remedy, which has often proved successful, is kousso-flowers. We give three or four powders, each containing seventy-five grains (grammes 5) of powdered kousso-flowers, in white wine, giving a glass of wine containing one powder about every half-hour. Rosenthal's "kousso tablets" are more agreeable to take and are very good, but they are more expensive. Twenty of these, of fifteen grains each (gramme 1), may be taken without danger within an hour with *café noir* or lemonade. During the period of treatment the patient must lie as quiet as possible in order to avoid vomiting. Up to the present time we have not had sufficient experience of the koussine or kosseine, prepared from the alcoholic extract of kousso-leaves, which is said to be very efficient in doses of thirty to forty-five grains (grammes 2–3).

Of the other remedies we may mention kamala. We give the powder or tablets, in doses of 75–150 grains (grammes 5–10). This is a mild remedy and used chiefly for children. We may also give oil of turpentine, one or two ounces (grammes 40–60) in two doses in milk—an efficient but rather dangerous remedy in these doses; and finally chloroform. Lately, physicians have recommended pumpkin-seeds, which have long been known as a popular remedy. One advantage is that they do not have the bad taste of almost all the other remedies for tape-worm. About one hundred and twenty seeds, or for children half as many, are peeled, pounded up, and mixed with sugar, or else made into an emulsion, and administered.

The treatment is to be regarded as absolutely successful only when we find the head of the tape-worm, as well as its joints, in the patient's dejections. We may best search for the head in the fæces by diluting the dejection repeatedly with water, and pouring off the water. The tape-worm then remains at the bottom of the vessel. As it may be that the head has been dislodged even if it has not been found, we should not repeat the treatment for tape-worm until, some months later there are indubitable signs that the tape-worm still exists.

Every tape-worm treatment is rather drastic, and hence it is well, after the treatment is over, to recommend the patient to be prudent in his diet, and to be careful about his digestive tract for some time. In persons who are very weak or who have some other disease, we do not willingly undertake to remove a tape-worm without urgent reasons; but in people who are otherwise healthy it always well to get rid of a tape-worm, even if it causes no severe symptoms. The chief reason for this is that tænia solium might occasion cysticerci in the brain. The best time for undertaking a treatment is when joints or large pieces of the worm come away quite frequently of their own accord. We should never prescribe a treatment on the mere statements or suspicions of the patient. We must always convince ourselves with complete certainty of the presence of a tape-worm in the intestine.

We must finally mention that the only efficient prophylaxis against acquiring a tape-worm lies in entirely avoiding the use of raw or half-cooked beef or pork. The more widely spread the taking of raw meat is, as in Abyssinia, the more common are tape-worms in man. Certain callings, like those of the cook or the butcher, are also especially exposed to infection.

2. Round-worms

(Ascaris lumbricoides)

Natural History.—Ascarides are pale-reddish, cylindrical worms, pointed at both ends, with the sexes in different individuals. The females are thirty or forty centimetres long, the males about twenty-five. At the cephalic end of the worm are found three lips furnished with fine teeth. The tail is straight in the females and curved in the males. In the female sexual organs (Fig. 56) sixty millions of eggs may develop, at a rough estimate. These eggs are often found in the fæces of persons who have round-worms in their intestines (see Fig. 50, 8). They have a great capacity of resisting external influences, and a worm-like embryo develops in them in about nine weeks. The further dissemination of round-worms takes place without any intermediate host in this manner: the eggs containing the embryo worms are, through some chance, swallowed and then grow in the intestines into sexually-mature worms. Experimental infection with the eggs of round-worms gives distinctly positive results (Lutz, Epstein).

The round-worms inhabit chiefly the small intestine. In severe vomiting they often reach the stomach and are vomited up. In individual cases they have been found in the bile-ducts, in the air-passages, and, after perforation of the intestine, in the abdominal cavity. The number of round-worms existing at the same time in the intestine may be very considerable. We find them most commonly in children and in adults from the lower classes. Round-worms have been repeatedly observed to crawl out of the anus, the mouth, or the nose of children during sleep.

The round-worm is also common in hogs and cattle as well as in man.

Symptoms.—In general, round-worms are innocent parasites, which may exist in large numbers in the intestines without any bad results. In other cases they cause symptoms similar to those ascribed to tæniæ—abdominal pain, languor, itching of the nose and skin, urticaria, burning in the eyes, salivation, etc.—symptoms which are all ambiguous, and whose definite connection with the presence of round-worms it is hard to make out. Occasionally they excite long-continued and persistent vomiting, or violent diarrhœa, these symptoms persisting until the worms are expelled, when they promptly cease. Leichtenstern found that it was not very exceptional for children to be noticeably anæmic when harboring ascarides, and to improve remarkably when the worms were expelled.

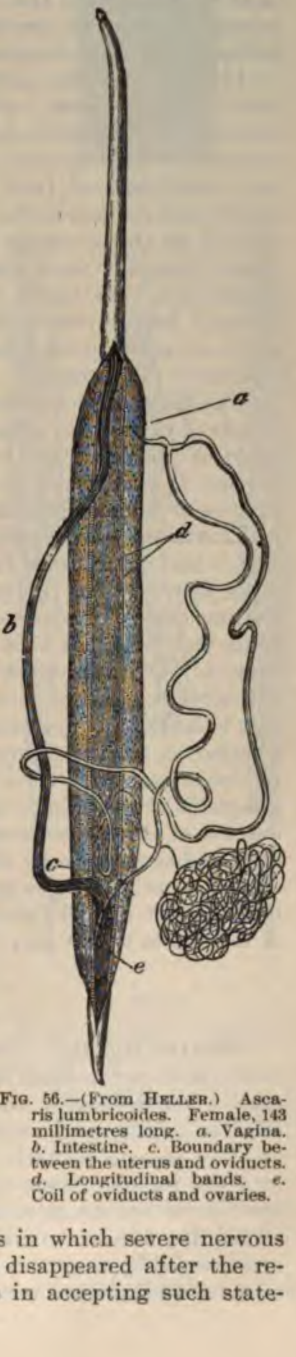


FIG. 56.—(From HELLER.) Ascaris lumbricoides. Female, 143 millimetres long. *a.* Vagina. *b.* Intestine. *c.* Boundary between the uterus and oviducts. *d.* Longitudinal bands. *e.* Coil of oviducts and ovaries.

The cases recorded in literature are quite numerous in which severe nervous symptoms have been caused by round-worms and have disappeared after the removal of the parasites. However cautious we may be in accepting such statements, nevertheless their credibility can not be wholly denied. We would mention especially convulsions, epileptiform seizures, choreic and cataleptic conditions, contractures, and temporary mental disturbances, which are claimed to be excited by ascarides. It is said that children with ascarides not infrequently exhibit mild nervous disturbances, such as headache, vertigo, mydriasis, and chills. All these symptoms, and also those above enumerated, are perhaps not so immediately dependent upon the presence of the worms in the intestinal canal, but are due rather to toxines developed under certain circumstances.

In some cases the presence of ascarides may excite much more severe symptoms by unfortunate accidents, as, for example, sudden suffocation from the entrance of a round-worm into the larynx. When a very large number of round-worms have been present in the intestine, severe symptoms of intestinal stenosis have been observed from their rolling together into a ball. If a round-worm crawls into the bile-ducts, it may give rise to jaundice, and even to the development of an abscess of the liver. In the abscesses of the anterior abdominal wall, usually termed "worm abscesses," the round-worms probably play a purely accidental part. We have to do in such cases with perityphlitic abscesses or with inflamed herniæ, which have perforated externally, by which the round-worms which are accidentally found in the intestines pass out, without having any causal relation to the abscess.

The **diagnosis** of round-worms is not usually difficult. Often a few worms appear of themselves, or as the result of a simple laxative. If not, search must be made in the dejections for the eggs of the ascarides. These are always easily found. It is advisable to obtain a particle of the contents of the rectum for the purpose of microscopic examination by introducing an elastic catheter.

Treatment.—The oldest remedy for ascarides is worm-seed — santonica. This is best given in the form of an electuary—santonica, a drachm (grammes 5); jalap, fifteen grains (gramme 1); and syrup, an ounce (grammes 30), to be taken in three doses—in combination with a cathartic. Of late, worm-seed, on account of its bad taste, has been almost wholly replaced by santonine, which is derived from it. This is prescribed in one- or two-grain (gramme 0.05–0.10) powders, or still more frequently in the form of santonine troches ("worm-tablets"), which may be had of any apothecary. It is well to give santonine also in connection with a cathartic, such as calomel. We give the patient one or two doses of santonine in the morning for three days, and on the fourth we give a cathartic. Severe symptoms of poisoning—spasms—have been seen only occasionally from the careless use of it. Milder symptoms, such as a yellowness of the urine and conjunctivæ, and xanthopsia, or seeing everything yellow, are somewhat more frequent.

In exceptional cases santonine fails to expel the ascarides. We may then try male fern or thymol (grains 8–24, gramme 0.5–1.50) in two or three doses given in the course of the day.

3. Oxyuris vermicularis

(*Seat-worms. Pin-worms*)

Natural History.—The oxyures are little round worms, the females ten or twelve millimetres long, the males only three or four (see Figs. 57 and 58). The eggs, when they reach the human stomach, develop very rapidly. The embryos, set free, collect in the small intestine and later mainly in the cæcum. As soon as they become sexually mature they pair, and then wander gradually down into the rectum, where they collect in great numbers. When the eggs inside the female become ripe, the female leaves the rectum and lays its eggs outside, where they are soon destroyed (Leichtenstern). The eggs may easily, however, be conveyed by fingers or by articles of diet, or by similar means, to the stomach of the same host or of some other person, whereupon the development of the worm begins afresh. As will be readily understood, children, with their untidy habits, are much more exposed to infection with the oxyuris than adults. The fact that the female oxyuris never lays its eggs in the intestine explains why the eggs of the oxyuris are rarely if ever found in the stools. The female crawls out of the anus to deposit its eggs. Male as well as female worms are discharged with the stools. The entire cycle of development of the oxyuris occupies about fourteen days. The number of these worms which may be present at the same time in the intestine is very considerable, so that "the entire mucous membrane of the colon is covered with them like a fur."

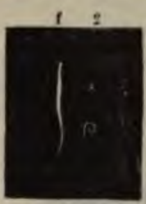


FIG. 57.

Symptoms and Treatment.—The oxyures found in the upper portions of the intestine and in the cæcum cause no symptoms whatever, but in the lower part of the rectum their presence causes local symptoms, especially a very severe feeling of itching and burning in the anus, which makes the child constantly scratch and dig with his fingers. This itching of the anus is most severe at night in bed. In girls the oxyures frequently travel into the vagina, by which an intense itching is also set up there, which sometimes leads to masturbation. In some cases in boys and men, oxyures have been found to be the cause of abnormal sexual irritation. Other clinical symptoms are rare, but sometimes there may be anæmia or nervous disturbances, and in rare cases marked intestinal catarrh, or inflammation of the vulva.

The diagnosis of oxyures is not difficult. Our attention is called to the itching of the anus, etc., and we look for worms. Single worms are easily found in the dejections, and often on the skin about the anus.

Treatment is directed first to the expulsion of the thread-worms from the rectum, and the prevention of any fresh self-infection. For this purpose the chief means are large injections, which must be employed persistently, once or twice a day, for weeks; for fluid we may choose simple warm water, or water containing vinegar, salt, or glycerine. Injections containing corrosive sublimate or carbolic acid are effective, but they should never be employed because of the danger of poisoning. It is difficult to remove completely the oxyuris from the upper part of the intestine, particularly from the cæcum. For this purpose the most efficient remedy seems to be santonine, perhaps combined with calomel or castor-oil. The itching at the anus is alleviated by cold compresses or by the inunction of small amounts of unguentum hydrargyri.

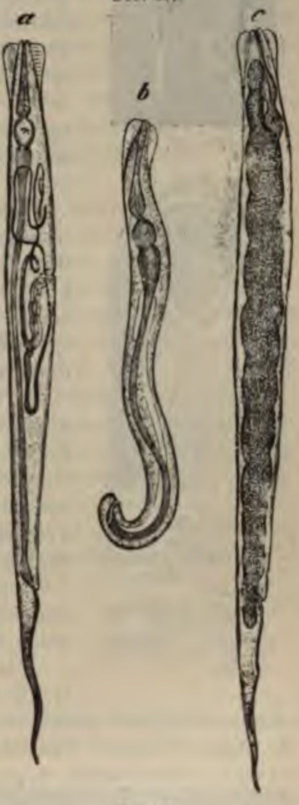


FIG. 58.

FIG. 57. — Oxyuris vermicularis. Natural size. 1. Female. 2. Two males.

FIG. 58.—(From HELLER.) Oxyuris vermicularis, enlarged. *a.* Mature female, not yet impregnated. *b.* Male. *c.* Female containing eggs.

Extreme cleanliness is very important. The anus should be frequently washed, and also the fingers, particularly the finger nails, to prevent the spread of the parasites.

[Enemas containing infusion of quassia, alum, eucalyptol, tannin, etc., are much in use. A plain enema should be given first, to unload the rectum and clean the membrane as far as possible, so that the anthelmintic may reach the worms when introduced.]

4. Anchylostoma duodenale

(Dochmius seu Strongylus duodenalis)

The *anchylostoma duodenale* is a worm first observed in upper Italy and in Egypt in large numbers, of which many inhabit the upper portion of the small intestine, especially the duodenum, but also the jejunum and ileum. The male is six to ten millimetres long, the female ten to eighteen. At the cephalic end (Figs. 59, 60) is found a bell-shaped mouth-capsule, which is provided with two small teeth on its dorsal edge, and four larger curved teeth on its ventral edge. With this sucking and biting apparatus the worm fixes itself firmly, like a wet cup, on the intestinal mucous membrane, and is nourished by the blood which it sucks out. The place in the intestine to which an anchylostoma has fastened may be recognized in the cadaver as a little ecchymosis. The worms sometimes bore completely into the inner part of the mucous coat.

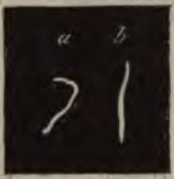


Fig. 59.

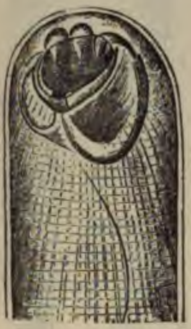


Fig. 60.

Fig. 59. —Anchylostoma duodenale. Natural size. *a.* Male. *b.* Female.

Fig. 60.—(From Heller.) Anchylostoma duodenale, enlarged. Head with bell-like mouth.

The eggs of the anchylostoma are discharged in great numbers with the fæces (*vide infra*). From these the larvæ develop and thrive, particularly in the dirty water of mud puddles and ditches; and by means of water that is spattered, or more often by dirty hands, they reach the mouth and the intestinal canal of man again, and then rapidly develop to sexual maturity.

If the intestine harbors many anchylostomata, the small but constant loss of blood caused by them is not without influence on the organism. In addition there are probably specific toxic influences which have an injurious effect upon the red blood-corpuscles. The symptoms of a severe anæmia gradually develop. Griesinger first made the discovery, in the year 1854, that the disease long known by the name of "Egyptian chlorosis" was caused by the anchylostoma duodenale. Since then confirmatory observations have been made in many parts of the tropics.

The disease caused by the anchylostoma has become particularly known in Europe because of the great frequency with which it occurred among the Italian laborers who were employed in building the Saint Gothard Tunnel. Since then many well-established cases have been found in other lands; e. g., Hungary; Germany, particularly in the brickmakers of the Rhine; also repeatedly in miners and laborers in tunnels.

The symptoms of the disease consist, as we have said, of a gradually increasing general anæmia, for which no special organic lesion can be made out objectively as a cause. The patient also suffers from very great general weakness and languor, dyspnœa, palpitation, headache, œdema, etc. The changes in the blood (oligocythæmia, poikilocytosis) are precisely similar to those seen in pernicious anæmia. The disease may last for months, or even years, and it often ends fatally, if it be not recognized and treated in time.

Leichtenstern has made numerous and accurate observations with regard to the brickmakers of Cologne. He states that no symptoms are observed for three or four weeks after infection with the embryos of the anchylostoma. Some five or six weeks after infection, when the parasites become sexually mature and breed, there appear bloody diarrhœa, intestinal colic, and accompanying progressive anæmia. At this time there is probably more shifting about of the parasites in the intestine, while they later become more fixed. This explains why the disease evinces a more acute and severe character at first, and then takes on the form of a chronic anæmia, with great diminution or cessation of the bloody stools.

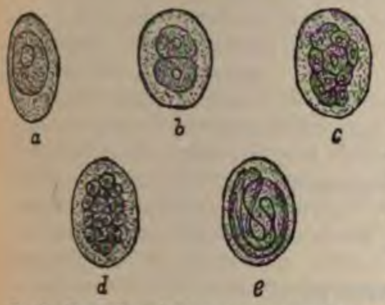


FIG. 61.—Egg of anchylostoma duodenale. *a* to *d*, various stages of segmentation; *e*, egg with embryo. (From PERRONCITO and SCHULTHESS.)

The diagnosis is easy if we only think of the possibility of anchylostoma. It is not very difficult to find an abundance of eggs in the fæces. They are of oval shape and characterized by the frequent presence in them of two or more spherules due to subdivision (see Fig. 61). The worms themselves are not usually found in the stools unless anthelmintics have been administered. Charcot's crystals are often found in the dejections previous to the appearance of the anchylostoma eggs (Leichtenstern).

If the disease is recognized, treatment is usually satisfactory. We prescribe the same anthelmintics as for the other intestinal parasites, particularly extract of male fern in suitable doses (2–3 drachms, 10–12 grammes), besides laxatives and injections. In this way it is often possible to banish the parasites entirely from the intestinal canal, and thus to induce, even in severe cases, a complete and often surprisingly rapid recovery. Besides extract of male fern, thymol has been found an effectual remedy. Of this, about two and a half to three and a half drachms (10–14 grammes) are given daily in doses of thirty grains (two grammes) each. For children the dose is proportionally less. Leichtenstern recommends that a few doses of calomel and one or two injections should be administered on the day preceding the specific treatment with male fern or thymol.

The prophylaxis from infection with anchylostoma is an important matter in industrial hygiene. The points chiefly to be considered are to enjoin the greatest cleanliness upon the workmen, to arrange properly for the reception and removal of the excreta, and to provide good drinking water.

5. Trichocephalus dispar

(*Whip-worm*)

The trichocephalus dispar is a worm four or five centimetres long, whose anterior part is very thin, but whose posterior part is decidedly thick (Fig. 62). It resides chiefly in the cæcum, but also in the colon, where it is often found in small numbers, and sometimes in abundance. The yellow or reddish-brown eggs of the parasite are characteristic, and they may be easily found in the fæces upon microscopic examination, but the parasites themselves scarcely ever come away spontaneously with the dejections. As a rule, the trichocephalus has no clinical significance, but lately Mossbrugger has reported a few cases of severe trouble in children, due to this parasite. The infection was supposed to be due to eating earth. The symptoms consisted in great anæmia and in long-continued and violent diarrhœa, with watery movements containing mucus and blood. The dejec-

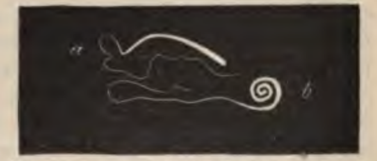


FIG. 62.—(FROM HELLER.) Trichocephalus dispar.

tions also showed many eggs of the trichocephalus, and Charcot's crystals. Treatment was very difficult and tedious, for the ordinary anthelmintics seemed to have slight effect. The most promising remedies in such cases would probably be extract of male fern and thymol.

SECTION VI

Diseases of the Peritoneum

CHAPTER I

ACUTE PERITONITIS

(Inflammation of the Bowels)

Ætiology.—There are two ways by which inflammatory agents most frequently reach the peritoneum: one is from the gastro-intestinal tract, and the other—in women—is from the genitals.

All the diverse forms of ulceration which attack the digestive canal may involve the serous layer. In such a case an inflammation arises which is at first limited, but which may under certain circumstances become more extensive. This inflammation may be regarded as analogous to that of the pleura in pneumonia; but the anatomy of the stomach and intestine is such that very often an ulcer in their walls ends in a complete perforation. If this occurs, the inflammatory germs contained in the primæ viæ at once escape into the peritoneal cavity and there excite an inflammation; which, from the specific character of its cause, is invariably purulent, and very frequently is at the same time septic or ichorous. The possibility of a peritonitis due to perforation, as a result of the various ulcerative processes of the stomach and intestines, has been frequently referred to in the previous sections of this work. Thus, it may occur in simple ulcer and in ulcerating cancer of the stomach; in typhoid, tubercular, or dysenteric ulceration of the intestine; in ulceration of the intestine above intestinal stenoses of many varieties; and in the small ulcers of the vermiform appendix due to the pressure of hard substances.

The female organs of generation are the other frequent source of peritonitis. In labor and premature delivery the genital tract is often directly infected. The infection may also occur, although much less frequently, at other times; for example, during menstruation. The various forms of inflammation which are thus set up, including endometritis, metritis, and parametritis, may in several different ways reach the peritoneum and excite peritonitis. A septic inflammation of the endometrium may involve the peritoneum by direct extension up the Fallopian tubes. In other cases it is through the lymph-vessels that a purulent metritis or parametritis spreads to the peritoneum. The larger parametritic abscesses may break into the peritoneal cavity. It is to be particularly noticed, however, that in many cases of septic puerperal peritonitis the uterus and its appendages are in a perfectly normal condition, having served merely as a gateway to the inflammatory agents without suffering any harm themselves.

Besides these two chief sources of peritonitis, numerous others are possible, although much less frequent.

Sometimes peritonitis is due to an extension of inflammation from other abdominal viscera. Hepatic abscess, suppurating hydatid cysts of the liver, ulcer of the biliary ducts, splenic abscess or infarction, purulent nephritis or pyelitis, abscess near the bladder or in the prostate, suppurating ovarian cysts, tubal pregnancy, psoas abscess, and Pott's disease—all these may produce peritonitis, either by direct extension or by perforation.

It is worthy of note that peritonitis may occur as a sequel of pleurisy. The pleural and peritoneal cavities are directly connected by the lymph-vessels of the diaphragm; and empyema as well as tubercular pleurisy (see next chapter) may spread to the peritoneum.

Penetrating wounds of the abdomen are a fruitful source of acute peritonitis. Surgical operations upon abdominal organs come under the same head. A large number of laparotomies proved fatal before antisepsis was introduced, because the inflammatory germs thus admitted excited a diffuse septic peritonitis. Even tapping the abdomen for ascites may cause acute peritonitis if the trocar is not aseptic. Abdominal injuries, in which the walls are not penetrated, very rarely, if ever, give rise to peritonitis. One way in which they have been said to produce it is by exciting internal hæmorrhage. In the new-born, peritonitis exceptionally results from infection through the navel.

Far less frequent are those cases of acute peritonitis which occur as a part of certain general diseases. In this class belong first the peritonitis associated with acute articular rheumatism (*q. v.*). This is usually benign. It must be regarded as analogous to the "rheumatic" inflammation which occurs in other serous membranes, including the endocardium, pericardium, and pleura. It is possible that rheumatic peritonitis of this sort may exceptionally occur as an apparently primary disease. It is a well-established fact that peritonitis may also develop in the course of acute or chronic nephritis. This we have ourselves observed. It is of course comparable with the inflammation which not infrequently attacks the pericardium and pleura in the course of nephritis. The exact cause of it has not yet been settled. Perhaps one factor is the retention in the blood of the solids which ought to be excreted with the urine. We must also mention that in very rare instances a specific gonorrhœal peritonitis occurs, either in connection with a constitutional infection from gonorrhœa, perhaps associated with gonorrhœal synovitis or endocarditis, or from the direct extension of gonorrhœal inflammation from other parts to the peritoneum.

From all that has been said, it is evident that, from a purely ætiological standpoint, peritonitis is by no means a stereotyped and uniform disease. The actual pathogenic agents, exclusive of the rare cases of mere intoxication, are always bacteria, but bacteria of diverse kinds. In the cases of peritonitis due to perforation, the bacterium coli seems to play an important part, and often also streptococci are influential. The cases of septic or puerperal peritonitis are mostly occasioned by streptococci; rarely the peritonitic exudation has been found to contain pneumococci, gonococci, proteus forms, and other germs. Not infrequently there seems to be a mixed infection from the start. Actinomycosis of the peritoneal cavity has been already briefly mentioned (see diseases of the pleura, page 303).

Pathology.—Like the analogous inflammations of the pleura and pericardium, peritonitis is divided into different varieties according to the character of the inflammatory exudation. The nature of the exciting cause of most cases of peritonitis is such that by far the most frequent variety is the fibrino-purulent. If the process involves the entire peritoneum—that is, if there is a "diffuse general peritonitis"—we generally find upon opening the abdomen that the parietal layer of the peritoneum and the outer surface of the intestinal coils are distinctly reddened, from marked vascular injection. There may even be small ecchymoses here and there. The serous membrane is clouded, a result partly of desquamation of its endothelium, and partly of the more or less abundant fibrinous exudation which covers the peritoneum with a sheet of coagulated fibrine. Very often the coils of intestine have formed numerous adhesions with one another (compare pleuritic adhesions). In cases of brief duration these can still be easily broken up, but after a prolonged illness they are extremely firm. There is usually also some free, fluid, fibrino-purulent exudation in the abdominal cavity. Its amount varies greatly. Sometimes there is only a small quantity of opaque fluid in the dependent portions of the cavity; sometimes there are many quarts, causing great distention of the abdomen. The exudation seldom inclines to a sero-purulent character. It is usually predominantly purulent. Very often the purulent exudation undergoes decomposition into the offensive sanious fluid of septic peritonitis. This is particularly apt to occur when the disease originates from an intestinal perforation or from puerperal poisoning. The perforation through the walls of the intestine is sometimes so large as to admit considerable amounts of intestinal gases and fæces into the peritoneal cavity. It is also possible that the putrefaction of peritoneal exudations may generate offensive gases. In rare instances the exudation is hæmorrhagic; but most cases of hæmorrhagic peritonitis do not belong here, but come rather under the tubercular or cancerous forms (*vide infra*).

In severe and protracted cases of peritonitis the intestine is involved to a certain extent. There is a collateral inflammatory œdema of its walls, causing sometimes a considerable increase in thickness, while at the same time they may be non-resistant and easily torn. The weakness of the muscular layer of the intestine may amount to complete paralysis, and thus permit excessive intestinal tympanites, either diffuse or local.

Milder forms of general peritonitis with sero-fibrinous, or chiefly serous, exudation are relatively infrequent. Under this head would come certain apparently primary and usually chronic cases with favorable issue, and also the peritonitis which sometimes occurs as a sequel of an ascites which has existed for some time (see next chapter). Probably also in those rare cases of peritonitis arising in the course of acute rheumatism and ending in recovery, the exudation has been sero-fibrinous.

We have spoken thus far of diffuse general peritonitis, but cases are not rarely seen of circumscribed or "encapsulated" peritonitis. Here, also, we have mild varieties with fibrinous exudations on the one hand, and on the other purulent inflammation. The milder inflammation is a result of the extension of the most varied forms of inflammation from neighboring organs. Thus, deep intestinal ulcers, for example, give rise to a mild circumscribed inflammation of the corresponding portion of the serous layer. A similar condition results from superficial splenic infarctions; from various hepatic diseases, when they reach the surface of the liver; and from numerous pathological conditions of the female genitals. In many of these cases the peritonitis takes a chronic course and leads to adhesions, and hence is called adhesive peritonitis.

Circumscribed purulent peritonitis has precisely the same ætiology as the general form, with this single difference, that firm adhesions are quickly formed around the spot whence the inflammation proceeds, limiting it and preventing it from involving the entire peritoneum. It occurs most frequently as a purulent perityphlitis (*q. v.*) consequent upon perforation of the vermiform appendix; and also as pelvic peritonitis, which is a possible sequel of most of the forms of puerperal inflammation to which the uterus and its appendages are liable. But we may also have encapsulated purulent peritonitis after the perforation of gastric and intestinal ulcers, or of suppurative processes in the biliary passages, and from similar causes. If the abscess is situated directly below the diaphragm it is termed subphrenic.

Histologically considered, acute peritonitis is perfectly analogous to the inflammatory processes which attack other serous membranes. The endothelium becomes degenerated, and, for the most part, is cast off. There is an exudation from the blood-vessels of a fibrinous fluid, which is partly coagulable, and with this exudation round cells escape in greater or less abundance. In the further progress of the disease there is an inflammatory new growth of vascular connective tissue, which probably originates chiefly from the endothelium and the permanent tissue-cells, but, according to some, starts in part also from the wandering cells. The new formation of blood-vessels certainly seems to be due chiefly to budding from the capillaries of the serosa. Thus arise the adhesions of connective tissue and the false membranes found in chronic cases between the different coils of intestine. They lead in process of time to marked thickening and retraction of the omentum and mesentery (*peritonitis deformans*). Most cases of purulent peritonitis prove fatal in the early acute stage. If a case recovers, the exudation undergoes fatty degeneration, and its cellular constituents are thus disintegrated and then are absorbed.

The results of circumscribed purulent peritonitis are detailed in connection with the clinical history.

Clinical History.—1. *Acute General Peritonitis.*—The following description applies chiefly to the severe purulent form, the one by far most frequently met with. It occurs in most instances after perforation, in puerperal cases, and after external injuries, such as surgical operations. In most of these cases the peritonitis is a secondary disease, so that it must obviously be greatly modified in its general characteristics and behavior by the original trouble. In the first place, the onset is modified. Many cases of peritonitis due to perforation begin abruptly, the patient having been previously in perfect health. Thus, as already mentioned, the first indication of a gastric or duodenal ulcer may be given by perforation. Most cases of perforation of the vermiform appendix present equally sudden and unexpected symptoms.

There are many other cases in which the symptoms of peritonitis supervene upon those of some grave disease already existing. For example, typhoid fever, intestinal tuberculosis or intestinal stenosis, may, by causing perforation, excite a peritonitis. Here the symptoms of this secondary disease may be more or less completely veiled by the other grave local and constitutional disturbances.

Again, an acute general peritonitis may, as we have already said, be the sequel to a local and circumscribed inflammation of the peritoneum. Thus, a purulent perityphlitis, or a purulent puerperal pelvic peritonitis, may finally become universal. In such unfortunate cases the change in symptoms is often gradual, and is not clearly pronounced.

We have now indicated certain variations from the general course of the disease; but, with these exceptions, almost every case of acute general peritonitis, whatever its ætiology, presents clinical symptoms which are so characteristic and typical that a general description of the disease will be both easy and advantageous.

The symptoms of acute peritonitis form two groups, the local and the constitutional. The latter are the result of the local disturbance acting upon the general condition of the patient.

Of the local symptoms, pain deserves to be named first. It is usually the earliest symptom; and, as the disease progresses, it is generally the excruciating abdominal pain which attracts most attention. The localization of the pain in the beginning of the illness may be of diagnostic value in doubtful cases, if such as to indicate the possible starting-point of the inflammation, for example, the vermiform appendix or a gastric ulcer. Later the pain extends over the whole abdomen. As a rule, there are brief remissions followed by fresh exacerbations. The pain is aggravated by voluntary movements, by deep inspirations, and probably by intestinal peristalsis. The abdominal tenderness is often extreme in peritonitis, and is very characteristic. The gentlest palpation is torture, and often the slightest pressure of the bed-clothes is almost unbearable. Frequently the greatest tenderness is in the umbilical region.

Acute peritonitis seldom exists without pain. The exceptions to this rule are seen chiefly in patients who are extremely prostrated, and whose sensibility and intelligence are much impaired. Here the peritonitis itself may escape notice—as in severe typhoid or in the last stage of tuberculosis.

Physical examination of the abdomen greatly aids the diagnosis in many ways.

As a rule, the abdomen is distended. This is an early symptom, and gradually becomes more and more pronounced. It is due mainly to the intestinal tympanites, which we have already mentioned, which sometimes becomes very great if the muscular fibers of the intestine are paralyzed. In the later stages the liquid effusion into the peritoneal cavity of course contributes to the prominence of the abdomen, but even then the distention is seldom so uniform or so broad as in ascites. In peritonitis, coils of distended intestine can often be recognized by their characteristic contour through the abdominal wall. Palpation also often shows a very characteristic diversity in the sense of resistance in different portions of the abdomen, occasioned by variations in the amount of exudation, by adhesions, or by the dilatation of some of the intestinal coils, and similar causes.

In general, if the abdominal wall is yielding and thin, the peritonitic distention will be greater, so that it is most marked in puerperal cases, where the preceding pregnancy has rendered the walls lax. In a person with powerful muscles and tense abdominal walls the convexity of the abdomen is seldom great. In some cases there is no convexity whatever. The walls may be as hard as a board, and the abdomen flat or slightly concave. In such cases the diagnosis may be difficult. Sometimes, again, the original retraction of the abdominal walls is succeeded by more or less distention of the abdomen.

Percussion over the distended intestinal coils yields a resonant and usually tympanitic sound. It is not till a considerable amount of liquid effusion has collected that there is dullness, most marked in the dependent portions of the abdomen. If there is much tympanites, however, quite a large effusion may exist without being detected on percussion. Percussion also gives results analogous to those of palpation, in that there is often a diversity in the quality of the resonance of different portions of the abdomen in peritonitis.

Usually there is too much pain to permit a careful examination of the change of dullness consequent upon change of decubitus. In general, the numerous adhesions between the separate coils of intestine also not infrequently interfere with the free motion of the peritonitic exudations.

Percussion not only gives information about the existence of a liquid, purulent effusion, but is also of value in determining the level of the diaphragm, as affected by abnormal abdominal distention. The upper limit of hepatic dullness is raised to the fifth or even the fourth rib. The heart is also pushed up. There is a tympanitic resonance above the margin of the ribs on the right side. The area of hepatic dullness is not only displaced upward, but is also evidently diminished. This is due in part to coils of distended intestine overlapping the anterior edge of the liver, and in part to the organ being tilted upward in such a way that its area of contact with the anterior wall of the body is less than normal. Various authors formerly laid great stress upon the total disappearance of hepatic dullness, regarding it as a sure sign that gas has escaped from the intestine into the abdominal cavity. The inference is not always correct. The liver may be displaced backward by coils of intestine, and hepatic dullness be thus abolished, although there is no air free in the peritoneal cavity.

If there is a considerable effusion, it is possible, as in ascites (*q. v.*), to get a sensation of fluctuation by gentle, quick palpation.

As a rule, auscultation of the abdomen does not throw much light on a case of peritonitis. In the distended coils of intestine we not infrequently hear all sorts of gurgling and splashing sounds. Sometimes we hear a peritonitic friction-sound, due to the movements of respiration, causing two rough surfaces to rub against each other. In particular, perihepatic friction is heard not very infrequently.

If the results of physical examination leave us still in doubt as to the presence of a peritonitic exudation, we may attain certainty by an exploratory puncture, and at the same time, if there be any exudation, determine its character.

Almost always the stomach and intestinal canal are disturbed by any severe case of peritonitis.

As to the stomach, vomiting is the most frequent and important symptom. Vomiting is often seen early in the disease, and recurs frequently as the illness progresses. It sometimes is spontaneous, and sometimes follows the ingestion of food. If spontaneous, the vomitus consists of watery mucus, usually of a greenish tinge. We do not know absolutely the cause of the vomiting in peritonitis. Apparently it is in part a reflex action, excited by the inflammation of the serous membrane. Possibly the external pressure of the exudation also affects the stomach; and it may be that absorbed toxines also excite vomiting. It must be added that vomiting may be absent in acute peritonitis. This is seen when the patient is comatose, and sometimes also when the peritonitis has developed upon perforation of a gastric ulcer, because the contents of the stomach are thus emptied out through the hole in its walls. The vomiting is usually accompanied by frequent eructations.

Of the intestinal symptoms, the reader has already become acquainted with the tympanites, and also with the fact that it is due mainly to a paresis of the muscular fibers of the intestine. This same muscular weakness furnishes an obvious reason for the persistent constipation usually observed in peritonitis; but we may have diarrhœa instead, from increased peristalsis and secondary intestinal catarrh.

The pushing up of the diaphragm has a noteworthy effect upon the thoracic organs. The lower lobes of the lungs are compressed, so that considerable dyspnœa results. The heart is likewise crowded upward, so that the apex-beat is usually to be felt in the fourth intercostal space.

Every case of acute peritonitis that is at all extensive has marked constitutional effects. These are in part the result of the wakefulness due to pain, and the restlessness and fever. But perhaps there are also definite reflex inhibitory influences, originating in the irritation of the peritoneal nerves and affecting chiefly the heart, just as Goltz in his well-known experiment killed a frog by blows upon the abdomen. The chief factor, however, is in all probability the toxines which are very readily absorbed from the peritonitic exudation by the peritoneum, and thus enter the circulation.

There is no other disease, except internal strangulated hernia—and the effect of that is perfectly analogous—which produces general collapse so quickly as does peritonitis. The countenance ("*facies abdominalis*") is rapidly altered, the cheeks fall in, and the eyes become hollow. The nose grows sharp and cool, the lips and tongue dry. The skin of the extremities is also cool and bluish, as a result of impaired circulation. The patient is extremely feeble. The chief cause of all these symptoms is the excessive weakness of the heart. The peritonitis has hardly begun before we find the pulse small and soft. In many severe cases the pulse finally becomes almost imperceptible. At the same time the pulse-rate increases, as is usual in collapse from any cause, so that 120 to 140 beats per minute is not an exceptional rapidity.

The temperature varies greatly in different cases. It may be high in the rectum, although the skin feels cool. Still, very high fever is not usual; and there are often considerable remissions. We even frequently observe the subnormal temperature of collapse. The number of respirations per minute is usually 30 to 40. This increased rate is due not only to the compression of the lower lobes of the lungs, but also to the pain caused by full inspirations and to the impeded circulation.

The intellect remains in most cases almost unimpaired to the end. There may exceptionally be mild delirium, or an approach to stupor, toward the close.

The course of acute general peritonitis in the great majority of cases is unfavorable. With the appearance of the grave symptoms just depicted the prognosis becomes almost hopeless. The course of the disease is also comparatively rapid. Marked variations in the intensity of the symptoms are infrequent. The grave local and constitutional symptoms persist, and, as a rule, the patient dies at the end of a few (two to six) days. Still, it is not well to make general dogmatic statements as to the clinical history, for the ætiology of each individual case impresses upon it individual characteristics. A peritonitis resulting from gastric or intestinal perforation is usually quickly fatal. The same is true of almost all cases of puerperal septic peritonitis. In a few cases, however, the inflammation is limited, by the encapsulation of the exudation. These may finally end in recovery through perforation of the abdominal walls or perforation into the intestinal canal. Now and then an acute general peritonitis may assume a chronic form. The effusion is mostly reabsorbed, and the newly-formed adhesions and false membranes contract into firm bands of connective tissue. The liver, spleen, and other abdominal viscera acquire a tough coating of connective tissue. The omentum and mesentery are shortened and thickened. Indeed, the omentum may roll itself almost completely up. Although the clinical symptoms become less severe, weakness usually persists, with gradual exhaustion and death. Often the intestine is so bent or pinched as to give rise to grave symptoms from stenosis.

Recovery from acute general peritonitis is very exceptional. If seen, it is usually in mild cases, such as sometimes occur after menstruation, abortion, or labor. Peritonitis as a complication of acute articular rheumatism is a very rare event. Its termination is generally favorable. In all cases of this kind the inflammation is probably not purulent, but sero-fibrinous.

2. *Acute Circumscribed Peritonitis.*—The local symptoms of this are essentially the same as we have just ascribed to the general form; except that, a smaller extent of tissue being involved, they are correspondingly limited. The pain and tenderness are confined mainly to one region, but its boundaries are never sharply defined. On palpation of this region, we find an increased resistance which is sometimes almost like that produced by a tumor. If there is an encapsulated effusion, we may detect fluctuation, particularly if the abscess is going to point outward. On percussion over the affected spot, there is either dullness or a muffled tympanitic resonance.

The constitutional symptoms are likewise those of general peritonitis, only usually less severe. Reflex vomiting does occur, but is seldom so persistent as in the diffuse inflammation. The physical weakness and symptoms of collapse are decided, but do not usually become extreme. There is generally an irregular fever, which may now and then assume an intermittent, pyæmic character. Most cases run a chronic course. If the illness be very much prolonged, death may finally ensue from general debility. Recovery is possible if the pus can be let out. This may be accomplished either by the surgeon or by Nature. Spontaneous discharge of the abscess may take place through the abdominal walls, into the intestine, or even, in rare instances, through the pleura into the lungs. But if the pus finds its way into the general peritoneal cavity, the peritonitis becomes diffuse and causes death.

To describe in detail each separate variety of circumscribed peritonitis would occupy too much space, and would also lead to useless repetitions. We have already spoken at some length of one especially important form—namely, perityphlitis. Perimetritis and pelvic peritonitis are chiefly puerperal affections, and are fully discussed by writers on gynæcology.

Abscesses which are very deeply situated—as, for instance, behind the stomach or in front of the spinal column, or perinephritic—may prove very difficult to diagnosticate, being so far out of reach. Sub-diaphragmatic abscesses containing air deserve a brief mention (*pyo-pneumothorax subphrenicus*). They are sometimes observed as a result of perforation of the stomach or transverse colon. Lying between the liver and the diaphragm, they crowd the latter upward and the liver downward, and are liable to be mistaken for pyo-pneumothorax. It is to be noted that in subphrenic abscess the lower limit of the lungs moves up and down with respiration (see above, page 438). Sub-diaphragmatic abscesses containing no air also occur, originating in the liver or spleen. Finally, there is a rare form of circumscribed purulent peritonitis to be noted, to which children seem especially exposed. It declares its presence by a painful fluctuating tumor above the left groin, which usually points into the rectum and ends in recovery.

Diagnosis.—The diagnosis of peritonitis is in many cases an easy matter, when we have the characteristic symptoms of tenderness and tympanites, vomiting, and collapse. Often the starting-point of the inflammation is equally obvious, in cases of secondary peritonitis supervening upon some disease which we have already clearly recognized, such as typhoid fever, gastric ulcer, or puerperal diseases. But where the peritonitis is apparently primary, we must inquire carefully into the previous history and the earliest symptoms of the attack, in order to form even a surmise as to ætiology.

The diagnosis is sometimes greatly obscured by the fact that under certain circumstances very similar symptoms may be excited by other disorders affecting the intestines. Thus, in typhoid fever there may be great tympanites and grave constitutional symptoms, with abdominal pain, so that peritonitis may be diagnosticated, while the autopsy, if there be one, discloses no signs of it. Deep ulcers of the intestine, however produced, may give rise to such great abdominal tenderness as likewise to simulate peritonitis. The differential diagnosis between peritonitis due to perforation and acute intestinal obstruction (*q. v.*) is often very difficult. In both, when there is severe constitutional infection, the symptoms are almost the same, and, furthermore, diffuse peritonitis may occasion such a paralysis of the intestine or such excessive tympanites as to prevent any motion of the bowels, and even to cause fæcal vomiting. On the other hand, intestinal obstruction is not infrequently complicated by general peritonitis, so that the two conditions may both exist simultaneously. The following are the chief points of distinction between them, although even these are not infallible:

PERITONITIS	INTESTINAL OBSTRUCTION
1. Begins with fever and with abdominal pain, often localized.	1. Begins without fever and without marked abdominal pain.
2. Abdomen very sensitive to pressure, hard, and tense.	2. Abdomen at first soft and not especially tender upon pressure.
3. Pain tends to abate as the disease goes on.	3. Abdominal pain gradually and constantly increasing.
4. No visible peristalsis of the intestines.	4. Visible peristalsis; intestinal coils can be felt like rolls.
5. Seldom fæcal vomiting, frequently hiccough and simple vomiting.	5. Distinct fæcal vomiting, hiccough rare.
6. Exudation demonstrable in the peritoneal cavity.	6. No free exudation in the peritoneal cavity.

brilliant, are encouraging, and further trials are the more imperative, because otherwise the prognosis of such cases is almost absolutely hopeless. The more promptly the inflammatory germs and septic matter are removed from the abdominal cavity, the greater is the possibility of permanent cure. The details of the surgical treatment of peritonitis must be sought elsewhere.

CHAPTER II

CHRONIC AND TUBERCULAR PERITONITIS

Ætiology.—Chronic non-tubercular peritonitis is a rather rare disease. It is found most frequently in post-mortem examinations of patients who have had for a long time ascites due to venous stasis—for example, in chronic cardiac or hepatic cases. The chronic peritonitis, however, is not the direct result of the passive hyperæmia in such cases, but is, as already hinted, due in most instances to the puncturing of the abdomen during life for the removal of the ascitic fluid. Exceptionally, a chronic peritonitis occurs as a sequel to some severe intestinal disorder, such as ulceration. Thus, chronic peritonitis is sometimes observed to follow typhoid fever.

Chronic peritonitis may furthermore be the result of an acute peritonitis. The latter seldom terminates in this way, but still it may, when rather mild and not quickly fatal. The encapsulated exudations of peritonitis usually persist a long while, as was implied in the preceding chapter.

There is still great uncertainty about the ætiology of those cases which start in a subacute or chronic manner, without special cause. Sometimes they seem to be due to an injury of the abdomen; in other cases there are special constitutional influences, such as alcoholism, which may occasion the disease. In children, and less often in adults, we sometimes see cases of "simple exudative peritonitis," with a serous exudation, which we are inclined to regard as analogous to the ordinary cases of pleurisy with effusion. It is possible that in such instances pathogenic organisms of various kinds occasion the inflammation; but in order of frequency there is scarcely a doubt that tuberculosis should be named first. Just as in pleurisy, many cases of what is apparently primary "simple" peritonitis turn out eventually to be tuberculous.

The tubercular is the most frequent form of chronic peritonitis. It is often merely a part of the tuberculosis of serous membranes in general (*vide* pages 289 and 365), of which mention has been already repeatedly made. In these cases it is usually due to a conveyance of the process from the pleura through the diaphragm. Another way in which tubercular peritonitis may arise is by infection from neighboring tubercular organs. Tubercular intestinal ulcers are among the chief causes of this kind, the ulcer extending to the peritoneum; or the peritonitis may be excited by tubercular retroperitoneal or mesenteric lymph-glands. In women tubercular peritonitis may be developed in consequence of tuberculosis of the genital organs. Tuberculosis of the uterus sometimes affects the Fallopian tubes by direct extension, and thence the virus enters the abdominal cavity and excites its specific inflammation. In conclusion, we have to mention that, in general miliary tuberculosis, the peritoneum also may be the seat of numerous tubercles, although these do not as a rule give rise to important symptoms.

Pathology.—In severe cases of chronic peritonitis—and scarcely any others have come to autopsy—the peritoneum is usually found to be considerably thickened. The intestinal coils are joined to one another and to the neighboring organs by numerous and extensive adhesions. The false membranes are often delicate and easily separable, but in some cases it is a hard matter to disentangle the confused mass into which the intestines have been rolled. Sometimes the liver and spleen are covered by firm, tough capsules. The omentum and mesentery are much shrunken; hence the name *peritonitis deformans*. The omentum may indeed be transformed into a single thick cord. As a rule, there is little liquid effusion, and perhaps none. In simple chronic peritonitis, such fluid as may be present is usually a cloudy serum, sero-pus being seldom seen.

There are sometimes many clots of fibrine suspended in the fluid. If the abdomen has been repeatedly punctured during the life of the patient, we often may recognize the separate punctures, on the inner surface of the peritoneum, from the hæmorrhages, adhesions, or similar changes which have occurred. In rare instances a peculiar form of chronic peritonitis has been observed as a sequel to punctures for ascites, called by Friedreich "chronic hæmorrhagic peritonitis with hæmatoma." In it almost the entire peritoneum is covered by a newly formed membrane permeated with large ecchymoses.

We will also mention briefly a peculiar form of chronic peritonitis, which has not yet been sufficiently investigated. This leads to the formation of numerous small nodules in the peritoneum, which are usually at first regarded as tubercles, but upon microscopic examination are found to be nodules of connective tissue.

Tubercular disease of the peritoneum may be divided into two forms: tuberculosis of the peritoneum, which may be acute or chronic; and tubercular peritonitis, which is usually chronic. In tuberculosis the peritoneum is covered with numerous tubercular nodules, varying in size from a millet-seed up to a pea; but there is not much coincident inflammatory change. In genuine tubercular peritonitis, on the other hand, the inflammatory changes above described are well marked, while sometimes it requires a microscopic examination to demonstrate the tubercular nature of the inflammation, by the detection of tubercles and cheesy degeneration in the newly formed tissue. Tubercular peritonitis is usually rather chronic, so that the adhesions are numerous and strong. The amount of liquid effusion varies, being sometimes considerable and sometimes scanty. Just as in tubercular pleurisy, it is not rare for the exudation to be bloody. In long-continued peritoneal tuberculosis the omentum is found irregularly thickened, and the whole intestinal cavity filled with tumor-like masses (lymph-glands, etc.). Among the more important associated conditions we have tuberculosis of other organs, and also the comparatively frequent combination of tubercular peritonitis with hepatic cirrhosis (*q. v.*).

Clinical History.—Diagnosis.—If an acute peritonitis becomes chronic, the violent symptoms gradually abate, while another group of symptoms takes their place. In other cases the chronic disease develops gradually and insidiously.

The sensitiveness of the abdomen is never so extreme as in the acute inflammation. Sometimes, to be sure, the patient complains of dull pains and a sense of abdominal oppression, but quite often the pain is either constantly or at times insignificant. On physical examination, we usually find moderate distention of the abdomen. Frequently this is not perfectly uniform, certain coils of intestine being especially prominent. Occasionally there is no abdominal distention whatever, the belly is flat or concave, and the walls are tense and unyielding. The abdomen is more distended if there is a large amount of liquid exudation or if there is extensive tuberculous new growth.

In many instances palpation furnishes very characteristic signs; for sometimes the thickening of the omentum and the numerous fibrous inter-intestinal bands above described can be felt through the abdominal walls as peculiarly resistant masses or uneven prominences. Indeed, if the omentum is rolled up, it may closely simulate a new growth. The same is true of enlarged lymph-glands. Not infrequently, particularly in tubercular peritonitis, the liver is enlarged so that its lower edge can be felt. But in other cases of chronic peritonitis there are no changes discoverable by palpation; or they may be concealed by an effusion or by the tenseness of the abdominal walls. A large exudation can be demonstrated by the great distention, or by its causing fluctuation, or by the signs yielded on percussion. As a result of the adhesions of the intestines to one another, the fluid does not change its position very readily upon change of position of the patient. This circumstance is indeed one means of differentiating between peritonitic exudation and ordinary ascites. Not infrequently the resonance upon percussion is very greatly impaired, even when there is no large amount of fluid exudation. This is probably due to thickening of the peritoneum, fibrinous deposits, and similar changes. Peritonitic friction-sounds are sometimes audible, particularly in the hepatic region. It has been already stated that the distortions and flexions which the intestines may undergo in chronic peritonitis may result in obstruction. In the same way the duodenum or the ductus choledochus may be so occluded as to occasion persistent jaundice.

The objective signs of both the simple and tubercular forms of chronic peritonitis have been embraced in one description, because the abdominal signs of the two are identical. To differentiate between them, other factors must be considered. We regard the patient's constitution and general appearance, and inquire into his family history, or discover if there are other ætiological factors, such as previous tubercular disease. A careful thoracic examination is extremely important. If we find the signs of coincident pulmonary tuberculosis, or of pleurisy, then it is almost indubitable that the peritonitis is tubercular. The course of the fever is very important. Persistent hectic fever must always arouse strong suspicions of tuberculosis. The character of the exudation obtained by aspiration may be significant, for the admixture of blood, as we have already said, occurs mainly in tubercular peritonitis; but we may also have serous exudation in tubercular peritonitis, just as in the ordinary tubercular pleurisy. Tubercle bacilli are not usually present in the exudation of tubercular peritonitis.

To diagnosticate simple tuberculosis of the peritoneum, when not attended by marked inflammatory changes, is generally a difficult matter. Often it is absolutely impossible. Frequently there is no abdominal pain or tenderness whatever. The abdomen is usually but moderately distended, as a result of the effusion present. If we find such a condition in a patient who is known to have some other tubercular affection, we are justified in suspecting that there is a simple tuberculosis of the peritoneum.

It is not unusual to observe the combination of hepatic cirrhosis with peritonitis, which we have already briefly mentioned. We have then splenic tumor and the other signs of portal congestion, in addition to the symptoms of chronic peritonitis, such as pain and fever. The amount of fluid in the abdomen is usually considerable. As a rule, the patients are hard drinkers. The hepatic cirrhosis may be the primary disease, and if so, it joins with the constitutional results of alcoholic excess in promoting the liability to tuberculous infection.

Particular notice should be given to the chronic peritonitis of children, already mentioned. The occurrence of ascites in children between the ages of two and ten years has been observed repeatedly, both by other authors and by ourselves. The ascites, which may be considerable, can not be traced to any cause, and after a few months completely disappears. The child during this time is usually rather pale and languid, but not much emaciated, nor does he suffer great local discomfort. There may be no fever. Since the cases often recover, their pathological anatomy remains obscure. Probably they are a mild form of simple chronic peritonitis. Still, of course, there may be other causes for the ascites, such as hereditary syphilitic disease of the liver.

In children, tubercular peritonitis plays an important part in general tuberculosis of the abdominal organs, a condition known as *tabes mesenterica*. In these cases the tuberculosis probably originates, as we have already said, in the intestine, so that usually we find the intestine, peritoneum, liver, and abdominal lymph-glands all simultaneously involved. The clinical symptoms are often due mainly to the peritonitis. The abdomen is distended and painful, and there is an effusion. Often there is also obstinate diarrhœa, as a result of tubercular intestinal ulcers, with persistent fever of an intermittent character, emaciation, and anæmia. The tubercular process may eventually involve the lungs, pleura, meninges, and other organs, or it may never extend beyond the abdomen.

As to the course of chronic peritonitis we have little to say. The simple chronic peritonitis may terminate in recovery, although on account of other co-existing lesions this event is rare, except in the special form which children present. Many cases of tubercular peritonitis prove fatal in a few months or weeks. The fatal termination of peritoneal tuberculosis may be occasioned by a septic peritonitis, due to perforation. We have seen two cases of this sort in which the tubercular focus perforated the intestinal wall from its outer side. In many instances, however, chronic tubercular peritonitis has a favorable issue, or at least there is very great abatement of all symptoms. This is particularly apt to be the case in what is called primary tuberculosis of the serous membranes in general (*vide supra*). If, in this disease, there is no simultaneous tuberculosis of the lungs, intestines, or other organs, then the final reabsorption of the exudation is possible, just as in tubercular pleurisy. It must be confessed that often the recovery is not permanent, for the tubercles may appear later in some other part of the body.

Treatment.—The means by which we can exercise a favorable influence upon the course of chronic peritonitis are scanty. Attention to nourishment, absolute rest in bed, and hygienic surroundings are very important; but, beyond this, treatment is mainly symptomatic. The chief local applications are poultices or fomentations, perseveringly employed. There is seldom such persistent and severe pain as to demand opiates, but they may be required for the diarrhœa which is apt to occur. On the other hand, enemata and mild laxatives may be indicated.

Of special drugs, the preparations of iodine should be mentioned, including iodide of potassium and syrup of the iodide of iron, although the use of these is seldom attended with obvious improvement. The persistent daily inunction of fifteen to thirty grains (one or two grammes) of mercurial ointment or oleate of mercury into the abdomen seems often to be beneficial. If there is considerable exudation, diuretine, acetate of potash, and similar diuretic remedies are indicated. Whether curative results are to be expected from tuberculine, further experience must determine.

Of late, the surgical treatment of peritoneal tuberculosis has had very favorable results. It has been observed that in many cases of tuberculous peritonitis, laparotomy, with as complete an evacuation of the exudation as possible (better than can be accomplished by mere puncture of the abdomen), exercises a wonderfully beneficial influence upon the course of the disease, and even seems in many cases to occasion complete recovery. This fact is interesting, although not yet wholly explained. Further observations must be collected with regard to this procedure, but our own experience justifies us in strongly recommending it. Particulars with regard to its employment (leaving the abdominal wound open and packing with iodoform gauze) are taught in works on surgery.

[In the opinion of the editor, what surgery has done for us in this affection is rather to demonstrate the curability of many cases than to work their cure. Formerly, if the diagnosis of tubercular peritonitis was made and the patient recovered, the very fact of recovery was held to invalidate the diagnosis. But the surgeon's knife has laid bare the miliary tubercles to the eye, given an exit to the serous exudation, and recovery has ensued. But we see other cases get well after simple tapping, and we see still others which present just as good a clinical picture of the affection and recover without interference of any kind. Such recovery may be temporary, tuberculosis breaking out afresh in the peritoneum or elsewhere, or it may be permanent. If, in spite of rest and appropriate general treatment, considerable effusion persists, aspiration or siphonage should be practiced. Laparotomy should probably be reserved for those rather rare cases which seem to be rapidly getting worse, and those in which the fluid reaccumulates after one or more tappings.

In expressing the belief that tubercular peritonitis recovers, the editor does not lose sight of the fact that multiple fibrous nodules occur disseminated over the peritoneum, indistinguishable by the unaided eye from tubercles.]

CHAPTER III

ASCITES

(*Hydroperitoneum*)

THE name ascites is given to a collection of transuded serum in the abdominal cavity, due to venous stasis. The peritoneal veins belong to the portal system, so that among the diseases which lead to ascites those which impede the portal circulation are chief. As we shall see in the next section, ascites is, therefore, of frequent occurrence in cirrhosis of the liver, syphilitic disease of the liver, compression of the portal vein by tumors, thrombosis of the portal vein, and similar disorders. Ascites is also frequently present as one of the dropsical symptoms in general circulatory disturbances, such as cardiac disease or pulmonary emphysema, and in the course of various acute and chronic renal affections.

The clinical significance of ascites is due partly to the local discomfort occasioned by the presence of any considerable amount of fluid within the abdominal cavity. Small quantities of serum are often unnoticed by the patient; but, where many quarts (fifteen to twenty, or even more) of transudation exist, the abdominal walls become greatly distended, and the patient has a very troublesome feeling of pressure, weight, and tension. What is of still greater importance is the crowding upward of the diaphragm. Respiration is thereby not a little impeded. If the ascites is great, the lower lobes of the lungs are so compressed that a considerable degree of atelectasis is produced.

To demonstrate ascites by physical examination is possible only when a considerable accumulation exists. Then the belly is prominent, its walls are tense and shining, and, the base of the thorax being gradually distended by the pressure of the liquid, the lower part of the thorax seems much broader than the upper. Distended veins are usually visible through the skin of the abdomen, like blue lines, here and there. As soon as the abdominal tension has attained a certain degree, fluctuation can be perceived, by laying both hands upon the abdomen and imparting gentle but quick impulses to the fluid through the walls. Percussion gives a dull sound everywhere that the fluid is in contact with the abdominal walls. Gravity, of course, leads the liquid to occupy the dependent parts. In the dorsal decubitus, and when the transudation is of medium amount, the dullness is bounded in the central and upper parts of the abdomen from a region of tympanitic resonance by a line concave toward the head of the patient. The surface of the liquid being horizontal, of course the dullness reaches nearer to the thorax along the sides of the abdomen than in the central line. We would add, that where the layer of ascitic fluid is thin we can obtain dullness only by light, superficial percussion. If the pleximeter or finger is pressed deeply in, the fluid is crowded to one side, and we get a tympanitic sound from the underlying coils of intestine. A factor of great diagnostic value is the change of dullness on change of position of the patient. If he lies upon one side, the fluid seeks the dependent portions of the cavity, and gives rise to extensive dullness there, while the opposite side now yields a tympanitic resonance. Or, if he changes to the other side, it in turn becomes dull, and the side previously dull becomes tympanitic. Similar differences are found between the results of percussion in a horizontal and in a sitting posture. It is only when the accumulation is very abundant that there is dullness over the entire abdomen.

The signs mentioned enable us in most cases to make a diagnosis of ascites with ease and certainty. It is, indeed, not always easy to distinguish a transudation of serum from the exudation of chronic peritonitis, for, of course, either sort of fluid would yield the same physical signs. Only, the change in the area of dullness consequent upon a change of position is less pronounced in case of an exudation, because the peritonitic adhesions impede the movements of the fluid; and we have, besides, all the other symptoms to guide us: there may be pain, fever, or thickening of the peritoneum discoverable on palpation, or signs of tuberculosis; or, on the other hand, there may be some cardiac or hepatic disease, which would render ascites probable. If the fluid is drawn off, its character will sometimes aid us in diagnosis. Ascites yields pure serum, oontaining almost no morphological constituents. Its specific gravity is usually less than that of a peritonitic exudation, because it contains less albumen. We may say that the specific gravity of the fluid found in peritonitis is generally above 1018, and that of ascites about 1012, or even lower. Hæmorrhagic ascites sometimes occurs in anæmic patients who are suffering from marked portal obstruction, as we have ourselves seen, for example, in hepatic syphilis.

Chylous ascites due to an abundance of fat in the fluid may appear as a result of occlusion of the thoracic duct, but it should be pointed out that ascitic fluid has sometimes a chylous opacity, even when it contains no fat (emulsion of albuminous substances ?).

There may be equal difficulty in the exclusion of ovarian cysts, particularly since the cysts are sometimes so large as to fill the whole abdominal cavity. We must first map out accurately the dullness on percussion, and also see if it varies with changes of position. In cases of ovarian tumor, change of position does not make much difference. The resonance on percussion of the deepest and most dependent portions of the abdomen may be misleading, in this way, that even in ascites a narrow zone here may be tympanitic. This should be remembered. Thus, just above the symphysis, there is sometimes a tympanitic resonance in ascites which might readily be mistaken for a proof of the existence of an ovarian tumor. The explanation is that in the places indicated a coil of intestine with a short mesenteric attachment may remain in contact with the abdominal wall in spite of ascitic accumulations. Further aid in the differential diagnosis is to be obtained from the history of the case (place where the swelling began), from a consideration of possible causative diseases, and from a vaginal examination, including direct palpation of the ovaries. In ascites the uterus is freely movable, while in case of ovarian tumors it is often bound down by adhesions. Further particulars may be sought in books on gynæcology.

The **treatment** of ascites, of course, depends largely upon the disease of which it is a symptom. As to the symptomatic treatment of ascites itself, we will confine ourselves to a few words about tapping. This operation is indicated when the local disturbances caused by the ascites are great; that is, if there is an unbearable sensation of pressure and tension, and, above all, if the crowding up of the diaphragm causes much dyspnœa. The instrument to be used is a common trocar of medium size. If strength permits, the puncture can be most conveniently done with the patient sitting in a chair, or crosswise on the edge of the bed. We then choose for the best place for the puncture the linea alba, about halfway between the navel and the symphysis. Of course we should first see that the bladder has been emptied. If the patient is very ill, we are often obliged to make the puncture while he is in bed; we then insert the instrument in the lower and left side of the abdomen, about halfway between the navel and the anterior superior spine. As a rule, the removal of ascitic fluid is easy and free from danger. The exudation should never be allowed to escape too rapidly. We may permit large amounts of liquid (five or ten quarts, or more) to flow slowly away at one tapping. We close the puncture with a piece of sticking plaster, or with iodoform-collodion. Often the fluid trickles out through the opening, because the abdominal walls have lost their elasticity on account of the persistent distention. We may then employ a suture to close it. After tapping, the laxness of the walls is favorable to palpation of the abdominal organs.

Inasmuch as tapping does not remove the cause, there is in most cases a very rapid reaccumulation of fluid. Thus the system is deprived of much albumen, and nutrition is impaired, so that not infrequently the operation is followed by decided loss of strength. Therefore, we should not tap in ascites, as a rule, unless the indications for the operation are urgent.

[If, as is very frequently the case, the fluid continues to drain away through the puncture after the trocar is withdrawn, good rather than harm results, provided the danger of irritation of the skin and of bed-sores is kept in mind and guarded against, and an instrument of moderate size is used.

Flint advocates early and repeated tappings if the fluid causes discomfort and does not yield to diuretics or cathartics. The pressure is removed in a measure from the abdominal and thoracic organs, and nutrition is thus promoted. The fluid is likely to return, but it does not always do so, or it may do so only slowly. He reports cases in which, after repeated removal, the fluid ceased to return and the patient remained apparently well. The result must depend, of course, chiefly on the underlying cause, which is sometimes very obscure.

In cases of cirrhosis the same principles govern Flint's treatment.]

CHAPTER IV

CANCER OF THE PERITONEUM

Carcinoma is the only new growth of any practical importance to which the peritoneum is liable. Primary endothelial cancer, analogous to the growth which attacks the pleura, is very rare. Cancerous growths here are usually secondary to cancer of the stomach, intestine, pancreas, liver, or some other organ. Often the secondary nodules are numerous, and almost as small as peas, presenting what is called miliary carcinosis of the peritoneum. They cover the omentum, mesentery, and parietal peritoneum. Separate nodules of larger size are less frequent. These may be found in the omentum, in Douglas's pouch, around the navel, or in other situations. Colloid cancer attains the most diffuse and extensive development of any variety. The retroperitoneal lymph-glands may also present at the same time large cancerous growths. Often the development of cancer in the peritoneum is attended with pronounced inflammatory disturbances—that is, we have a cancerous peritonitis.

The **symptoms** of peritoneal cancer resemble in many points those of chronic tubercular peritonitis. Simple miliary carcinosis may be very insidious and give rise to no special symptoms, so that it often is unsuspected. In many cases a moderate amount of fluid collects in the abdomen, and this, if we are aware of the existence of a primary cancerous growth, may lead us to surmise a secondary peritoneal carcinosis. The symptoms are much more pronounced if there is cancerous peritonitis. In that case there is usually very severe pain, marked abdominal distention, and constipation. We may sometimes feel the larger nodules in the omentum or upon the inner surface of the anterior wall of the abdomen, or even those in the lowest part of the abdomen, by palpation through the vagina. We have repeatedly observed the development of palpable nodules in the navel in carcinosis of the peritoneum. If the exuded fluid be drawn off, it is sometimes merely serous, but it may be hæmorrhagic. When the new growth has been diffuse, and particularly in case of colloid cancer, the exudation has repeatedly been found to present a milky opacity. Sometimes this fluid also has been tinged with blood. The opacity is due to fat, from fatty-degenerated and disintegrated cancer cells. Occasionally the microscope reveals characteristic cancerous elements in the fluid. [Diagnosis based upon the microscope alone has repeatedly proved to be erroneous.—V.]

The **diagnosis** can not be made with any positiveness unless, as a sequel to a primary cancerous growth already demonstrated, we observe the evident tokens of peritoneal disturbance, such as free fluid, pain, points of resistance on palpation, and distention of the abdomen. Other factors are the patient's age, cancerous cachexia, and secondary glandular enlargements, particularly in the groins.

Treatment must be confined to efforts at mitigation of the suffering. Warm applications, morphine, and supporting measures are chiefly employed.

SECTION VII

Diseases of the Liver, Bile-ducts, and Portal Vein

CHAPTER I

CATARRHAL JAUNDICE

(Icterus catarrhalis. Gastro-duodenal Catarrh with Jaundice)

Ætiology and Pathological Anatomy.—Catarrhal jaundice is still the almost universal term applied to the most common form of jaundice. It develops in previously healthy individuals, usually in a rather acute manner, and sooner or later (as a rule, after some weeks) terminates in complete recovery. The names "catarrhal" or "gastro-duodenal" jaundice rest upon the supposition that the entire process is due to a catarrhal inflammation of the duodenal mucous membrane. This catarrh is said often to spread from the stomach to the duodenum and in the duodenum to invade the region of the duodenal papilla and extend to the common duct. The common duct, it was claimed, became obstructed by the swollen mucous membrane or by the tough mucus which was secreted, and thus blocked up the bile secreted in the liver. This mechanical obstruction was regarded as the cause of the jaundice.

Of late it has been objected to this old doctrine of catarrhal jaundice that the supposed catarrh of the duodenum and larger bile ducts has never yet been absolutely demonstrated. In a few cases of jaundice, in which at autopsy a plug of mucus has been found in the lower portion of the common duct or the duodenal papilla, it is said that the explanation of the mucous plug is simply that the mucus has collected because the bile has not been secreted, and has not flowed through the duct. The jaundice should not be regarded at all as due to the obstruction, but rather as a result of a disturbance of the function of the liver cells (*paracholia*), such that the bile is not secreted into the minute bile-ducts, but directly into the capillaries and smallest lymph-channels.

In our opinion, this latter theory seems to be somewhat artificial and certainly not absolutely established, although it seems to offer a simple explanation of many other forms of jaundice, particularly of that following certain poisons. There is only one respect in which we think that there is an urgent necessity for change in the old doctrine of catarrhal jaundice. We do not associate the essential process with ordinary gastric and intestinal catarrh consequent upon such causes as errors in diet, but we regard the disease as in most cases an independent one due to a special cause. This cause is in all probability a definite infection, the particulars with regard to which are as yet entirely unknown; but meanwhile we can scarcely imagine that its anatomical effect is anything other than the development of an inflammatory catarrh of the bile-ducts, with a probably varying involvement of the stomach and duodenum. It is, of course, very possible that there may also be toxic influences at work, but at present their thorough study seems impracticable.

The main reason for assuming that there is an infectious factor in the development of catarrhal jaundice is, that not at all infrequently the disease appears in epidemics and endemics. Rather extensive epidemics of jaundice have been repeatedly observed in barracks, prisons, and communities in a manner to be explained only by the supposition of some local source of infection. In some instances the epidemics of jaundice have followed revaccination, obliging one to think of the transfer of the pathogenic organism by means of inoculation. In the frequent sporadic cases it is not usually possible to demonstrate any special cause for the disease, except that there is often a connection with some marked error in diet, with catching cold, or some mental excitement, such as anger. In most instances there is nothing of this sort to be discovered, and this again suggests an infectious origin for the disease. Catarrhal jaundice is observed chiefly in youthful individuals; sex has no marked influence.

Symptoms and Clinical Course.—Very frequently, though not invariably, there are, for some days before the appearance of the jaundice, definite signs of gastro-intestinal disturbance, as well as certain constitutional symptoms. Obstinate vomiting, marked pain in the stomach, and similar symptoms are not infrequent. Very often the trouble begins with loss of appetite, a bad taste in the mouth, nausea, a sense of pressure in the epigastrium, eructations, and constipation, accompanied by languor. Soon after, the patient himself notices the yellow color of the skin, or his attention is called to it by those about him.

We still hold provisionally to the view that the cause of catarrhal jaundice is mechanical; and we desire, with a view to preventing repetition, to describe here with some minuteness those symptoms which occur in varying severity in all cases of obstructive jaundice.

The bile is secreted in the liver under an extremely low pressure, and consequently a comparatively slight obstruction in the bile-ducts suffices to prevent the discharge of bile into the intestine. In ordinary catarrhal jaundice, and also in other varieties of jaundice, the obstruction to the flow of bile is seldom complete, and if so, only for a time. Still a considerable amount of bile collects, and distends even the intrahepatic ducts. As soon as this stasis has reached a certain point, the stagnant bile is absorbed by the hepatic lymph-vessels. Thus the bile and all its constituents are poured into the blood by way of the thoracic duct and carried to all parts of the body. No more than a few days need elapse before the bile-pigments are absorbed into the tissues, and give rise to the evident yellow color of the skin and visible mucous membranes which we call jaundice. Usually the yellowness of the conjunctiva is the first thing to attract attention. Later the entire skin becomes yellow, and the same color is plainly visible in the mucous membrane of the mouth and throat, especially after we have produced temporary anæmia by pressure, as in the lips. Of course, the internal organs, which we can not see, are likewise stained. Any abnormal collection of liquid will also have a marked yellow color. The cornea, the peripheral nerves, and the cartilages alone escape unstained. In other parts we may not only find this diffuse impregnation with the biliary pigments, but even solid granules of the latter.

A jaundiced patient often presents other indications of the presence of biliary coloring matter than the color of his skin. There is often an itching of the skin, which may be very troublesome. It may be so bad at night as to disturb sleep. The scratching thus induced often causes numerous excoriations and fissures, which may even occasion quite large furuncles. Urticaria is also sometimes observed. A peculiar disease of the skin, which has been described in connection with jaundice, is called *xanthelasma*. It presents bright-yellow spots, usually somewhat elevated, which are found mainly on the eyelids, though also on other parts of the body.

The remaining symptoms of hepatogenous jaundice may be divided into two groups. The first group comprises the symptoms excited by the presence of the biliary constituents, and particularly of the biliary acids, in the blood, while the second group is due to the lack of bile in the intestinal canal.

We have seen that, when the biliary outlets are occluded or narrowed, the constituents of the bile are absorbed by the lymphatics. We have already learned in part what becomes of the bile-pigment thus conveyed into the blood-vessels. That it also displays poisonous properties has been maintained by some investigators, but has not yet been absolutely proved. The presence of the bile-acids in the blood at any rate is of considerable clinical importance. Physiology has shown that these acids possess certain injurious qualities, and, among others, the power to destroy red blood-corpuscles. But in reality few if any blood-corpuscles are destroyed by the bile-acids in the blood, because they are too much diluted, and, besides, seem in large part to be quickly decomposed after absorption. These acids do really, however, excite certain nervous centers in a way to give rise to decided clinical symptoms. The most frequent effect is that produced by the cholate of sodium upon the cardiac ganglia, or possibly also upon the center for the vagus, and it is evinced by a slowing of the pulse. This is an almost invariable phenomenon, provided there be no fever or other complication, and is seen not only in simple catarrhal jaundice but in all cases of hepatogenous icterus. The pulse-rate is from 64 to 50, or even less. Slight irregularity in the heart's action is not infrequent. There are certain other nervous disturbances often seen in jaundice, referable to the presence in the blood of biliary constituents, and in particular of biliary acids. Sometimes there is a striking languor and muscular weakness, or headache, or the patient is "out of sorts." Grave nervous symptoms, sometimes seen in jaundice and grouped under the name of *cholæmia*, are discussed in another chapter. It also deserves a brief mention here that many cases with marked jaundice have a noticeable tendency to bleeding—that is, a sort of "hæmorrhagic diathesis." Hæmorrhages into the skin and in the viscera are quite often seen, and also epistaxis and analogous occurrences.

We come now to a consideration of those symptoms which arise because the bile does not reach the intestine. In this regard the standpoint of modern physiology is essentially different from the old views. Formerly the bile was valued as an important digestive secretion; now it is regarded by many merely as an excretion, containing certain end-products of metabolism; but perhaps this view is extreme, for the very fact that the bile is discharged into the upper part of the intestine suggests that it has a certain significance in the processes to which the intestinal contents are subjected, and there is certainly one function of the bile which is indisputable—viz., that it promotes the absorption of fat. The bile both contributes to the emulsifying of fat, and also promotes the passage of the fat-drops through the intestinal walls into the lacteals. Now, in hepatogenous icterus this work remains undone, as is shown by the fatty stools. From time immemorial the white clay-colored stools of jaundice have been well known, and are employed as the best measure of the completeness of biliary retention. The light color of the stools is due partly to the lack of biliary pigment, for it is that chiefly which imparts to normal fæces their dark-brown color; but the characteristic white clay color is due exclusively to the presence of undigested fat in large amounts. We have ourselves performed the experiment of putting a patient with extreme hepatogenous icterus upon a diet containing as little fat as possible, and have found that the stools then became light brown, and not at all like clay. Upon microscopic examination of the fæces in jaundice, sheaf-like aggregations of crystals are almost invariably observed. These were formerly supposed to be tyrosine, but Oesterlein has shown them to be in reality lime or magnesia soap.

Besides this influence upon the absorption of fat, there have been two other properties ascribed to the bile, having apparent significance for the pathology of jaundice. In the first place, there is the antiseptic power of the bile; this was said to explain the fact that in patients with biliary obstruction the fæces usually have a particularly bad odor, and also that there are often excessive tympanites and flatulence. Secondly, the bile was reputed to exert an influence upon intestinal peristalsis—hence, the constipation frequently seen in jaundice. Of late, however, authorities have felt doubts about ascribing either of these properties to the bile. The foul odor of the fæces may be associated with the imperfect digestion of fat, and the constipation with the change in the diet and with other circumstances.

We must mention one other point: If the obstruction to the discharge of bile is actually situated at the papilla duodenalis—and this was formerly regarded as certain in catarrhal jaundice—then the discharge of pancreatic juice must also be affected, and we must also consider the results of the exclusion of this secretion from the intestinal contents; but we do not possess definite knowledge as to the actual effects of such an exclusion.

We must now inquire what becomes of the absorbed bile. As to the biliary acids, we have already said that they probably undergo decomposition. Of the other constituents, including the taurine and cholesterine and the pigmentary matter, we know the fate of the last-named only—that is, we have learned how Nature seeks to rid herself of this foreign substance. As soon as the amount of bile-pigment in the blood and tissues becomes considerable, excretory efforts are made, in which the kidneys take the chief share. Certain changes take place in the urine almost simultaneously with the first appearance of a jaundiced hue in the skin; and these changes are due to the urine containing excreted biliary coloring matter.

The urine of jaundice is generally recognizable from its color, which is dark brown, like beer. The foam caused by shaking it is not white, but decidedly yellow. A bit of white filter-paper dipped in the urine is stained yellow. If the urine is shaken with chloroform in a test-tube, the chloroform dissolves the pigment, and, on being allowed to collect at the bottom of the tube, displays a decided yellow color. This is known as the "chloroform test." Another reaction which usually gives a satisfactory result, but not always, is Gmelin's. If urine containing bile-pigment is slowly poured down the sides of a test-tube containing a few cubic centimetres of nitric acid, the zone between these two liquids exhibits a fine play of colors. The oxidizing effect of the acid upon the biliary pigment produces a number of colored rings, the highest and most characteristic of which is green; next comes blue, then violet and red. Gmelin's test often shows very prettily if one filters the urine and then adds a drop of nitric acid to what remains upon the moist filter-paper. The characteristic colored rings form around this drop. Instead of nitric acid, we may use tincture of iodine, diluted with nine parts of water. If a small amount of this solution is poured upon urine containing bile, in a test-tube, there is usually developed a ring of a beautiful green color between the two fluids.

The biliary acids also may be detected in the urine of jaundice; but the process is somewhat tedious, and the knowledge gained is of no great practical importance.

The urine very often contains morphological elements which are characteristic. Nothnagel was the first to describe minutely the icteric casts—that is, hyaline casts which usually have a yellow tinge and quite often are completely covered with dark-yellow granules or with epithelium. The urine may contain a little albumen also, but this is not constant. If we add acetic acid, there may be an opacity developed which is said to be due to nucleine. All these changes in the urine point to a slight degree of damage done the renal parenchyma by the biliary pigment excreted.

[The presence or absence of albumen depends largely on the amount of the biliary constituents and on the length of time they continue in action on the kidneys; their effect on these organs is more or less that of an irritant.]

The sweat-glands also take part in the excretion of bile-pigment. The latter can be demonstrated in the perspiration of jaundiced persons, as well as in their urine. Not infrequently the patient's linen is colored yellow by the sweat. On the other hand, no bile-pigment is found in the tears, saliva, gastric juice, or secretions other than those mentioned.

Having now considered the phenomena common to all cases of hepatogenous icterus, we revert to the subject of simple catarrhal jaundice. The prodromal gastric symptoms usually last a few days, more rarely a week or two, when the skin becomes evidently jaundiced and the other results of the icterus are also seen. The urine grows dark with biliary pigment, the stools become light-colored and more or less clay-colored. The nervous system is not usually seriously deranged, but still most patients feel very languid, and have anorexia and a tendency to constipation. The pulse often becomes somewhat slower than normal, and sometimes the temperature also is subnormal, 97° or 98° (36°–36.5° C.), but there may be slight fever.

In most cases the physical examination of the liver is of interest, the organ being, as already mentioned, enlarged from the retained bile. Accordingly, the lower boundary of hepatic dullness usually extends the breadth of two or three fingers below the edge of the ribs, and not infrequently the lower margin of the organ can be plainly felt. Often the gall-bladder is so distended, both by bile and possibly by the mucus which the bladder itself secretes, that it projects from under the edge of the liver. In such cases, as Gerhardt tells us, we may sometimes make out by percussion a convexity in the lower line of hepatic dullness which corresponds to the gall-bladder. If the abdominal walls are lax, we may even feel the distended viscus. As a rule, there is not much distress in the hepatic region, although now and then there is a certain sensation of pressure or tension.

The symptoms depicted seldom last longer than a few weeks. Usually a patient who takes proper care of himself begins to feel better in even less time. The urine grows lighter colored, the stools darker, and the pulse more rapid. The yellow color of the skin often remains visible for quite a while, although gradually diminishing, even after the patient feels perfectly well; but at last the jaundice disappears also and recovery is complete. Relapses are indeed possible, particularly after errors in diet; but they are rare.

The termination of catarrhal jaundice is, therefore, almost invariably favorable. The entire course of the disease occupies about three to six weeks, rarely a longer period. It is a very exceptional occurrence, but one which we must always think of as possible, for this apparently mild and secure condition to be suddenly merged into the grave, pernicious variety of jaundice. (See the chapter on acute yellow atrophy of the liver and pernicious jaundice.)

Diagnosis.—Catarrhal jaundice is usually easily diagnosticated. The diagnosis is made chiefly from the course of the disease—the development of jaundice, preceded by gastric symptoms, in a previously healthy person, and generally in a youthful individual. It is very important to exclude other conditions which might occasion jaundice. We must consider, therefore, whether the history of the case suggests the presence of gall-stones (pain, hepatic colic), and be vigilant in our physical examination to detect a possible cirrhosis or new growth. In the case of elderly patients, particularly, it is not rare for what was at first regarded as an attack of ordinary catarrhal jaundice eventually to disclose itself as a grave chronic disease, such as cancer of the gall-bladder. We should not make a diagnosis of catarrhal jaundice until we have carefully weighed all the rational and objective signs.

Treatment.—Most cases of catarrhal jaundice terminate favorably and require no active treatment. Rest and prudence are indicated, and the diet should be carefully regulated, that the gastro-duodenal catarrh may not be aggravated. Fat must not be eaten, for, as we have seen, it is not assimilated, and only excites abnormal processes of decomposition in the intestinal canal. Lean meat, bread, soups, if not too rich, vegetables, cooked fruit, and lemonade or tamarind-water are allowable. Milk is to be avoided as a rule, because of the fat it contains, but yet it is often well borne. Alcohol is to be allowed very sparingly if at all.

We should also employ internal remedies to mitigate the assumed gastric catarrh. The various stomachic tonics are frequently prescribed. Rhubarb is a favorite drug. A very good medicine is Carlsbad water, or the artificial Carlsbad salts, of which latter the dose is half a tablespoonful to a tablespoonful, in a tumbler of warm water, before breakfast, or twice a day. The alkalies are said to have a favorable influence upon the gastro-intestinal mucous membrane, and they are also laxative; that they furthermore promote the secretion of bile, as many assume, is doubtful; but there is no doubt that the flushing of the kidneys by an abundance of liquid is beneficial. If there is obstinate constipation we are obliged sometimes to resort to more powerful remedies, such as castor-oil, calomel, or rhubarb.

Lately much enthusiasm has been displayed about the treatment of catarrhal jaundice by large enemata of cold water. The injections are said to overcome the biliary retention by exciting peristalsis, and possibly by also promoting the secretion of bile. Once a day a quart or two of water, at 60° to 70° (12°–18° R.), is injected, and is retained as long as possible. The good effect is said to be observable in a few days, both in the general condition of the patient and in the diminished amount of bile-pigment in the urine, as well as the darker color of the stools. Other physicians recommend injections of a pint to a quart (½ to 1 litre) of lukewarm water several times a day. We have tried both methods repeatedly without being able to convince ourselves that the course of the disease is thus rendered much different from that under simple dietetic management; still, one may try irrigations, especially if the case is obstinate. Whether there are any internal remedies which promote the secretion of bile is very doubtful. The drug which is most recommended for this purpose, and is much employed in catarrhal jaundice, is salicylate of sodium, in the dose of eight to fifteen grains (gramme 0.5–1.0), several times a day. The biliary acids have also been tried, in the form of purified ox-gall, and also salol, podophyllin, and many others. We believe that all these remedies may be perfectly well dispensed with.

The effort has also been made to empty the gall-bladder by manipulation. Gerhardt states that sometimes the distended viscus can not only be felt through the abdominal walls (*vide supra*), but it can be so firmly compressed as to squeeze its contents into the duodenum. Sometimes the obstruction is said to yield suddenly, as if a plug were driven out. This method has not been extensively adopted. It seems applicable only in a limited number of cases, and is probably not free from danger. Several authorities have recommended external faradization as a means to stimulate the gall-bladder to contract and discharge its contents. We believe that few will adopt the suggestion.

Among symptoms, the troublesome itching deserves particular attention. The remedies which may be recommended for this are bathing with cold water, rubbing the skin with slices of lemon, or the application of a 2-per-cent. solution of carbolic acid, or of salicylic acid or thymol dissolved in alcohol (1 to 3 per cent.); also menthol (headache pencil) and chloroform mixed with olive-oil. Sometimes temporary relief is afforded by a dose of antipyrine or salicylate of soda internally.

CHAPTER II

ACUTE FEBRILE JAUNDICE

(*Infectious Jaundice. Weil's Disease*)

THERE is a peculiar acute infectious disease which was first described by Weil, and then more fully by Fiedler and others, which seems to be always associated with an acute infectious inflammation of the gall-ducts, and, having jaundice for a symptom, it may properly be described in this connection.

The disease is most frequent in the months of summer. It attacks by preference young and middle-aged men. Fiedler was struck by the frequency with which butchers suffered from it. The symptoms usually begin suddenly. Extreme chilliness, fever, headache, and malaise are almost always present at the onset. Jaundice usually appears on the second day, or soon after, and it may become severe. Its immediate cause is doubtless obstruction of bile, for the stools are colorless, and the urine contains an abundance of bile-pigment. The constitutional symptoms remain for several days quite severe. The patient complains of violent headache, wakefulness, and vertigo. Sometimes there is evident stupor or mild delirium. Upon physical examination we not infrequently notice herpes on the lips, besides the icterus. The tongue is coated. There is nothing unusual about the lungs or heart, except that the pulse is apt to be quite rapid. The abdomen is not particularly distended. The liver is often enlarged, but not always. An acute splenic tumor is very often, but not invariably, present. There is usually diarrhœa. Vomiting may occur. The urine almost invariably contains albumen, and, as a rule, we find blood, epithelium, and casts, indicating a considerable amount of nephritis. In a few cases there are anuria and uræmia. Finally, a very characteristic symptom is the violent pain in the muscles, particularly in the calves of the legs, of which most patients complain. Epistaxis and hæmorrhages into the skin have also been repeatedly observed.

With these symptoms the disease persists for from five to eight days, during which period the fever is often very considerable. Temperatures of 105°–107° (40°–41° C.) are not rare. Then the fever falls by crisis or rapid lysis, although seldom with perfect regularity. At the same time the other symptoms abate also, and convalescence ensues after an illness of ten to fourteen days in all. Many of the milder cases recover promptly, while others are prolonged by various sequelæ and fresh exacerbations. An unfavorable termination seems to be exceptional: it may result from uræmia.

It is highly probable that this perfectly specific disease, the most important symptoms of which are, as we have seen, jaundice, fever, swelling of the spleen, albuminuria, and pain in the muscles, is an acute infectious process, with a special involvement of the biliary ducts; but we do not yet possess any precise information as to its ætiology. Possibly we do not always have to deal with absolutely the same morbific process, but with several allied conditions. We have no information about the pathological anatomy, because the disease is too benign to furnish autopsies, and the few bacteriological examinations which have been thus far reported have led to no decisive conclusion.

The **treatment** must be purely symptomatic. In the beginning calomel is useful. Later we prescribe antipyretics, hydrochloric acid, salicylate of sodium, and stomachics. The headache is often relieved by an ice-bag; the muscular pain by the inunction of chloroform oil. From the first the diet must be such as not to aggravate the nephritis (milk, simple soups).

CHAPTER III

BILIARY CALCULI

(*Hepatic Colic. Cholelithiasis*)

Ætiology.—Gall-stones are of very frequent occurrence; Riedel asserts that some two millions of the inhabitants of Germany have gall-stones, although this estimate seems to be very high. Despite their frequency, we have little positive knowledge of their causation. We can merely state certain circumstances which in all probability favor the formation of these concretions.

Biliary retention certainly acts in this way, both directly and by leading to an increased consistency and increased concentration of the bile. As a result, certain constituents which were before held in solution are thrown down. And yet this cause, however potent (*vide supra*), can not be regarded as the only one. The chemical composition of gall-stones suggests that their formation must be preceded by certain abnormal chemical processes of decomposition and of transformation. We can not otherwise explain why the constituents of gall-stones should differ, as they do in many ways, from the matters which normal bile holds in solution. For example, the pigment in gall-stones is never found unchanged, but invariably exists in composition with lime. Now, normal bile contains only a trace of lime, so that long ago Frerichs expressed the opinion that the lime comes from the mucous membrane of the gall-bladder. It is an important fact that the cholesterine, and probably also a portion of the pigmentary matters, are held in solution in normal bile by the combination of sodium with the biliary acids which it contains. If this sodium salt were decomposed from any cause, the matters named would naturally be precipitated. The decomposition of the salts formed by the bile-acids is greatly promoted if the bile acquires an acid reaction; but of the circumstances in which this last-mentioned change occurs we do not yet have any accurate knowledge. Lately Naunyn, after extensive investigations, has propounded a theory that the formation of calculi is due in most cases to a primary disease of the mucous membrane of the gall-bladder and the bile-ducts. The disease causes the destruction of great numbers of the epithelial cells of the mucous membrane, with a resultant production of cholesterine, and of a compound of lime with bilirubine. Thus are formed friable masses of detritus, which are gradually changed into gall-stones, in the interior of which it is still possible at an early stage to demonstrate the original pultaceous mass. As the cholesterine and the compound of lime with bilirubine continue to form by crystallization, and fresh deposits of firm material take place, the stone gradually grows harder and larger. Very often also there is added at this stage a deposit of carbonate of lime. The cause of the original lithogenous catarrh is not known. Perhaps the mere mechanical obstruction of bile is causative, perhaps infection. In particular, the bacterium coli commune, which originates in the intestines, seems to be important—or at least, if not this, a very similar kind of bacillus. If the supposition were confirmed that infection of this sort is a factor, it would also be natural, as shown by what has been previously stated, to consider that such an infection would have an influence upon the bile itself.

We have rather more knowledge as to predisposing causes than about the chemical processes involved in the formation of gall-stones.

Age seems to be an important factor. The great majority of patients are over forty. Gall-stones are much less frequent between twenty and forty years of age; and in children they are very rare, although they have been observed in the new-born. One reason why elderly people are so liable to this trouble is said to be the senile weakness of the muscular fibers of the gall-bladder. Thus stagnation and retention of bile are promoted. It has also been suggested that in old age the bile may contain an excessive amount of cholesterine and lime. It must be confessed that this explanation is not very satisfactory.

Sex also has a decided influence. All authors agree that gall-stones are more frequent in females than in males, the proportion being about three to two. An explanation of this fact has been sought in the sedentary life of women, and particularly in the mechanical effect of women's clothes and tight lacing, impeding the outflow of bile. It is a fact which certainly deserves consideration that we are very apt to find gall-stones and a corset-liver in the same individual. Also, the influence of pregnancy can not be denied. Gall-stones are found in women who have borne children much oftener than in others.

Much has been said about certain peculiarities of constitution, in their relation to the formation of gall-stones; and it is asserted that the mode of life may favor their development—as when too much food is taken, particularly an excess of fat and meat, and when there is insufficient bodily exercise. In general, statements of this sort must be viewed with much suspicion; still, we can not deny that cholelithiasis is seen with striking frequency in obese women, and that the trouble seems to be somewhat more common among the favored classes than among the poor. There often seems to be a hereditary predisposition to gall-stones. It has been said that there is a relation between cholelithiasis and gout, arterio-sclerosis, and other diseases, but this is not at all certain. It is an interesting fact that sometimes the same individual has gall-stones and renal calculi.

In many cases there may be disease of the liver and of the bile-ducts themselves, leading to persistent obstruction, and thus causing gall-stones. It must be confessed that it is often very difficult to decide about this point, for gall-stones, as we shall see later, may themselves lead to secondary disease of the liver, such as biliary cirrhosis and carcinoma; so that when gall-stones and some other hepatic disease are both present it is often impossible to decide which of the two lesions was primary.

Occurrence, and Chemical and Physical Properties of Gall-stones.—The place where gall-stones are most frequently formed and found is the gall-bladder. We may find in it any number, from one or two up to a hundred and more. The size varies from that of a grain of sand to that of a hen's egg. The large stones may completely fill the gall-bladder; and sometimes the smaller stones are numerous enough to fill it also. The stones usually lie free in the bladder, although exceptionally they may be found adherent to its walls. Rarely the bladder presents a diverticulum, in which a stone has been formed. The lining membrane of the viscus often presents quite a severe catarrhal inflammation. This may have preceded the formation of the stone (*vide supra*), but, on the other hand, it may have developed as a result of the mechanical irritation of the mucous membrane occasioned by the concretions. Very often the wall of the gall-bladder and the neighboring tissues are affected by mild or even severe inflammatory processes, necrosis, perforation, and similar lesions (*vide infra*).

Stones which are found in the larger bile-ducts are not formed in them, but they have become wedged in them while on their way to the intestine. Sometimes, however, stones are formed in the liver itself ("hepatic calculi"). These are seldom primary, but are usually the result of biliary obstruction because of persistent stenosis of the common duct. Hepatic gall-stones of this sort may reach a diameter of a half to one centimetre. In such cases the small intrahepatic bile-ducts are usually a good deal widened; or occasionally they present niches in which the stones lie. As a rule, the hepatic parenchyma surrounding the stone is in a state of chronic or acute purulent inflammation (*vide infra*).

In form, gall-stones vary infinitely. The smallest are irregular masses, well described by the name of "gall-sand." The larger stones are more or less round, oval, or polyhedral. The polyhedra are usually due to the mutual rubbing and pressure of a number of stones upon one another. In color, the stones vary according to the amount of pigment they contain, from almost black or dark brown to a lighter greenish or bright yellow shade. A fresh gall-stone always sinks in water; but when dry, gall-stones contain air and generally float. On cross-section, the hard stones are found to be either homogeneous or composed of layers. As a rule, there is a nucleus, darkly pigmented, which is surrounded by a lighter-colored envelope, itself either made up of concentric layers or evidently crystalline. Often the outermost layers are still distinguishable as peculiar darker and harder strata. If we examine thin scales of gall-stones microscopically we shall find in them (as in renal calculi) an organic cement which holds the minute crystals together (Posner).

As to chemical composition, gall-stones are usually divided into several groups. By far the most frequent variety is made up of cholesterine and pigment (bilirubine) mixed together in greatly varying proportions. The pigment is in great part combined with lime. On the average, stones contain about seventy to eighty per cent. of cholesterine. Besides these two chief ingredients, most stones also contain lime and magnesium. In color, they are light or dark according to the smaller or greater proportion of coloring matter they contain. Stones of pure cholesterine are less common. These are usually found singly, are soft, and often are almost transparent. Most cholesterine stones have a nucleus of pigment and lime in combination. Pure pigmentary concretions are rare, and are generally small, like coarse sand. A still greater rarity is a stone made up entirely of lime. Such a stone is small and very hard.

Anatomical and Clinical Results of Gall-stones.—It is by no means rare for gall-stones to be present even in considerable number and size without causing the slightest discomfort. This is proved by the fact that often at autopsy gall-stones are found by chance, although there had never been a symptom suggesting their presence. In such cases it is evident that the concretions have formed very gradually in the gall-bladder, and never have led to mechanical or inflammatory disturbance.

In other cases, probably very numerous, the gall-stones occasion certain disturbances, but these are comparatively insignificant and not of such a character as to enable the physician to make an absolute diagnosis. Patients of this sort complain at times of slightly painful or disagreeable sensations in the stomach and liver, particularly after bodily exertion, and they may also have other symptoms, such as slight dyspepsia. The objective examination yields nothing positive. The physician suspects a gastric disorder, a floating kidney, or a neurosis. It is important that he should always in such cases, particularly in women, consider the possibility of gall-stones; for it is possible, if there is an actual cholelithiasis, that at any time there may be severe symptoms, the correct interpretation of which will be rendered much easier if these mild prodromata have been duly appreciated.

In a third class of cases the gall-stones cause severe and sometimes actually fatal illness. The symptoms may be very characteristic, so that they can be recognized easily and absolutely; but not infrequently the condition is difficult to interpret, and misleading.

The form of the disease which can be recognized most certainly and which has been longest known to clinicians is characterized by the occurrence of distinct and peculiar paroxysms. These are usually termed biliary colic. An attack of this sort may occur suddenly, while the patient is apparently in perfect health. More often, however, the first distinct attack is preceded for a long time by mild disturbances (*vide supra*), although these latter often receive little attention, and are apt to be misinterpreted.

The typical attack of biliary colic begins either with absolute suddenness or after some slight prodromata, such as chilliness, nausea, and malaise. It may occur at any time, day or night. Usually there is no exciting cause to be discovered. Sometimes the paroxysm is preceded by a slight injury or some other apparently harmful influence, such as errors in diet and the like. The chief symptom of the attack is the pain. This may be extreme from the start, or it may begin mildly and then rapidly or slowly become extreme. Usually the patient refers the pain chiefly to the epigastrium and to the right hypochondrium, but from here it radiates into the left side, the back, the shoulders, and even the right arm, and sometimes again down into the hypogastrium and the thighs. In some paroxysms the pain increases to extreme severity; and convulsive movements have been repeatedly observed, particularly in nervous persons, as a result of the pain. The pain may be accompanied by nausea, eructations, and repeated vomiting. The bowels are usually constipated, but sometimes there is diarrhœa. The constitutional disturbance is marked; the patient feels extremely feeble and exhausted, and gives the impression of anguish and collapse. The pulse is small, usually accelerated, but sometimes infrequent. The bodily temperature is normal in a minority of the cases; often there is slight fever, and sometimes a considerable rise of temperature, to 104° (40° C.) and more. At the beginning of the attack, and also later, there is apt to be chilliness, or a well-marked rigor; and this is often, but not invariably, associated with high fever.

Upon objective examination of the abdomen we find its upper portion, particularly the region of the liver and gall-bladder, slightly prominent or even markedly so. The same region is sensitive to pressure, and this tenderness may be diffuse or localized. Sometimes an enlargement of the liver can be distinctly made out (*vide infra*), and sometimes when the abdominal walls are yielding we can, upon careful palpation, feel the enlarged, tense, and tender gall-bladder (*vide infra*). In exceptional cases we can even feel the grating of the stones upon one another, and with a stethoscope hear the crepitation thus produced; but in other cases it is impossible to make out with certainty any change, either in the liver or the gall-bladder; or the abdominal walls may be so thick and rigid that a good examination is impossible. In any case of this sort the physician looks with special attention for the appearance of jaundice. Even a slight icteric hue of the eyes and the skin is important, for the appearance of jaundice is often decisive in leading to an absolute diagnosis. Sometimes the jaundice is very marked; in such cases the urine contains an abundance of bile pigment. It can not, however, be too much insisted upon that it is by no means exceptional to have no jaundice at all in an attack of biliary colic (*vide infra*). With regard to finding gall-stones in the fæces, *vide infra*.

The duration of an attack of biliary colic varies greatly. Extremely mild attacks may last merely a few hours or a day; more severe attacks continue two to four days and even much longer. It is equally impossible to establish any universal rule as to the frequency with which the attacks recur. Sometimes a second attack will come after a short interval; sometimes not until months or years have elapsed. Not infrequently there will be several attacks in close succession, and then none for a long time; or there may never be any more. Again, the early attacks of simple colic may be followed later by a complicated disorder, due to one of the numerous sequelæ to which gall-stones may lead.

We intend to discuss the remote results of gall-stones, but we must first answer the question, What abdominal processes occasion the attacks of biliary colic, of which we have just presented the clinical picture. Until within a few years the answer to this question seemed very simple. Almost all authorities conceived that the attack was due mainly to the moving about of the calculi and their expulsion. It was said that the stones moved into the cystic duct because of their own weight, the pressure of the diaphragm, and, above all, the contractions of the gall-bladder; and being once in the duct, they were forced along through the cystic and the hepatic ducts. The attack was successfully ended when at last the concretion escaped from the common duct into the duodenum. The incarceration of the stone in the common duct necessitated jaundice. If the stone stopped in the cystic duct, or, as was often supposed to be the case, went back into the gall-bladder again, there would be no jaundice.

This old doctrine of the causes of biliary colic is now known to be in large part incorrect, a better understanding having been obtained, incidentally to operations upon the gall-bladder and the bile-ducts. In particular, Riedel has made extensive and careful observations, and has contributed many new facts and views to the doctrine of cholelithiasis. Still, complete insight into all the conditions has been by no means attained.

Even now it must be confessed that small stones can be driven from the gall-bladder through the cystic and common ducts into the intestine in the way above mentioned, but the progress of the small stone in this manner into the intestine usually causes no symptoms at all, or, if there are any, they are slight and ambiguous. It may be that now and then a somewhat larger stone is forced from the gall-bladder into the intestine and excites a genuine attack of biliary colic, in the old sense of the term; but in most cases the attack which we clinically term biliary colic is probably due, only to a very slight extent, to the incarceration of a calculus, or to cramp-like contractions of the gall-bladder and biliary ducts, but it is rather an inflammatory process. So long as the stones in the gall-bladder remain there without exciting any secondary change, and so long as the bile flows freely around them, in and out, there are no clinical symptoms, although there is very often a gradual development of what is called dropsy of the gall-bladder. If a stone enters the neck of the bladder and there increases in size, or if the cystic duct becomes obstructed by the swelling of its membrane, then the entrance of the bile into the gall-bladder is impeded. The constituents of the bile which is in it are absorbed, and the contents of the gall-bladder change to a yellowish serum, which may finally be almost colorless. This process often goes on without any symptoms, but frequently there are slight suggestions of chronic inflammation. The wall of the gall-bladder becomes thickened; there may be a gradual formation of manifold adhesions between the serous coat of the gall-bladder and the neighboring parts, such as the omentum, duodenum, or transverse colon; and these adhesions are in many cases the cause of the mild and indefinite pains and other unpleasant sensations of which many patients with gall-stones complain for a long time previous to their first genuine attack. When a gall-bladder has been changed in this way, there is liability to frequent and sudden acute inflammatory symptoms; acute calculous cholecystitis is the true anatomical basis of biliary colic.

We do not possess accurate knowledge as to the cause of these acute inflammatory attacks or exacerbations. In our judgment, it is more probable that from the start infectious germs are factors in promoting the inflammation—perhaps the bacterium coli commune, perhaps other bacteria. These usually originate in the intestine, and opportunity for their pathogenic activity is afforded by the mechanical irritation and pressure of the gall-stones. The ordinary brief attacks of biliary colic are due to simple cholecystitis with a serous exudation, which usually gets well of itself in a few days. This serous or "catarrhal" cholecystitis occasions swelling of the gall-bladder, pain, fever, and vomiting. The calculus caught in the neck of the gall-bladder may, of course, have many other stones behind it; but it is not pushed onward if it has reached a certain size; it remains till the inflammation passes away. We have in this case a so-called "unsuccessful attack of biliary colic" (Riedel). These attacks are, of course, as a rule not associated with jaundice. If, however, the stone is small and the cystic duct patent, the stone is pushed on by the pressure of the inflammatory exudation which collects behind it. We have now the pain due to the inflammation and also that due to the incarceration of the stone, and as soon as the stone reaches the common duct, and for a time blocks the discharge of bile, we almost invariably have obstructive jaundice. If the stone is successfully driven through the common duct into the duodenum, the pain and inflammation usually promptly cease. By careful search we may find one or more stones in the fæces. In brief, we have to deal with a so-called "successful" attack of biliary colic. The distinction between unsuccessful and successful attacks is, of course, not always self-evident; but it should be noted that, according to Riedel, unsuccessfu attacks are much more frequent than the successful. It is true that in the successful attacks there is always jaundice, and in the unsuccessful attacks there is usually no jaundice, yet this criterion is not absolute, because, as Riedel was the first to point out, jaundice may occasionally be present in the unsuccessful attacks. In such a case the jaundice, of course, is not due to closure of the common duc by a stone—that is, it is not lithogenous, but it is inflammatory. The catarrha inflammation of the gall-bladder may sometimes extend to the bile-ducts and produce jaundice, because of inflammatory swelling of the common or hepatic duct.

This simple cholecystitis or cholangitis (inflammation of the bile-ducts) doe not by any means comprise all of the pathology of cholelithiasis. In many case there is not a simple cholecystitis or cholangitis, but a purulent inflammatio which is either purulent from the start or a sequel to a serous exudation. Th bacteria are factors in this process there can be no doubt, and it is the author opinion that we can scarcely draw any sharp dividing line between serous an purulent inflammation of the bile-ducts. The danger of inflammation varie according to the power of the infecting bacteria, and the greater or less vulne ability of the infected tissue. It is certain, however, that the purulent inflammation will cause severer clinical symptoms. The condition now presented is no longer a simple attack of colic, but a far more severe and persistent constitutional disturbance, associated with pain in the hepatic region, vomiting, possibly jaundice, and very likely signs of enlargement of the gall-bladder and the liver. There are high fever (often accompanied with rigors), general prostration, and cerebral symptoms. In milder cases there is only a purulent cholecystitis. In the severer cases there is a purulent diffuse cholangitis alone, or combined with purulent inflammation of the gall-bladder. To enumerate here all the possibilities of the condition is impossible. Even severe derangement of this sort is not incompatible with spontaneous recovery, but in some cases, if it is too late for surgical aid, there is a general septic infection and death.

We shall refer to a great number of other sequelæ of gall-stones when we are considering the fate of the calculi after they have left the gall-bladder and reached the bile-ducts. As above mentioned, the smaller stones may be driven by a successful attack of biliary colic into the duodenum, but in other cases the stones are stopped on the way, either in the cystic or in the common duct. If the stone lodges in the common duct there is usually long-continued jaundice with secondary changes in the liver, with which we shall later become better acquainted (*vide infra*, biliary hepatic cirrhosis). The inflammation may, however, abate—the stone remaining quietly where it is lodged, and the duct gradually dilating and, in a certain measure, accommodating itself to the stone; but the mechanical effect of the foreign body will be very likely eventually to occasion fresh attacks of inflammation and the gradual formation of chronic adhesions and distortions of the surrounding parts. There is special danger when there is a considerable pressure-necrosis of the underlying tissue. This immediately opens the door to inflammatory germs, and results in secondary suppuration, which may be either diffuse or localized. In the worst cases there may be purulent phlebitis of an abdominal vein, with metastatic abscesses in other organs.

Precisely similar consequences may, however, arise from stones which are situated in the gall-bladder itself (usually at its neck), with a consequent suppurative infection of the neighboring parts. Perforation into the abdominal cavity leads to purulent peritonitis. If adhesions have already been formed, as is usually the case, there may be perforation in any one of many directions, either outward with escape of the pus and often the gall-stones, and perhaps with the formation of a biliary fistula, or into the transverse colon, or more rarely into the stomach, the portal vein, or the urinary tract; but chief in relative frequency and importance is perforation from the neck of the gall-bladder into the duodenum, for this, as Virchow and Fiedler have pointed out, is the most common way by which large gall-stones reach the intestine and come to be discharged with the fæces. Many of these processes may, under favorable circumstances, lead to spontaneous recovery from cholelithiasis. The gall-bladder, under the influence of suppuration, may finally completely shrivel up, perhaps retaining in its folds one or more calculi; but much oftener, if there is no prompt surgical intervention, the termination is unfavorable. The associated clinical conditions are manifold and they can not be sketched in detail, and the end may come in an acute and stormy manner, or after a long illness marked by many fluctuations. Still, we feel bound to emphasize, in closing, that the frequency of these many grave results of cholelithiasis which we have mentioned must not be over-estimated. In comparison with the extremely great number of gall-stones which produce either no symptoms at all, or none which suggest danger to life, the severe cases of purulent inflammation after cholelithiasis are few.

Diagnosis.—It is evident from what has been already said that often the diagnosis of cholelithiasis is easy and indubitable, while in other cases the symptoms and course of the disease are obscure and ambiguous. The attacks of colic are certainly the most characteristic symptom. We should therefore make it a rule, in case of severe paroxysmal pain in the region of the stomach or liver, particularly in middle-aged women, to think of the possibility of gall-stones. If an attack of this sort is associated in even a slight degree with jaundice, the diagnosis is usually perfectly clear. If there is no jaundice—and frequently there is none—the diagnosis is less certain, but still it can often be made because of the general character of the painful attack; the indications of localized peritoneal irritation, such as slight swelling of the abdomen, tenderness on pressure, and vomiting; and finally because of the discovery on palpation of an enlarged gall-bladder. In every case importance must be laid upon a careful inquiry of the patient or his friends as to any previous attacks or any mild indefinite prodromata, etc. In a certain number of cases, however, it will not be possible to get beyond a mere suspicion that there are gall-stones, and occasionally it will be impossible to avoid confusion with cardialgia, intestinal colic, renal colic, the pains of a movable kidney, and visceral neuralgia. This is particularly apt to happen when the attack is not a well-developed one with characteristic symptoms, but is more indefinite and suggests cardialgia, dyspepsia, and similar conditions. A careful and thorough examination of all the organs that might be involved, such as the stomach and kidneys, may in many cases aid us; and often the truth is revealed by the further course of the disease; but many cases remain obscure. The hope that gall-stones might be recognized during life by means of the Roentgen rays has, unfortunately, not been fulfilled.

We must say a word about finding the gall-stones in the fæces. Of course, the discovery of the *corpus delicti* in the evacuations after an attack of colic makes the diagnosis absolute. They are to be searched for by mixing the stools with water, and then passing them through a sieve. But, as we have pointed out above, the "unsuccessful" attacks of biliary colic are probably much more frequent than the "successful." We can not, therefore, be surprised that in many cases of cholelithiasis no stones can be found in the dejecta, and Riedel is right in maintaining that it is not worth while to search for the stones except in those attacks which are associated with jaundice, that is, those that are not unlikely to be "successful." It should be pointed out further that we should be on our guard not to mistake for gall-stones hardened portions of fæces—for instance, compacted vegetable matter.

Often the diagnosis is very difficult in cases of severe purulent inflammation, originating from gall-stones. If there are such symptoms as indicate a peritonitis localized in the hepatic region, associated with fever and rigors, and suggesting circumscribed suppuration, we must always consider the possibility of gall-stones being the primary cause. We should make a careful local examination and consider the general conditions, such as age and sex, and we shall often be greatly helped by inquiring carefully into the previous health of the patient—e. g., with regard to any characteristic colic. It must be confessed that it will not always be possible to avoid confusion between this and other suppurative processes, particularly those originating from an abnormally situated appendix, or from suppurating echinococci. No special diagnostic rules can be formulated, for the symptoms are peculiar in almost every single case. Even when we are able to make a correct diagnosis of cholelithiasis we are often unable to estimate the exact condition, the extent of the secondary inflammation, the number and the situation of the stones, or the presence of secondary adhesions.

Prognosis.—The prognosis of gall-stones must really be termed doubtful in every case, that is, if we provisionally exclude the possibility of cure by surgical means. The presence of gall-stones, as the reader already knows, may entail a number of dangerous sequelæ, and we can never foretell whether the patient will suffer from these or escape them; still, there is no doubt that in many cases, particularly when the stones are small (*vide supra*), there may be a spontaneous and permanent recovery, while in other cases there may be disturbances of the most varied kind, persistent, or constantly recurrent, and yet never absolutely serious. There is no occasion for describing here a second time the numberless possibilities of cholelithiasis and their prognostic significance.

There is one important symptom which is interesting from the standpoint of general pathology and which must be noted here—viz., the development, subsequent to cholelithiasis, of cancer of the gall-bladder and bile-ducts. This possibility is, of course, of great clinical importance, and agrees with the general observation that persistent mechanical irritation of the mucous membrane and scar formations may occasion the development of carcinoma. The formation of secondary cancer after cholelithiasis is precisely analogous to the growth of cancer after ulcer of the stomach, or secondarily to stone in the kidney.

Treatment.—Our means of efficient prophylaxis against the formation of gall-stones are very limited. The most we can assume is that an entirely suitable regimen, and particularly the avoidance of any tight lacing or similar mechanical interference with the excretion of bile, will prevent or at least delay the development of any tendency to the formation of calculi. If gall-stones have been once formed, our task as physicians consists merely in moderating the disturbance which they produce; in guiding, so far as possible, all sequelæ to a favorable termination; and finally in effecting the removal of the stones from the body, so far as this is necessary and within our power.

If the symptoms of the patient remain comparatively slight, and especially if the diagnosis of gall-stones has not yet been absolutely established, energetic treatment is not demanded. We regard it as important that in such cases, as soon as we conceive the possibility of cholelithiasis, the patients should enter upon a proper rest-cure. Very often female patients of this sort are regarded as nervous, are sent into all sorts of health-resorts, and treated with electricity and massage, and, of course, without success. Instead we should induce such a patient to spend a few weeks of complete rest in bed, or at any rate, lying on a sofa with easy clothing; prescribe a cautious and nourishing diet, and the regular application of warm poultices upon the hepatic region; and administer before breakfast and in the middle of the forenoon a half pint of warm Carlsbad Mühlbrunnen water. We may then hope for a satisfactory improvement in the symptoms due, in our judgment, to the cessation of the slight inflammatory irritation. Often patients of this sort are sent to Carlsbad. We will not deny that Carlsbad water may have some favorable influence upon cholelithiasis, but we believe that the exertion of the journey and the promenades and other activities in which the patients are apt to indulge after their arrival, are often serious drawbacks. That Carlsbad water, as such, is of real value in expelling the stones or in actually dissolving (!) them, we find difficult to believe.

Whenever there is a well-marked attack of biliary colic we have special indications for treatment. Of course, in this case also, we must immediately enjoin complete rest in bed.

The pain demands the prompt employment of narcotics, particularly of morphine or opium. If the suffering is extreme, a subcutaneous injection of morphine is by far the best and quickest remedy; but the internal use of opium is also to be recommended. We may give every two or three hours fifteen or twenty drops of the tincture, or half a grain to a grain of powdered opium (gramme 0.03–0.05).* Other narcotics, such as chloral or belladonna, are scarcely ever necessary, but we may sometimes combine extract of belladonna, which is much praised by many physicians, with the opium. Of external applications upon the hepatic region the best is a warm or hot poultice. It is exceptional for the patient to prefer an ice-bag. Usually, gentle rubbing of the hepatic region with chloroform-oil (a mixture of equal parts of chloroform and olive-oil) does good. In a few cases the patient experiences relief from a prolonged warm bath. If there is violent vomiting we give tincture of opium, bromide of potash, cocaine, or bits of ice. If there is marked collapse, such stimulants must be employed as wine, strong black coffee, or even injections of ether and camphor. When the acute symptoms have ceased, we must still persist for some time in a cautious diet and in bodily quiet, and it is often well to prescribe a gentle laxative, such as Carlsbad salts or rhubarb.

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* [When giving repeated doses of opium for biliary or renal colic, it should be remembered that the pain will cease abruptly as soon as the stone ceases to obstruct the passage, and that severe toxic

When the attack is completely over, the question arises how to guard against the occurrence of others. The answer to this question depends mainly upon the view we take of the present attack, whether we regard it as a "successful" or an "unsuccessful" one. If the attack was successful—that is, if calculi were found in the intestinal evacuations after it—we may hope that in case there are more stones present they also may be discharged. Whether we possess artificial means of insuring this discharge is extremely doubtful. Still, many remedies and methods of cure have been recommended as exerting a favorable influence in this direction. Here again we must mention first the use of Carlsbad and similar waters, such as Homburg, Ems, and Vichy. Many physicians believe that they have seen good results from the persistent use of turpentine, particularly in the form of what is called Durande's remedy; that is, a mixture of ether and turpentine in the proportion of three to two, of which two or three times a day twenty to thirty drops are given; and the same is true of salicylate of sodium, of which half a drachm to a drachm (grammes 2–4) is given daily for a considerable period, either alone or in combination with bicarbonate of sodium. We should also mention in this connection the administration of large doses of olive-oil. This is extremely unpleasant for the patient, and very uncertain in its effect, but still is highly praised by many physicians. A mixture of six or seven ounces (grammes 200) of olive-oil, five drachms (grammes 20) of brandy, and the yolk of two eggs, with a few drops of oil of peppermint, is given in two portions at a short interval. [Most of the oil is passed in lumps, which have a superficial resemblance to gall-stones, and are sometimes called such.] Of course, we should also regulate the diet and the life of the patient in a suitable manner, and this may be the most important prescription of any.

If, however, the attack was unsuccessful, or particularly if several unsuccessful attacks have occurred, there is scarcely room to hope that any internal remedy will result in expulsion of the stone. It may be too large. If, then, we do not choose to let the case take whatever course it will, there is only one remedy available, and that is the operative removal of the stones by opening the gall-bladder. If we consider the dangers which are entailed by the presence of stones that can not be evacuated by the natural passages, we must agree with Riedel when he says a "single unsuccessful inflammation of the gall-bladder is a sufficient indication for immediate operation." To be sure, no one will fail to recognize that the circumstances in practice may prevail over purely theoretical considerations, however lucid the latter may be. Many patients will not consent to an operation after a single brief attack which, although painful, has ended in apparently complete recovery, and the conscientious physician will consider in

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effects of the drug may then appear. In bilious colic the distance to be traversed by the stone is relatively short, and in that affection especially the inhalation of ether or chloroform is sometimes the best, and, indeed, the imperative treatment.]

any actual case the indisputable dangers of the operation, and the possible doubt as to diagnosis. It is a very different matter when the unsuccessful attacks are frequently repeated or when there is persistent discomfort, so that the patient is deprived of all joy in life and all personal activity. Then we ought not and dare not longer delay in urging him to operation. The surgical treatment of cholelithiasis in such cases already possesses a record of many brilliant successes.

There is likewise an urgent indication for prompt operation when the symptoms indicate a purulent cholecystitis or a localized abscess, due to gall-stones. It would probably be better if such complications of cholelithiasis were avoided by anticipatory surgical interference, but this desirable result will never be attained in practice, because in many cases the purulent infection of the gall-bladder or the bile-ducts occurs without having been preceded by any marked symptoms. In these suppurative cases, surgical intervention often not only saves life but works a permanent cure, unless the purulent infection has already extended to the bile-ducts of the liver itself, or has led to general pyæmia. We can not in this place enter into the particulars of the surgical treatment of gall-stones, or the removal by operation of stones fixed in the cystic or common ducts. Complete information will be found in surgical monographs.

CHAPTER IV

SUPPURATIVE HEPATITIS

(*Hepatic Abscess*)

Ætiology.—Exclusive of traumatism there are two ways by which bacteria may penetrate into the liver, there to excite a suppurative inflammation—namely, by the blood and through the bile-ducts. In the circulatory system the main route is by way of the portal vein, by which pyogenic organisms from the intestines reach the liver. This explains why many ulcerative processes in the intestine, such as severe dysentery, are followed by hepatic abscess; and why purulent pylephlebitis (*q. v.*) and other suppurative processes within the portal system may have a similar sequel. In general pyæmia, the germs must take a very circuitous route in order to reach the liver. They must, on leaving the primary abscesses, first enter the veins and the lungs, and then gain the liver by way of the hepatic artery. It has been well known for a long time that suppurating wounds of the head are followed by hepatic abscess with comparative frequency. Perhaps it may exceptionally happen that infectious matter enters the hepatic veins by "retrogressive embolism" from the vena cava.

The germs which make their way into the liver from the bile-ducts invariably originate in the intestine. In these cases the hepatic inflammation is almost always preceded by disease of the biliary passages. The most frequent cause by far of this variety of hepatic abscess is the formation of gall-stones in the gall-bladder and in the liver. This has been fully discussed in the preceding chapter.

Among us, hepatic abscesses are rarely occasioned in other ways than those indicated; but in the tropics it is said that quite a large number of apparently primary hepatic abscesses are met with. Their origin is not yet explained, although in this case also there is most probably an invasion of pyogenic bacteria from the intestine.

Pathology.—The smallest and as yet imperfectly developed embolic abscesses best illustrate the mode of formation. We find the blood-vessels choked with micrococci, and the cells of the surrounding parenchyma void of nuclei and in process of disintegration. Along the course of the blood-vessels nuclei are very abundant. These are due to white corpuscles which have escaped through the vascular walls. The cells and the liquid exudation rapidly increase, and there is complete destruction of the hepatic parenchyma, and the formation of an abscess in its place. This extends in all directions. Large abscesses may at last involve an entire lobe. In other cases the suppurative process is limited by encapsulation. Sometimes quite large portions of the liver become necrotic and slough off, under the influence of what is called "sequestrating" suppuration. We almost invariably find some shreds of hepatic tissue in the pus of hepatic abscesses. When hepatic abscess has been occasioned by gall-stones, the suppuration has usually been due to direct extension of the process from the bile-ducts to the parenchyma of the liver. Not infrequently gall-stones are found in the pus of such cases.

Small abscesses may perhaps be absorbed, but they are in most cases merely symptomatic of pyæmia or some such disease, which is itself incurable. Larger abscesses may point into neighboring organs. If they are discharged into the abdominal cavity, diffuse peritonitis follows. The most favorable termination, and one repeatedly observed, is perforation through the abdominal walls, after these walls and the liver have been joined by adhesions. They may also break into the pleural cavity, the pericardium, the intestine, and the pelvis of the right kidney.

Clinical History.—An absolutely complete clinical description of hepatic abscess is impossible, because, as we have seen, it may be a symptom of such diverse pathological processes. Hepatic abscesses are often found post mortem which had given no previous indication of their presence; this is frequently true in pyæmia. In other cases there are symptoms, in part directly referable to the seat of inflammation, and in part due to its influence upon neighboring organs.

Enlargement of the liver can often be made out by percussion or even by palpation. It is the result of swelling and hyperæmia involving the entire organ. Extensive abscesses may give much more definite signs of their presence, however, if situated on the anterior surface of the organ. They are sometimes felt through the abdominal walls as hemispherical and actually fluctuating tumors. It is not so very rare for tropical hepatic abscess to attain these dimensions.

Pain in the right hypochondrium, although it may be entirely absent when the abscesses are small, even if they are numerous, is often violent and persistent when the abscess is large. It is excited by the tension of the peritoneal covering of the liver, or by perihepatitis. The pain often radiates—and with especial frequency, it is said, into the neighborhood of the right shoulder.

The course of the fever may prove a strong diagnostic point. When the abscess is chronic and encapsulated there may, it is true, be no fever whatever; but, as a rule, fever does exist, and it often presents a very characteristic intermittent character. There are great elevations, usually ushered in by a chill, and succeeded by deep depressions of temperature accompanied by perspiration. If the hepatic trouble is merely a symptom of general pyæmia, then the fever is to be ascribed to the latter; but if there are signs of a severe local hepatic disease, such as pain, enlargement, and perhaps jaundice, and if these febrile attacks come on at irregular intervals, we should always consider the possibility of abscess of the liver. In the cases of large tropical abscess this sort of fever is the rule. It is most frequent with us in cases of purulent pylephlebitis and of abscess excited by gall-stones. The "*fièvre intermittente hépatique*" of the French is in most instances due to the presence of gall-stones in the liver, with secondary suppuration and abscess formation.

Among the secondary symptoms of hepatic abscess jaundice is prominent. It is not invariably present, however, occurring only when the abscess has compressed some large biliary duct, and has thus given occasion to the absorption of bile by the lymphatics, or when an extensive cholangitis has simultaneously developed, sufficient to cause jaundice. In rare instances the abscess compresses the portal vein and thus causes ascites. There may be pulmonary symptoms of considerable importance, even when there are no actual pulmonary complications. This is because the right half of the diaphragm is crowded up by abscesses projecting from the convex surface of the liver. Hiccough is sometimes a source of distress, and it may be due to the pressure of the abscess upon the stomach. Vomiting is also a rather common and often very troublesome symptom.

There is almost always great constitutional disturbance. The patient has no appetite, and loses flesh, particularly if there are frequent febrile exacerbations. Often there are severe nervous attacks. Very exceptionally, the disease remains latent for a long time, and does not disturb the general health to any great extent.

The course of the disease depends mainly upon the nature of the original disturbance. Severe pyæmic cases, in which hepatic abscesses develop, are generally brief, and are almost invariably fatal. Abscesses due to gall-stones, and the large abscesses which are apparently idiopathic, are generally chronic, lasting for weeks, or even for many months. Cases exhibit manifold diversities, according to the position, size, number, and sequelæ of the abscesses. Among the possible results, we would once more call attention to perforation into neighboring organs. If the pus is discharged externally, recovery may ensue; as also if the pus reaches the intestinal canal or the bronchi, which seldom happens. Perforation into the abdominal cavity always excites a fatal acute peritonitis. As a general rule, hepatic abscess finally proves fatal, recovery being exceptional. Death is due either to the gradual loss of strength or to some complication.

Treatment.—Local bleeding, counter-irritation, purgatives, and emetics are among the remedies which are advocated, but we can hardly expect them to exert much influence upon a hepatic abscess. The best way is to treat the case purely symptomatically, seeking to keep up the patient's strength and mitigate his suffering until, if we are very fortunate, we have a chance for operative interference. When once the other symptoms are re-enforced by the discovery on palpation of a fluctuating tumor, the diagnosis is established, and the pus should be evacuated and the cavity drained. Particulars about the operation should be sought in works on surgery. More than one case of the large tropical abscess have been cured in this way; but the cases which are most common among us—namely, embolic abscesses and those excited by gall-stones—hardly ever afford any opportunity for surgical interference.

CHAPTER V

CIRRHOSIS OF THE LIVER

(*Chronic Diffuse Interstitial Hepatitis. Laennec's Cirrhosis. Gin-drinker's Liver. Hob-nailed Liver*)

Ætiology and Pathology.—Cirrhosis of the liver is usually defined as a diffuse interstitial inflammation, chronic in duration, and resulting in a secondary atrophy of the true hepatic parenchyma. This conception makes the disease perfectly analogous to "chronic interstitial inflammation" of the kidney and many other organs. Weigert's careful study of the processes of "chronic interstitial nephritis" has shown that at least a large part of the changes which take place in the connective tissue are not primary, but secondary, and the consequence of a primary destruction of the genuine renal parenchyma. The question naturally suggests itself, whether the same may not be true of the apparently closely allied phenomena of hepatic cirrhosis. We feel, therefore, that in most cases of hepatic cirrhosis the origin of the disease consists of a primary injury and consequent partial destruction of the hepatic cells, whereupon follow a secondary proliferation and final contraction of the interstitial tissue, just as is seen in lesions of the parenchyma of the kidneys, spinal cord, and heart.

Such a conception would be extremely compatible with one fact about the ætiology of the disease—namely, that chronic alcoholism is universally regarded as a potent predisposing cause. Hence the English name, "gin-drinker's liver." The harmful influence of alcohol can be appreciated if we remember that on being absorbed it is carried directly by the blood-vessels through the portal vein to the liver. According to the usual view of the disease, the poison excites a chronic inflammation of the connective tissue; while, according to the new view, the alcohol exerts a specific injurious influence upon the hepatic cells proper, impairing their nutrition, and finally causing their destruction. That the disease attacks the periphery of the lobules and the interlobular connective tissue is equally consonant with either theory. It is well known that the capillary anastomoses of the portal vein are situated between the lobules.

Hepatic cirrhosis is most common in those who drink distilled liquors, while excessive indulgence in less strongly alcoholic beverages, such as wine and beer, leads less frequently to cirrhosis. Still, we have repeatedly seen the disease in beer drinkers.

The abuse of alcohol is surely the chief but by no means the only cause of cirrhosis, for quite often the disease attacks persons in whose case no such ætiology is possible. In such instances we are seldom able to demonstrate the real cause. The excessive use of spices, and other analogous substances, has sometimes been regarded as causative. It is also said that malaria and the acute infectious diseases sometimes leave behind them a tendency to cirrhosis. In our opinion such cases are due to the long-continued action of chemical agents and bacteria, about which we do not as yet possess any accurate knowledge, which are conveyed from the intestine to the liver. These cases of "genuine hepatic cirrhosis" are analogous to the not infrequent cases of "genuine interstitial nephritis" of unknown origin. Perhaps in many instances, also, there is a constitutional and excessive sensitiveness of the hepatic cells to the injurious influences to which they are exposed while performing their normal functions.—The form of cirrhosis which follows diseases of the bile-ducts, and also "syphilitic cirrhosis," will receive separate consideration.

Cirrhosis of the liver is seen much oftener in men than in women, and usually occurs in middle life. This is in harmony with the fact that the chief cause is the abuse of alcohol.

The anatomical changes are generally divided, without regard to the way in which they are brought about, into two stages. In the first stage the liver is uniformly enlarged, resistant, with its edge blunt, and its surface at first perfectly smooth, but later presenting little dimples. On section, the increased consistency, or "interstitial induration" of the liver, can be readily perceived. The acini are separated from one another by a relatively thick band of grayish-red interstitial tissue, and are at first readily distinguishable. Later on, the interstitial hyperplasia invades the acini themselves, and they cease to be discernible. The microscope shows that the cause of this increase in size and firmness of the organ is due exclusively to the abundant cellular infiltration and the new formation of connective tissue between the individual lobules. The neighboring cells of the parenchyma exhibit signs of disintegration, undergoing either simple atrophy or else fatty degeneration.

The second stage corresponds with the process of contraction of the newly formed connective tissue, but in this stage the destruction of the proper hepatic tissue has already assumed grave proportions. On the old theory, the parenchyma perishes because of the great disturbance of circulation in the portal capillaries, great numbers of which are obliterated by the shrinking of the connective tissue. We ourselves, as we have already intimated, incline rather to Weigert's view, that the atrophy of the hepatic cells is, as a rule, the direct result of a primary pathological lesion of the hepatic tissue. Under this process of contraction the liver undergoes progressive atrophy, and its surface becomes mammillated. The size of the nodules varies. The size of the whole organ may be reduced one half, or even more. Frequently its general contour is considerably modified. Often the diminution of the size of the liver is confined mainly to the left lobe. The serous coat of the liver is usually thickened and rendered opaque; the hepatic tissue feels very resistant and cuts with difficulty, often with a grating sound; the color of the cut surface is usually a rather light yellow (κιρρός = yellow). Upon microscopic examination, we find merely vestiges of parenchyma, embracing which are wide, firm bands of connective tissue. Even within the acini there is decided interstitial hyperplasia along the blood-vessels. The hepatic cells exhibit various stages of fatty degeneration and simple atrophy. Brown masses of pigment are often found here and there, which have been left behind by the hepatic cells now destroyed. Regenerative changes can also be detected quite frequently. The most common of these is the formation of small biliary passages in the broad bands of interstitial tissue.

The division of hepatic cirrhosis into two stages is somewhat schematic, for there is really no sharp dividing line between them. The same liver may in different places illustrate both stages simultaneously. Thus, the surface is often distinctly granular, while the liver as a whole remains hypertrophic. In many cases there seems to be no so-called "first stage" at all, and the previously normal liver is directly transformed into a contracted liver, perhaps comparable to the kidney of genuine interstitial nephritis.

Clinical History.—The onset of the disease is usually insidious. At autopsies quite an advanced stage of cirrhosis is sometimes found, to which not a single clinical symptom had pointed; and it is often observed that the duration of unambiguous symptoms is much shorter than the degree of anatomical change discovered post mortem would have led us to expect.

It is, however, true that certain prodromata may appear long before the genuine cirrhotic symptoms; but there is generally room for doubt whether these prodromata are excited by the incipient hepatic disease or whether they are not due to other coincident affections, such as the chronic gastric or intestinal catarrh which drunkards so often have. There are anorexia, nausea, epigastric uneasiness, eructations, constipation, and sometimes vomiting. There is evident constitutional disturbance in many cases, but in others the strength is unimpaired. The severer symptoms usually date from the time when disturbance of the portal circulation arises. We have already stated that the diseased process is most active in the interlobular connective tissue—that is, where the portal capillaries are situated. When the contraction of the connective tissue has resulted in the destruction of a large number of these portal capillaries and the minute veins from which they spring, the portal circulation is inevitably impeded, and there arises a passive congestion of the whole portal system. The signs of this are soon manifest.

The stasis in the veins of the peritoneum is, as a rule, the first to attract attention, from the ascites which it occasions. The distention of the abdomen and the sensation of weight and pressure due to this effusion, are often the first things which excite the patient's attention and lead him to seek medical advice. Later hypertrophic. The demonstration of atrophy by percussion in the later stages of the disease is less reliable. The ascites often interferes with such an attempt. We may also be misled by coils of intestine distended with gas and perhaps lying in front of the liver. If, however, after guarding against error, we constantly find the area of hepatic dullness diminished, the sign has some value. Moreover, it is not infrequently possible to make out by palpation that the liver, although reduced in size, is hard and uneven. This is easier after the removal of ascitic fluid, when the abdominal walls are lax, and also if the attachments of the liver have been loosened so that it sinks downward.

General nutrition is usually much impaired in the later stages of the disease. At first the patient may retain vestiges of his former corpulence, but finally he grows emaciated. Anasarca may exceptionally occur toward the close; but there is frequently considerable œdema of the lower extremities, and even of the scrotum and the dependent portions of the abdominal walls. The cause of this is a purely local one—the pressure of the ascites impedes the return of blood from the lower limbs to the heart. Thus arises a condition which is characteristic of portal obstruction in general, and hepatic cirrhosis in particular, and has been termed "œdema of the lower half of the body." There is ascites with œdema of the loins, the scrotum, and the lower extremities; while the upper extremities, the upper half of the trunk, and the face are entirely free from œdema.

Occasionally there are ecchymoses into the skin, the mucous membranes, the retina, and other parts. These are probably due to malnutrition of the vascular walls. The gastric and intestinal hæmorrhages have been already discussed.

In uncomplicated cases of the disease there is no fever. The pulse, even when there are no cardiac complications, is small and often somewhat rapid. Respiration may be much embarrassed, as a result of the upward pressure of the diaphragm.

At first the urine presents no characteristic changes. When the ascites has become considerable and there is œdema, the urine grows scanty, dark, and concentrated, and often has an abundant sediment of urates. It should be mentioned that some observers have found a diminished excretion of urea. This is perhaps due to a disturbance of the urea-generating function of the liver, about which both earlier and more recent investigators agree. In a few instances the urine has been found to contain a trace of sugar. As a rule, however, the urine contains no sugar whatever, not even when an attempt is made to induce an alimentary glycosuria.

It remains for us to describe briefly the collateral circulation which may be developed in cirrhosis, so as to enable the blood of the portal system to reach the systemic veins. The clinical history of the disease does not indicate that this attempt at compensation is very successful. We may have: 1. Communications between the veins of the mesentery and of the abdominal walls. 2. Communications between the coronary vein of the stomach and the veins of Glisson's capsule on the one hand, and the phrenic veins on the other. 3. Anastomoses between the internal hæmorrhoidal and the hypogastric veins. 4. As pointed out by Baumgarten, enlargement of the not yet completely obliterated umbilical vein in the ligamentum teres. Through all these the blood may flow from the portal system into the veins of the abdominal walls—that is, in the reverse of the normal direction. Then in a few exceptional cases the veins around the navel become varicose, a phenomenon which has been termed the "*caput Medusæ.*" Much more frequent and more important is the finding of unnaturally distended and tortuous veins in the skin of the abdominal walls, when there is ascites. These veins usually extend upward from the neighborhood of Poupart's ligament. This phenomenon is due to the fact that because of the ascites the flow of venous blood from the lower extremities through the iliac veins is impeded (*vide supra*), and consequently a part of the venous blood takes its course through the inferior and superior epigastric veins to the mammary veins.

The complications which are seen in many cases are probably due in part to the same influences which occasion the cirrhosis. In this list belong, for example, cardiac hypertrophy, intestinal nephritis, and chronic pachymeningitis. Of special interest, because of its diagnostic and therapeutic importance, is the combination of hepatic cirrhosis with tubercular peritonitis. As we have already seen (page 531), hepatic cirrhosis is probably in most cases the primary disease, and occasions a predisposition to the development of tubercular peritonitis. The clinical picture in these cases is a combination of the symptoms of hepatic cirrhosis, such as a palpable granular liver, splenic tumor, and yellowish complexion, with the symptoms of tuberculosis, such as irregular distention and tenderness of the abdomen, persistent fever, and emaciation. If we consider the ætiology (alcoholism), we may, therefore, sometimes make a complete diagnosis of this combination, particularly if at the same time the tuberculous inflammation attacks other serous membranes, such as the pleura.

As to the general course of cirrhosis, its duration can not easily be determined because the onset is usually insidious. As a rule, the disease lasts one to three years, or rarely longer. In many cases the symptoms are insignificant for the first six to eighteen months. Then the disorder takes on a severer form, perhaps rather suddenly. Ascites appears, for example. These graver symptoms persist, until after a few months the patient dies. The course of the disease reminds one of cardiac cases, where for a long while the compensatory changes avert any distress, until on a sudden the circulatory disturbances become pronounced and persist to the end.

The **prognosis** is always unfavorable, at least when the symptoms have once become well marked. It may be that in the earlier stages the disease can be checked or even permanently cured; but even this is open to doubt. No case recovers in which the diagnosis of cirrhosis is certain.

Death is due either to intercurrent disease, or more often to gradually increasing exhaustion. In some few cases severe cerebral symptoms suddenly appear: there are coma, general convulsions, and delirium; and these usually are soon fatal. Just how these nervous phenomena originate we do not certainly know (see the chapter on acute yellow atrophy of the liver).

Diagnosis.—The diagnosis of hepatic cirrhosis is not usually self-evident. It may be made with some positiveness if a patient who has a previous history of alcoholic excess gradually develops ascites and splenic tumor, and if upon palpation we find the liver unnaturally firm and hard, either enlarged or shrunken, perhaps with a roughened surface, and in any case distinctly to be felt; yet in palpating the granular surface one is very liable to errors, and particularly the separate kernels of fat in the *panniculus adiposus* of the abdomen are apt to be mistaken by inexperienced observers for irregularities in the surface of the liver. The difficulties in the percussion of the liver have been already indicated. Often a patient does not come under observation until a considerable ascitic effusion has already taken place, so that physical examination of the liver and spleen is rendered very difficult, if not impossible. Then we must first exclude any general disturbance of circulation as a cause of the ascites. If the heart, lungs, and kidneys are found to be normal, and if there is no œdema in the upper half of the body, it is very probable that there is a local derangement of the portal circulation, particularly if the patient is positive that the abdominal enlargement was the first evidence of dropsy; but we have still to determine whether the cause of this derangement is cirrhosis of the liver. This may be assumed to be the case if the whole course of the disease warrants the assumption, and if the history furnishes that most frequent of all ætiological factors, chronic alcoholism. Otherwise we must be cautious, for portal obstruction with precisely similar results may be due to other causes—such as the external pressure of tumors or portal thrombosis. Many forms of hepatic syphilis (*q. v.*) can not be differentiated from cirrhosis by mere clinical observation of the hepatic disorder. Here it is only the ætiology and the demonstration of other signs of syphilis that can justify the assumption that the disease in hand is of specific origin. It is often a great aid in diagnosis to examine the liver directly after tapping. The abdominal walls are then relaxed and soft, so that the liver may be made out very distinctly. It should be borne in mind that irregularities of considerable size are more frequent in hepatic syphilis ("lobulated liver"), while cirrhosis is suggested by a more uniform granulation.

It is also very difficult in many instances to exclude chronic peritonitis. The ætiology may aid us. Other points are, that in chronic peritonitis there may be tenderness on pressure, the abdominal distention is less uniform, and there is no enlargement of the spleen. Another point is that the specific gravity of a simple ascites due to passive congestion is often below 1015, while an inflammatory exudation usually reaches higher figures. Hæmorrhagic ascites, as a rule, indicates tuberculous or cancerous peritonitis. The combination of hepatic cirrhosis and chronic peritoneal tuberculosis (*vide supra*) may be diagnosticated with a certain probability if, besides symptoms of cirrhosis and peritoneal disturbance, there are definite indications of a tuberculous affection; such are a phthisical constitution, persistent fever, and the existence of a tuberculous disease in some other organ, particularly the pleura.

Treatment.—In treating hepatic cirrhosis the first necessity is a strict regulation of the diet. The earlier in the disease proper diagnostic rules are followed, the more likelihood of benefit. The most important principle in regulating the diet is to avoid all ingesta which in any way might irritate or damage the hepatic cells. We forbid, therefore, all alcoholic beverages, or at most allow very small amounts merely to stimulate the appetite. We likewise forbid sharp spices, pepper, mustard, horse-radish, onions, and similar articles. In general, the amount of meat should be limited. We may allow milk, buttermilk, eggs, vegetables, and cereals. In suitable cases we may try the prescription of an exclusive milk diet for some weeks. Quite a number of authors report favorable results from a milk-cure of this sort, but the individual peculiarities of every case must be considered.

If the disease has already developed we must resign ourselves to merely symptomatic treatment; at least, the specific benefit which iodide of potassium is said to exercise in hepatic cirrhosis is very doubtful, and is probably based merely upon cases of syphilitic disease of the liver.

The symptomatic treatment of hepatic cirrhosis has to deal mainly with the disturbances due to portal congestion, and the associated gastro-intestinal catarrh. In such cases experience shows that the best results are obtained by the cautious use for a considerable time of laxatives. In early cases we should resort first to salines, particularly Carlsbad salts. We may prescribe artificial Carlsbad salts, dissolved in warm water, or the genuine Carlsbad water to be drunk at all times. If the patient's circumstances permit we may even recommend in suitable cases a visit to Carlsbad. Other appropriate health-resorts are Marienbad, Kissingen, Ems, and Neuenahr. If the saline remedies are ill-borne we employ vegetable laxatives, such as rhubarb and aloes. In case there is already a considerable degree of ascites, good results may sometimes be obtained by administering drastic purgatives, among which gamboge has earned a special reputation. Many observers have seen benefit from calomel, giving three times a day three grains (gramme 0.2) for a period of two to three days. Calomel may be given repeatedly in this way at intervals of from five to eight days. Of course, caution is necessary in its employment. Probably it does good both as a laxative and also as a diuretic (*vide infra*). Sasaki, in Japan, has seen excellent results in hepatic cirrhosis from the administration of cream of tartar in considerable doses, two to four drachms (grammes 10 to 15) a day, associated with a nourishing diet, including milk and scraped raw meat. After all, however, laxatives should not be employed if they disturb the digestive apparatus.

If ascites has already developed, we may administer diuretics besides the drastic purgatives; trial may be made of acetate of potassium or sodium, squills, diuretine, and the species diureticæ of the German Pharmacopœia (containing lovage, rest-harrow, licorice-root, and juniper berries). English authorities recommend the balsam and the resin of copaiba as particularly efficient in the various forms of ascites. The dose is about fifteen minims (gramme 1) a day. The best mode of administration is in gelatine capsules. This remedy occasions in some cases a rapid increase in diuresis, and a consequent diminution in the ascites. Sometimes, also, pure urea (2.5–4 drachms, grammes 10–15, daily, dissolved in water) acts as a good diuretic. If the heart is feeble, digitalis may cause a decided increase in the amount of urine, and it is also worth while to try a combination of digitalis with calomel.

If the ascites is so excessive as to occasion much local discomfort and to impede respiration, the removal of the fluid by paracentesis may afford relief. The details of this proceeding were given in the last section. Many physicians recommend tapping as early as possible, before it is absolutely necessary. The relief is said to be more lasting in that case; but this proposal has by no means met with universal assent, and, as a rule, the physician will not tap until the abdominal tension, the dyspnœa, and other symptoms occasioned by the ascites render it necessary. Unfortunately, the relief is only temporary, for the fluid collects again with great rapidity after tapping; yet it is sometimes possible to delay the reaccumulation for a considerable time by compressing the abdomen with an elastic bandage, and by using the above-mentioned remedies (diuretics and digitalis) directly after tapping.

[In 1896 Drummond and Morison proposed an operation to relieve the venous stasis, by producing adhesions between the liver, spleen, and omentum and the parietal peritoneum. A few successful cases have been reported. Three known to one of the translators all died.—V.]

CHAPTER VI

BILIARY CIRRHOSIS AND HYPERTROPHIC CIRRHOSIS OF THE LIVER

There are two forms of cirrhosis which differ in many respects from the disease just described: they are called biliary cirrhosis and hypertrophic cirrhosis of the liver. Charcot and Hanot were the first to call attention to them. Since then the literature of the subject has become quite extensive, but all doubts and differences of opinion are not yet settled.

1. **Secondary Biliary Cirrhosis of the Liver.**—Whenever there is retention of bile in the liver for any length of time, no matter what causes it, certain changes may result. The small and the medium-sized bile-ducts become distended, and granules of pigment are deposited, both in the interlobular connective tissue and within the acini themselves. Besides this, however, and undoubtedly because of the noxious influence of the retained bile, the hepatic cells not infrequently un-

dergo destructive changes. In accordance with the general rule, connective tissue gradually fills the gaps thus left in the parenchyma, and, more than this, the interstitial hyperplasia is so great as to increase the size of the organ. If, therefore, there is persistent obstruction of the common duct by a gall-stone, or a cicatricial stenosis, or a tumor pressing from without upon the duct, the liver will, in all such cases, be found to be larger, firmer, and richer in fibrous tissue than normal—in a word, "cirrhotic." Hence this condition does not represent an independent disease, but is a result of chronic biliary retention, in whatever way occasioned. It is appropriately termed "secondary biliary cirrhosis." That retention is really the cause of this change has been proved by experiments, for it has been shown that ligature of the common duct in animals causes well-marked biliary cirrhosis.

2. **Primary Hypertrophic or Biliary Cirrhosis.**—The secondary cirrhosis just described is due to occlusion of the large bile-ducts. There is also a not very common primary form of biliary cirrhosis, usually known as hypertrophic cirrhosis. French authors have given it the name of "*cirrhose hypertrophique sans ascite avec ictère*," out of regard to its most important clinical symptom. That there is an essential difference between this form and the ordinary "atrophic" cirrhosis of Laennec is manifested by the clinical behavior of the disease.

The specific cause of hypertrophic bilious hepatic cirrhosis is as yet entirely unknown. We can merely surmise that we have to do with a chronic inflammatory process, excited by some toxic or infectious agent, and that the process probably originates in the small bile-ducts of the liver.

Often this disorder attacks hard drinkers, but they are not by any means its only victims. While, in the common form of cirrhosis, ascites is usually the earliest grave symptom of disease, in hypertrophic cirrhosis a slight jaundice generally appears simultaneously with the first indefinite symptoms of pressure in the region of the liver, languor, and anorexia. This jaundice rapidly increases, and persists throughout the illness. In ordinary cirrhosis there may be, as we have said, hardly any jaundice, or, at any rate, it is a rather late symptom, and even then it is seldom extreme. On the other hand, ascites, as a rule, is slight or absent in hypertrophic cirrhosis. It is true that there have been cases with great ascitic effusion, but it never comes till the disease is quite far advanced.

On physical examination the liver is usually found to be considerably enlarged, and its surface is smooth, as a rule, or rarely it displays some low elevations, due perhaps to variations in the fullness of the blood-vessels. In general there is said to be this important difference between the ordinary and the hypertrophic forms, that in the latter the newly formed connective tissue evinces little tendency to contraction, so that the liver remains large, even late in the course of the disease, and does not shrink. This is certainly true of the majority of cases, so that after an illness of two or three years the liver is still found to be large and smooth; but further observations are necessary in order to determine whether there may not sometimes be contraction toward the end of the disease.

Along with the chronic jaundice and the marked enlargement of the liver there is a third important symptom—viz., chronic splenic tumor, usually of considerable size. As there are no indications of portal obstruction, we must conclude that the spleen is not enlarged from passive congestion, as is usually the case in ordinary alcoholic cirrhosis, but as the result of hyperplasia; and post-mortem examinations confirm this conclusion. The origin of the hyperplasia is not clear. In one case we found not only splenic tumor but a distinct hyperplasia of many mesenteric and peritoneal lymph-glands; which suggests the possibility of infectious influences. A corroborative fact is that not infrequently the entire course of the disease will be marked by slight elevations of temperature. The other symptoms of the disease are in part the direct result of the jaundice, such as the slow pulse, the itching, and the changes in the urine; while others, such as languor and weakness, are the results of the constitutional disturbance. The dejections are not always colorless and fatty, as in case of obstruction of the large bile-ducts. Many patients have a noticeable tendency to hæmorrhage, particularly to hæmatemesis. In the case mentioned above, the fatal termination was due to constantly repeated and profuse hæmorrhages from the stomach, for which the autopsy disclosed no anatomical cause in the stomach or intestine. It seemed as if the hæmorrhages had taken place by diapedesis.

The entire duration of the disease is about one or two years; but it may last much longer. The prognosis is almost always bad. Occasionally a case will exhibit marked temporary improvement or an apparent arrest of the disease, so that, in spite of the size of the liver, the patient is able to do light work. Death comes, when there are none of the above-mentioned hæmorrhages, as a result of gradual exhaustion, or is suddenly ushered in by coma, convulsions, and other grave nervous symptoms, usually ascribed to cholæmia (*vide infra*).

That a chronic hepatic disease of so peculiar a nature should be marked by peculiar anatomical changes would seem certain, even from clinical observation. In general, this view is confirmed, although further and careful investigations are much needed. Upon microscopic examination there is always found an abundant new growth of a tissue containing many cellular elements, surrounding the small bile-ducts of the liver. In other particulars the histological changes are very similar to those of ordinary hepatic cirrhosis. The new growth of connective tissue is found in the interior of the hepatic lobules, but this can scarcely be regarded as a radical difference. Still, one gets the impression that the entire pathological process of hypertrophic cirrhosis originates in the bile-ducts and the tissues surrounding them, while the changes of ordinary alcoholic hepatic cirrhosis start from the branches of the portal vein. The absence of contraction in the new tissue of hypertrophic cirrhosis seems to be consonant with the fact that the anatomical changes suggest hyperplasia rather than inflammation (compare the changes in the spleen and the lymph-glands).

The diagnosis of hypertrophic cirrhosis can sometimes be made with considerable positiveness, and sometimes can merely be regarded as probable. The gradual development and persistence of jaundice and the presence of an enlarged liver and spleen, but usually without marked ascites, would suggest the disease strongly, especially if long-continued observation enabled us to exclude carcinoma and other diseases. Sometimes, however, it is very difficult to decide whether the biliary cirrhosis is primary or secondary, for the conditions giving rise to secondary biliary cirrhosis (occlusion of the bile-ducts from scars, new growths, or calculi) may be obscure. A careful investigation into the history of the case with regard to biliary colic and similar symptoms might be decisive. Other factors are the general course of the disease and the presence of a splenic tumor, unaccompanied by ascites. An enlarged spleen is scarcely ever seen in this way in ordinary chronic jaundice of obstructive origin.

The treatment should conform to the principles laid down in the chapters on jaundice and ordinary cirrhosis of the liver. Sacharjin has lately recommended the persistent use of small doses of calomel—one grain (gramme 0.06) several times a day.

CHAPTER VII

ACUTE YELLOW ATROPHY OF THE LIVER

Ætiology.—Acute fatty degeneration of the liver occurs both as a primary disease and as secondary to other hepatic disorders, or as a symptom of constitutional diseases. Secondary acute fatty degeneration in rare instances accompanies severe acute infectious diseases, such as typhoid fever, recurrent fever, septicæmia, and puerperal disease. It also appears, with equal rarity, in the course of cirrhosis of the liver or of persistent biliary retention; and it is a constant phenomenon in acute phosphorus poisoning. Indeed, the effects of phosphorus resemble the symptoms of primary acute yellow atrophy in many ways so closely, even post mortem, that formerly the two were often confounded.

Primary acute yellow atrophy of the liver is a very rare but extremely severe disease which almost invariably leads to speedy death. There is generally no determinable cause, and its victims are struck down in blooming health. It is most common in young adult life between the fifteenth and thirty-fifth year. Children and elderly people have been occasionally attacked. Females are much more liable to the disease than males; and pregnancy increases the predisposition to it.

As we have said, we can not as a rule find any exciting cause. It is stated that sometimes the onset has been preceded by some violent emotional excitement, or excess in alcohol, or the like; but how important these factors may be is not at all clear.

It is an interesting fact that sometimes the disease becomes rather more frequent than usual, and endemic. For instance, several members of one family may be attacked. This favours a theory as to the nature of acute yellow atrophy which a majority of the present investigators seem inclined to adopt. The theory referred to is suggested not only by the whole course of the disease, but by the pathological appearances, and places it in the category of acute infectious diseases. It must be confessed that as yet we know nothing about the intimate nature of the infection.

Pathology.—The chief change found post mortem is in the liver, and it has determined the name given to the disease.

The liver is much atrophied, sometimes being only one half or one third its normal size. This makes its capsule often seem contracted and wrinkled. The organ is usually soft and flabby, so that in some places it seems as if the finger could be pressed into it. The color of the surface, and for the most part of the cross-section also, is yellow, like ochre or saffron; but the cut surface may be parti-colored, having red and yellow spots interspersed. Hence the names "red atrophy" and "yellow atrophy." The arrangement and relative extent of these patches may vary exceedingly. The red places look as if they had collapsed, and seem tougher than the yellow. They correspond, as we shall soon see, to the more advanced stages of the affection, while the yellow spots have undergone less change. The lobules are, as a rule, no longer distinguishable by the naked eye. Such lobules as can still be made out seem abnormally small and have a gray periphery.

On microscopic examination, we find that the essential change is an intense and uniform fatty degeneration of the hepatic cells, affecting the entire parenchyma. Only a few cells still retain their normal condition. The others are filled with large and small fat-globules, and many are already suffering evident disintegration and absorption. Where the degeneration is furthest advanced, fat, detritus, and pigment alone remain. Inasmuch as the lymphatics rapidly absorb and remove the fatty and albuminoid granules, there is finally little left except blood-vessels and connective tissue. The blood-vessels are frequently quite congested, and thus occasion that red color which the naked eye detects in the more advanced, broken-down portions. Frerichs made an interesting discovery, which deserves mention, of the existence of leucine and tyrosine crystals both in the parenchyma and in the blood-vessels. Bilirubine crystals also are sometimes found in the detritus, and more rarely in the interior of the hepatic cells.

Not only the liver, but many other organs present fatty degeneration: the heart in particular, the kidneys, and rarely the muscles; but the process is always most intense in the liver. The skin (*vide infra*) and most of the viscera are evidently tinged with jaundice.

Acute splenic tumor is invariably present. This suggests that the disease may be infectious. That the disease is a constitutional one is also to be inferred from the numerous ecchymoses in the skin and the interior of the body, especially in the mucous membrane of the stomach and intestines, in the serous membranes, in the pelvis of the kidneys and the kidneys themselves, and more rarely in the brain and heart. This, again, is like the grave septic diseases. The blood itself is dark, with few clots. Leucine and tyrosine have repeatedly been detected in it. The peritoneum and other serous cavities sometimes contain a considerable amount of serum.

Clinical History.—The disease is usually divided into two stages, the first of which corresponds to the milder prodromal symptoms, the second to those severe symptoms which are alone characteristic. In many instances, however, the first period is wanting, or is so brief that the patient is plunged almost without warning into the gravest condition.

The prodromata in most cases consist of constitutional disturbances and mild gastro-intestinal symptoms. The patient is languid, without appetite, and disinclined to exertion. There are headache, nausea, vomiting, slight pains in the gastro-hepatic region, and sometimes moderate fever. In a few days jaundice usually appears. This is almost invariably taken for an ordinary catarrhal attack.

After some days, or it may be weeks, the second stage begins. The chief characteristic of this is the occurrence of grave nervous symptoms. First there is violent headache, with sleeplessness and marked restlessness. The intellect is usually somewhat dulled even now, and articulation is slow and clumsy. The mental confusion usually advances very rapidly to a noisy and violent delirium. The excitement becomes at times maniacal. The patient screams and storms, and he can hardly be kept in bed. Often there are convulsive twitchings of individual muscles; and there may be typical epileptiform attacks, but this is not common. After one or two days, or rarely longer, the excitement abates, and is followed by sopor, which soon passes into deep coma. At death the patient is usually perfectly unconscious. It is exceptional for the excited stage to be wanting; in such cases the first nervous symptom is sopor.

The cause of the nervous symptoms has not yet been explained in a way to silence discussion. The same theories which have been set up to account for the grave form of jaundice in general (see appendix to this chapter) have also been employed to elucidate the nervous phenomena of acute yellow atrophy. Thus, some refer them to cholæmia, some to acholia. In acute yellow atrophy of the liver it might also be that the primary specific processes of infection or intoxication are factors in the development of the severe cerebral symptoms.

The jaundice, which is present even in the first stage, afterward usually deepens. The urine contains bile-pigment, and many investigators have also found bile-acids in it. If these latter are present, it suggests that the jaundice is not (as was formerly believed) hæmatogenous—that is, the result of a destruction of red blood-corpuscles and the transformation of their pigment into biliary coloring matter—but is due rather to a retention of bile. Just how this retention arises we do not yet know definitely. The obstruction can not be in the large bile-ducts, for the gall-bladder is usually found empty. Hence the cause of the retention of bile and of the jaundice is probably a derangement of the smaller biliary passages within the liver. We should add that in a few rare cases there has been little or no jaundice.

On physical examination of the liver during the last stage of the disease, there is usually a striking diminution of hepatic dullness, corresponding to the atrophy of the organ. Generally the first change to be detected is a contraction of the left lobe, as shown by the development of tympanitic resonance in the epigastrium. At the commencement of the illness, the hepatic dullness is normal or slightly increased in area. If the disease proves very rapidly fatal, the organ may never become very small. In most cases, though by no means in all, there are pain and tenderness in the hepatic region, but these are seldom so great as in phosphorus poisoning.

The enlargement of the spleen has been already mentioned as an almost constant symptom of the disease. Even during life some increase of the area of splenic dullness can usually be made out, and sometimes the spleen can be felt under the edge of the ribs.

The occurrence of the hæmorrhages, which have already been referred to under the pathological lesions, can often be demonstrated during life. The cutaneous ecchymoses can, of course, be seen, and the hæmorrhages in the mucous membranes may give rise to hæmatemesis, bloody stools, bleeding from the female genitals, or epistaxis. The hæmorrhages are due probably to the impaired nutrition and diminished resisting power of the vascular walls occasioned by the grave constitutional disturbance.

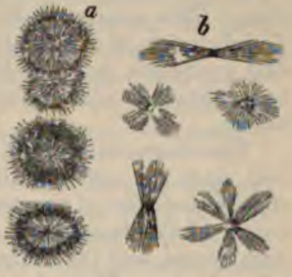

FIG. 63.—*a*. Leucine crystals. *b*. Tyrosine crystals.

The condition of the urine in acute yellow atrophy is very interesting. The amount is either normal or slightly diminished, and the specific gravity is somewhat increased. Often there is a trace of albumen. We have already mentioned the presence of bile-pigment. The point of chief interest, however, is one that Frerichs discovered and various others have since confirmed—namely, the great diminution of urea and the appearance in its place of several other substances, which are likewise the products of the decomposition of albuminoid matter, and represent, in all probability, the first steps in the formation of urea. Of these substances, the most important are leucine and tyrosine. Their characteristic crystals can sometimes be detected by the microscope in the urinary sediment (see Fig. 63). The crystals may also sometimes be obtained by allowing a drop of the fresh urine to evaporate slowly upon an object-glass. The proper way, however, to demonstrate these crystals is to precipitate the urine with basic acetate of lead, to remove the lead from the filtrate by means of sulphuretted hydrogen, and then to evaporate the filtrate thus obtained to the point of crystallization. There are some other abnormal constituents to be found in the urine besides leucine and tyrosine; but what their significance is we do not know. Among these are sarcolactic acid, oxymandelic (paraoxyphenylglycolic) acid, peptonoid substances, and large amounts of kreatine.

It at once suggests itself that this disappearance of urea and appearance of leucine and tyrosine, which are regarded as preparatory stages in the formation of urea, gives valuable support to Meissner's and Von Schröder's idea that this substance is manufactured in the liver.

As to the other organs little need be said. Vomiting is very frequent in the second stage, as well as in the first. It usually ushers in the severe cerebral symptoms. The stools are, as a rule, clay-colored, as is usual in jaundice. There is generally constipation. The pulse is rapid, often reaching 140 to 160 beats per minute, and is also small and compressible. It is this acceleration of the pulse, contrasting with its usual slowness during the first stage, which, along with the vomiting, announces the onset of dangerous symptoms. The pulmonary signs are seldom marked, although there may be bronchitis or a pneumonia due to the inhalation of foreign substances. During the coma which precedes death respiration is usually hurried, and often deep and noisy. Sometimes it is irregular.

The temperature is generally approximately normal. Toward the fatal termination there may be a subnormal temperature. Still more frequently the temperature rises before death, and even sometimes grows higher after death, reaching 107.5° (42° C.) or more.

In case the disease attacks a pregnant woman, abortion or premature delivery is almost certain to occur.

The entire duration of the disease depends mainly upon the length of the first stage. This may be wholly absent, or may be brief, or may occupy several weeks. The duration of the second stage, reckoning from the occurrence of grave cerebral symptoms, is generally only a few days (two to four), rarely a week. The termination is almost invariably fatal. In a few cases recovery has been observed.

The diagnosis can not be made till the second stage. The symptoms of the first stage are indistinguishable from those of simple catarrhal jaundice. With the development of the grave symptoms all doubt usually vanishes. The general course of the disease, the deep jaundice, the cerebral disturbances, the cutaneous ecchymoses, and the character of the urine, form a clinical picture resembling no other disease. The only cases where there can be any uncertainty about the diagnosis are the exceptional ones in which there is no jaundice. It is of practical importance to distinguish this disease from acute phosphorus poisoning (*q. v.*). The differential diagnosis is to be made from the history of the case and from the following factors:

ACUTE YELLOW ATROPHY OF THE LIVER	PHOSPHORUS POISONING
1. Prodromes are usually present, but may be absent.	1. Prodromes are almost always present.
2. Jaundice develops early, sometimes becomes marked.	2. Jaundice does not appear until rather late, and it may be slight to the end.
3. Liver seldom enlarged, becomes atrophic from the start; pain in hepatic region not the rule.	3. Liver much enlarged until death, and very painful.
4. Maniacal delirium very frequent.	4. Seldom marked delirium.
5. Not infrequently fever, sometimes high fever.	5. Not infrequently fever is absent throughout.
6. Leucine and tyrosine often found in the urine.	6. Leucine and tyrosine found in urine exceptionally.
7. Spleen often enlarged.	7. Usually no enlargement of the spleen.

After what has been said, we need hardly add that **treatment** is unavailing. Usually laxatives are employed, particularly calomel. The nervous symptoms are combated by an ice-cap and baths and narcotics; the vomiting, by opium and bits of ice; and the cardiac weakness, by stimulants.

APPENDIX

PERNICIOUS JAUNDICE. CHOLÆMIA AND ACHOLIA

Reference has been repeatedly made in the preceding chapters to the possibility of the sudden supervention of grave nervous derangements in the course of various hepatic diseases. These nervous symptoms resemble one another so much in the different instances of their occurrence that we are forced to believe them always due to the same cause.

These symptoms are relatively most frequent when there is chronic biliary retention. Whether this retention be the result, for example, of obstruction of the common or the hepatic duct, or of stenosis of the biliary passages from a carcinoma involving the opening of the common duct into the duodenum or that duct itself, the patient may quite suddenly fall into a condition which in many respects corresponds to the second stage of acute yellow atrophy just described. Grave cerebral disturbances declare themselves, with delirium, convulsions, and coma. There are hæmorrhages into the skin and into the mucous membranes, and in a few days the patient dies. Usually the end is attended with high fever. We have ourselves seen a temperature of 107.4° (41.9° C.) in a case of cancer at the duodenal extremity of the common duct. It is this group of symptoms which is usually termed pernicious jaundice; but almost precisely similar phenomena may suddenly appear in hepatic cirrhosis, when there is no great degree of jaundice, if any.

Exactly what produces these grave results in acute yellow atrophy and the other disorders just enumerated, we are not certain. In the main three theories have been propounded in explanation. The first theory, the latest champion of which is Leyden, attributes pernicious jaundice to cholæmia—that is, to the accumulation in the blood of the constituents of bile, and in particular of the biliary acids, as a result of absorption. This accumulation, it is said, is promoted by the paralyzing effect of the jaundice upon the activity of the kidneys. Opposed to this theory is the fact that these same symptoms may occur when there is no marked hepatogenous jaundice.

Bouchard and other French physicians ascribe to the liver the function of rendering harmless the products of the decay of albuminous matters developing in the intestine, which may be absorbed and enter the circulation. Destruction of the hepatic cells would consequently lead to auto-intoxication with these intestinal ptomaines. In Germany this doctrine has gained few adherents.

The view which we are most inclined to accept is that of Frerichs. He has given to the group of symptoms under discussion the name of acholia. These symptoms he ascribes to the pernicious influence of those substances which under normal conditions are manufactured by the liver into bile, but which in such cases accumulate in the blood and the tissues. As Frerichs himself has said, and we believe very justly, this view should be extended to include all other metabolic functions of the liver, and especially the production of urea. There is no doubt that the processes of metamorphosis are very active in the liver. That these should be incomplete when there is a severe anatomical lesion of the liver, or when there is persistent biliary obstruction, is very likely, and this limitation of activity might occasion an accumulation of all sorts of material in the blood. We may, therefore, regard acholia, using the word as Frerichs does, as entirely analogous to uræmia, which latter appears not only in diseases of the kidney but also when the ureters are obstructed. In this sense the name cholæmia would be preferable to the term acholia, to represent a condition analogous to uræmia—taking the word cholæmia to mean the intoxication of the blood with all sorts of substances due to the imperfect metamorphosis going on in the liver, and not, as Leyden formerly held, the saturation of the blood with the completely formed components of the bile.

The termination of cholæmia or acholia is almost always unfavorable. In cases of this kind there is usually marked fatty degeneration of the hepatic parenchyma to be found post-mortem, as the anatomical expression of its suspended function.

CHAPTER VIII

ICTERUS NEONATORUM

(Jaundice of the New-born)

FREQUENTLY the normal red color of the skin in children changes on the second, third, or fourth day after birth to a distinctly yellow, jaundiced hue. The yellow tinge is deeper on the face and trunk than on the extremities. There are no special digestive or constitutional disturbances. Still it may be taken for the rule that weaklings and premature infants more often present this jaundice than do vigorous babes. The abnormal hue is almost certain to vanish in a week or two, and leave no sequelæ. The termination is unfavorable in those instances alone where there is some special complication, not directly connected with the jaundice as such.

The ætiology is a disputed matter. A large number of theories of all sorts have been set up, no one of which to this day has gained universal acceptance. Formerly there was considerable tendency to regard the jaundice as hæmatogenous—that is, as due to the transformation of the pigment of broken-down blood-corpuscles into bile-pigment. Points which seemed to support this view are the light (not jaundiced) color of the urine and the yellow color of the stools (from bile). But more accurate examination has shown that the urine does contain biliary pigment, as do also the kidneys of such infants as happen to die during the existence of the jaundice; and the biliary acids have been clearly shown to be present in the serous transudations. It is therefore assumed at present by most authors that icterus neonatorum is hepatogenous; but how the biliary retention and consequent absorption are caused we do not know. Perhaps the bile is not ejected properly, from weakness, or the ducts may be narrow, or temporarily plugged by desquamated epithelium. Birch-Hirschfeld has called attention to the fact that after birth there is a tendency to considerable passive congestion of the liver, with resultant œdema of Glisson's capsule and pressure upon the interlobular bile-ducts. Probably more importance, however, attaches to a circumstance which Hofmeier has noted, that in the first days after birth many red corpuscles are disintegrated, and consequently there is a comparatively large secretion of bile. This might easily lead to congestion of the smallest bile-ducts and so to icterus; but it should be said this view has its opponents. Quincke has an entirely different explanation of *icterus neonatorum*. He goes back to an old theory enunciated by Peter Frank. According to this, the jaundice of the new-born is due to the absorption by the intestine of a large amount of biliary coloring matter from the meconium in the large intestine. Now, if the *ductus venosus* remains open for some little time after birth, as it often does, this biliary pigment is carried not to the liver, but by means of the *vena cava inferior* directly into the systemic circulation. The meconium is rich in bile pigment, and moreover there is an abundant secretion of biliary coloring matter in the new-born, and in them bilirubine is not changed into urobiline in the intestine, and finally the renal secretion of the new-born is at first very scanty. Consequently, the absorption of the bile-pigment from the intestine results in jaundice.

It is well to mention in conclusion that in very rare instances there is complete congenital stenosis, or even entire absence, of the larger bile-ducts. Then deep jaundice comes on soon after birth, and is persistent. The children become extremely emaciated, and, after a few weeks, inevitably perish.

CHAPTER IX

SYPHILIS OF THE LIVER

Ætiology and Pathology.—Syphilitic disease of the liver occurs both when the syphilis is acquired and when it is congenital. Congenital syphilitic disease of the liver may be diffuse or localized, and it causes a cellular infiltration in either case. If the changes are extensive, the organ is hard and considerably enlarged; or, if the newly formed connective tissue has undergone contraction, the liver is smaller than normal, and its surface is uneven. In some cases of hereditary syphilis, distinct gummata of considerable size have been observed.

In acquired syphilis, hepatic disease is one of the so-called tertiary symptoms, and does not usually develop, at least to any great extent, until several years after infection. Indeed, it may be a very late result. It may take the form either of a diffuse syphilitic hepatitis, or of circumscribed gummata or syphilomata. The diffuse hepatatis does not present essentially different anatomical appearances from those of ordinary cirrhosis, although in syphilis the changes are seldom so uniformly distributed throughout the organ. The gummata are the most characteristic, and the most important clinically. They may form separate tumors the size of an apple or even larger. The convex surface of the organ, particularly that portion near the suspensory ligament, seems to be a favorite location for the new growth. The same is true of the porta hepatis, where Glisson's capsule enters the liver. At the autopsy the gummata have in most cases already begun to undergo contraction. If so, the liver is usually smaller than normal, and traversed in various directions by deep furrows, which divide it into lobes. These furrows are due to firm cicatricial bands, among the fibers of which we may sometimes find necrotic and cheesy vestiges of the gumma proper. Often there is evident syphilitic endarteritis in the smaller and sometimes also in the larger branches of the hepatic artery and portal vein.

Clinical History.—Circumscribed syphilitic changes in the liver often give rise to no symptoms whatever. It is only when the disease comes to disturb the portal circulation that a series of symptoms result, which, for evident reasons, may be analogous in all essential points to the effects of ordinary cirrhosis. Whenever the syphilitic growths contract so as to obliterate a large number of branches of the portal vein, or whenever a gumma happens to be so situated as to compress the trunk of the portal vein itself, then the well-known results of portal obstruction are inevitable, the chief being ascites and enlargement of the spleen. The disturbance of circulation often gives rise also to anorexia and digestive disorders. Experience shows jaundice to be rare in hepatic syphilis, but it may appear when the lesions involve the larger bile-ducts or a considerable number of the smaller biliary passages.

On physical examination, the results vary according to the form and the stage of the disorder. Sometimes the larger gummata may be plainly felt through the abdominal walls, usually as flattened hemispheres. Frequently, also, we can feel the edge of the enlarged organ, and can detect that the edge is less sharp than normal. In other instances the separate elevations and prominences can be made out. The area of dullness on percussion of course varies in different cases.

It deserves mention that hepatic syphilis quite often causes severe pain, sometimes over the entire region of the liver and sometimes in just one spot. Pain is by no means felt in every case. With the pain there may be great tenderness on pressure.

The course of the disease is usually tedious, and it may occupy many years. Probably, too, lesions exist in many cases long before there are any symptoms. Just as in cirrhosis, ascites is usually the first thing to attract the patient's attention. Improvement and temporary arrest of the disease are more frequent than in ordinary cirrhosis. Still, in most cases, if the lesions are at all extensive, the termination is unfavorable.

The **diagnosis** is not always easy. Usually the objective changes in the liver, the ascites, and the enlarged spleen, indicate hepatic trouble, but we are often unable to determine just what the trouble is. Naturally, the ætiological factors are of great importance. In a toper we would think rather of the common form of cirrhosis. If there is a syphilitic history, or if we find scars in the throat, irregularities in the surface of the bones, or other signs of a specific dyscrasia, we would naturally ascribe the hepatic disorder to the same cause. As to special signs, if the prominences on the liver are rather large in contrast to the smaller granulations of common cirrhosis, syphilis is somewhat more probable. Severe pain in the right hypochondrium also suggests syphilis rather than cirrhosis. It should also be considered that the course of hepatic syphilis is usually much more protracted than that of ordinary hepatic cirrhosis.

Treatment.—Whether we feel certain that syphilitic hepatitis exists, or merely suspect it, specific treatment should be tried. Mercury and potassic iodide should both be given, but probably the iodide is the more valuable of the two. These remedies can be successful only when the gummata are still in process of formation. Our therapeutic efforts produce no impression upon the cicatricial bands, the contraction of which is the main cause of derangement. Indeed, even the absorption of gummata may not always have a favorable influence upon the patient, because of the mechanical disturbance due to the consequent cicatricial change. Hence, in general, the results of antisyphilitic treatment are not very favorable.

For symptomatic treatment, the reader is referred to the chapter on cirrhosis of the liver.

CHAPTER X

CANCER OF THE LIVER AND BILE-DUCTS

Ætiology and Pathology.—Primary cancer of the liver is very rare, but secondary or metastatic cancer of this organ is met with often. The chief explanation of this latter fact is the slowness of the blood-current in the liver, which favors the deposition of the cancerous germs suspended in the blood.

Secondary hepatic cancer may be a sequel to primary cancer of any organ. It is most often seen, however, when the primary growth lies within the portal system, in the stomach, intestines, rectum, œsophagus, or pancreas. In some instances the projection of the primary growth into the lumen of a branch of the portal vein has been directly demonstrated, thus furnishing an obvious source for metastasis. The secondary cancers in the liver may be very numerous. They are found both within the organ and upon its surface. If superficial, they form flattened protuberances, which are often dimpled in the middle. If the new growth is extensive, the liver may be greatly enlarged, so as to occupy a great part of the abdominal cavity.

As we have said, primary cancer of the liver is very unusual. It may occur either in the form of separate large nodules, or as a more diffuse cancerous infiltration pervading the greater part of the organ. Histologically considered, the primary growths are of cylindrical-cell carcinoma, apparently originating from the epithelium of the minute bile-ducts, but also, according to some authors, sometimes starting from the cells of the parenchyma.

Primary cancer of the larger bile-ducts is of more frequent occurrence than genuine primary hepatic cancer, and therefore it is of more importance clinically. The gall-bladder may also be the seat of primary carcinoma. From these sources may proceed abundant metastatic growths in the liver itself.

As to the ætiology of hepatic cancer we can be brief. The disease is most frequent in advanced life, from forty to sixty, following in this the general rule for cancer. Of special exciting causes, there is only one to be mentioned—that is, biliary calculi. As we have already remarked (see page 551), the formation of cancer subsequent to cholelithiasis has been observed so many times that mere coincidence is out of the question.

Clinical History.—Diagnosis.—Many small nodules of cancer, as well as large masses which are favorably situated, may exist in the liver without exciting any symptoms. If there is an undoubted primary cancer in another organ, such as the stomach, we must always remember the possibility of metastatic growths in the liver; but they can not be proved to exist, unless they alter appreciably the size or shape of the organ. Sometimes their existence may be inferred, when we observe either ascites and enlargement of the spleen from pressure on the portal vein, or jaundice from pressure on the bile-ducts.

On palpation, we are often able to make out plainly one or more tumors in hepatic cancer. These tumors are in the region of the liver, and they are directly connected with it, as we can prove by marking out its limits by percussion and palpation. Another characterstic sign is that almost all hepatic tumors can be felt to move with respiration, on account of the inspiratory depression of the diaphragm pushing down the liver and all that is joined to it. Percussion over a hepatic tumor almost invariably gives flatness, in contrast to the muffled tympanitic resonance of many gastric tumors.

The most characteristic condition is not a very rare one; in it the liver is the seat of a very large number of cancerous nodules. In such cases the organ is usually much enlarged. Often we can detect by mere inspection a great, irregular prominence in the hepatic region, pressing forward the flabby, atrophied walls of the abdomen, reaching down to the level of the umbilicus, or even lower, and moving with respiration. On palpation, we can feel most of the anterior surface of the liver, and the separate cancerous nodules scattered over it. These usually are as large as walnuts, or even apples, and they are often umbilicated. The lower or anterior margin of the liver can often be made out plainly, and it also is often the seat of nodules; and we can sometimes feel nodules on the lower surface of the organ.

The other clinical phenomena in hepatic cancer have several causes: (1) The primary disease, such as cancer of the stomach; (2) the general cancerous cachexia, as shown by languor, emaciation, and possibly a slight œdema of the ankles; and (3) the possible compression of the blood-vessels or bile-ducts. This compression is not infrequent, and produces a moderate or even a large ascitic effusion. Even in these instances the spleen is seldom much enlarged as a result of the passive congestion, because the universal emaciation and anæmia counteract the tendency to increase in size. Jaundice is relatively more frequent in cancer of the liver than is ascites. It is caused by compression either of the hepatic duct or of the minuter bile-ducts. On the other hand, however, we can easily see that hepatic cancer may exist without producing either icterus or ascites.

The differential diagnosis of hepatic cancer from cancer in other organs is sometimes extremely difficult. This is particularly true of pyloric cancer, and especially so when the pylorus has become adherent to the liver, which often is the case. Cancers of the omentum and of the colon sometimes simulate hepatic cancer, but they seldom move so decidedly in respiration as do hepatic tumors. Given a new growth in the liver, it is usually comparatively easy to distinguish between carcinoma and other tumors. In case of syphilitic new growths, we have to consider the previous history and other signs of syphilis, also the eventual contractions and diminution in size of the liver, the usually longer duration of the disease, and the general condition of the patient. Echinococci have, as a rule, a much more regular shape, like a flattened ball; they seldom occasion marked cachexia, and also have a much longer clinical history. Large abscesses are very rare in our climate and they can usually be recognized by special objective conditions. Moreover, they cause fever (rigors), which is exceptional in carcinoma. Other primary tumors of the liver, such as sarcoma and adenoma, are very rare, and therefore of no great clinical importance. There is, however, one rare but important tumor of the liver which we must mention—viz., melanotic sarcoma, which may appear as a secondary growth in the liver. It may be of enormous extent, either diffuse or nodular, and secondary to melanotic sarcoma of the choroid or of the skin.

When we have decided that cancer of the liver is present, the question arises, Is it primary or secondary? In the first place, primary cancer here is so rare that the probabilities are in favor of a secondary growth. Not infrequently the primary tumor can not be detected during life. Thus a small cancer of the stomach, or a flat œsophageal cancer, or carcinoma of the pancreas, are all easily overlooked. Cancer in the rectum may be detected by digital examination. If we find many nodules in the liver and no primary trouble elsewhere, then there comes the possibility that there is primary cancer in the gall-bladder or the bile-ducts. In rare instances palpation may discover the gall-bladder in a state of cancerous degeneration close underneath the liver; but usually the viscus will be small and contracted, and the flat and ulcerating growth is not noticeable, except from the inside. It is particularly in those cases of hepatic cancer in which there is at an early period great and persistent icterus, without evidence of carcinoma in any other organ, that we should be most apt to think of primary cancer of the bile-ducts.

The duration of hepatic cancer is usually not prolonged. The first decided evidences of its existence no sooner present themselves than marasmus and cachexia rapidly develop. The fatal end comes in a few months, or at latest within a year.

The prognosis is hopeless. Treatment can avail only to alleviate somewhat the patient's sufferings.

CHAPTER XI

ECHINOCOCCUS OF THE LIVER

Natural History and Pathology.—Inasmuch as it is the liver which suffers most frequently from invasions of the echinococcus, we will here state the main general points relative to the troubles produced by this parasite.

The *tænia echinococcus* (see Fig. 64) is a small tape-worm about four millimetres long, and composed of four joints. It inhabits the intestinal canal of the dog. The head is small and bears a beak which is provided with some thirty to forty hooklets, arranged in two rows. Behind the circle of hooklets are four suckers. The last joint of the tænia is by far the largest, and contains some five hundred eggs. Man is infected by ingestion of the eggs. The great frequency of the echinococcus in Iceland is explained by the close intimacy of the Icelanders with their dogs. In Germany the echinococcus is most frequent in Mecklenburg and Pomerania.

If a human being has become infected, the embryo pierces the intestinal wall, and is carried with the blood-current or the lymph-current to some remote organ. Most of the eggs no doubt are destroyed in the stomach, as may be inferred from the fact that multiple echinococci are much less frequent than solitary individuals. In a great majority of cases the embryo passes through a branch of the portal vein into the liver and there fastens itself; but the echinococcus may be developed in other organs—including the lungs (*vide* page 278), the bones, the brain, the kidneys, and the heart. A hydatid cyst develops from the embryo, and is filled with a non-albuminous fluid. The cyst is composed of an external cuticle of laminated structure, and an inner, parenchymatous layer, which contains muscular fibers and blood-vessels. Surrounding the cyst, as it lies in the infested organ, there is gradually developed a thick capsule of connective tissue.

After the cyst has continued its growth for some four to six months, being now about the size of a walnut, there are generated upon the inner surface of the capsule, from the parenchymatous layer, so-called breeding capsules, containing numbers of echinococcus-heads, or "*scolices.*" Each scolex has four suckers and a circlet of hooks. It can draw itself into the breeding capsule and also produce a prominence upon the outer surface of the latter (see Figs. 65, 66, and 67). The number of new-formed scolices in an echinococcus is very great. The sac grows very slowly up to about the size of a fist, or even larger; its wall has a pale, yellow color, is transparent, and displays an extremely characteristic laminated structure. The firmly attached breeding capsules are visible from the outside through the walls, as minute points.

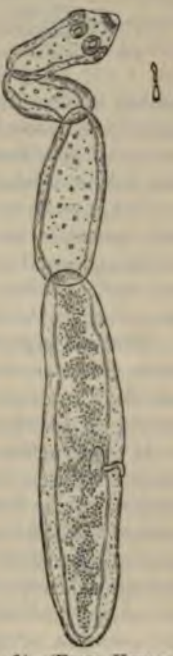

Fig. 64.—(From Heller.) Tænia echinococcus, enlarged. Above, at the right, echinococcus, of natural size.

The primary cyst often gives rise to secondary "daughter vesicles," and these to "granddaughter vesicles." Some of these are formed in the cuticle, others from the breeding capsules. In man they generally grow inward—that is, are endogenous (*echinococcus hydatidosus*)—and finally become detached. Hundreds of them may sometimes be found free in the liquid contents of the cyst. In animals the daughter vesicles are more often exogenous (*echinococcus veteri-*

norum seu granulosus). A peculiar kind of echinococcus, which was formerly regarded as a kind of new growth, is that called by Virchow *echinocoocus multilocularis*. This is a resistant tumor containing much connective tissue, and permeated with numerous small alveoli. The gelatinous contents of the alveoli consist of echinococcus vesicles. The growth probably originates from a single germ, which by persistent exogenous proliferation invades the lymph-vessels (perhaps also the blood-vessels and the bile-ducts).

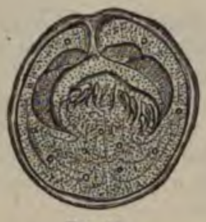

FIG. 65.

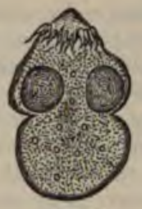

FIG. 66.

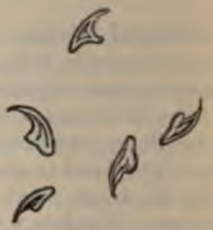

FIG. 67.

FIGS. 65 and 66.—(From HELLER.) Echinococcus scolices, free, drawn in and turned outward.
FIG. 67.—Echinococcus hooklets.

The growth of a hydatid cyst is very slow, and it may continue for years. At last, however, the echinococcus dies. The cyst then undergoes considerable though gradual contraction, and both walls and contents become fatty-degenerated and calcified. In the mortar-like detritus it is usually possible to find some characteristic hooklets.

Clinical Symptoms.—As long as the cyst in the liver retains moderate dimensions, there is usually no discomfort. Frequently the cysts perish and become calcified, without having ever attracted attention, and are found post-mortem merely by accident.

If the cyst becomes very large, it causes a sensation of pressure and pain in the hepatic region. In rare instances, unusually large cysts, if situated on the convex surface of the liver, may crowd up the diaphragm so as to compress the lower portions of the lung and induce dyspnœa. In such cases we find on the anterior or posterior wall of the thorax an abnormal protuberance, with dullness and diminished respiratory murmur. If a large echinococcus grows downward into the abdominal cavity it occasions pressure, tension, and pain in the abdomen. Compression of the portal vein may lead to ascites, and compression of the bile duct, in rare cases, to jaundice. The tumor can usually be made out with ease by inspection and palpation. It is approximately hemispherical, smooth, not tender, and feels rather firm and elastic. There is often a sense of fluctuation, while the so-called hydatid thrill—to be elicited by quick pushes with the flat of the hand—is recognized distinctly in but few cases.

The condition may be complicated (1) by rupture of the echinococcus-sac into neighboring parts, and (2) by secondary suppuration. Both occurrences may be apparently spontaneous, or the result of traumatic influences.

Of the possible directions in which rupture may take place, penetration of the pleural cavity should be particularly mentioned. It usually results in an acute empyema. Here we have to do partly with secondary infection occasioned by pyogenic bacteria, and partly also, as it seems, with the influence of toxic substances (toxalbumens) which are present in the echinococcus fluid (*vide infra*). Penetration into the lungs with rupture of the echinococcus-sac leads to severe pulmonary symptoms. The patient may cough up constitutents of the bile and small echinococcus vesicles, or portions of the same. Perforation into the abdominal cavity occasions purulent peritonitis. If viable daughter-vesicles are set free in the abdominal cavity, they may settle on some fresh spot and continue to grow. As extreme exceptions are to be mentioned rupture of the echinococcus-sac into the pericardium, the bile-ducts, the intestine, the *vena cava*, and the pelvis of the kidney.

Suppuration of an echinococcus in the liver leads to the clinical symptoms of hepatic abscess—increasing tenderness and fluctuation of the tumor, fever, aggravation of the constitutional symptoms, etc. Perforation of the abscess into neighboring structures may occur, in the same way as above mentioned. Spontaneous recovery is possible but infrequent. In general, suppuration of an echinococcus-sac is a dangerous condition, unless there is surgical interference.

We must devote a few words to *echinococcus multilocularis* of the liver. As has been said, the tumor in this case is large, very firm and hard, and with a surface which is either smooth or rough. Tenderness on pressure is exceptional. On the other hand there is a feeling of painful distention in the hepatic region. Comparatively often (H. Vierordt) there is jaundice, which may be extreme. The spleen is usually enlarged. The bodily condition is fairly good for a long while, but later marasmus develops, particularly if ascites or œdema occurs. Secondary suppuration in the interior of the tumor also leads to fever and rapid loss of strength. The disease lasts one to two years, sometimes much longer.

Diagnosis.—The diagnosis of echinococcus of the liver depends chiefly upon the demonstration of a very slowly developing tumor in the liver, hemispherical, fluctuating, and painless, with no marked influence at first upon the general health. If the patient comes from a country where the echinococcus is particularly frequent, of course this renders the diagnosis easier. Formerly it was the custom to aspirate the tumor for diagnostic purposes. In this way we can, in many cases, obtain from the interior of the sac a light yellow non-albuminous fluid, which often contains sugar and succinic acid; and in it sometimes, but of course not invariably, are found upon microscopic examination the characteristic hooklets, or little pieces of the laminated membrane. Lately, however, physicians have become much more cautious about puncturing echinococcus-cysts, because the procedure is sometimes followed by symptoms which are probably due to the poisonous influence of the escaping contents of the cyst (*vide supra*). Not infrequently after puncture there occurs urticaria or severe toxic symptoms, such as collapse, vomiting, dyspnœa, diarrhœa, chills, and epileptiform attacks; and these may even prove quickly fatal, although occurrences of this sort are infrequent. They have brought great discredit upon the practice of aspiration for diagnostic purposes in case of echinococcus.

The diagnosis of the multilocular echinococcus of the liver is always very difficult. If made at all, it demands a careful consideration of all the separate symptoms, and particularly of the general course of the disease, in order to distinguish the condition from hepatic carcinoma, cirrhosis, or syphilis.

Treatment.—Inasmuch as internal remedies, such as iodine and mercury, are absolutely useless, the only treatment of echinococcus is by surgical means. In case the echinococcus tumor causes little if any discomfort, the patient is usually loath to consent to an operation, but it should be recommended in all cases, for it protects the patient from the possibility of later dangerous developments, and in itself is no grave matter. A description of the many methods of surgical treatment will be found in the text-books on surgery.

CHAPTER XII

CIRCULATORY DISTURBANCES IN THE LIVER

1. Hepatic anæmia is seldom extreme except in cases of profound gener anæmia, and it has no clinical importance, so far as we are aware.

2. Passive congestion of the liver is of frequent occurrence and is of impo tance. It may arise in any disorder which disturbs the systemic circulation. is oftenest seen in connection with heart disease, particularly mitral disease. also follows pulmonary emphysema and chronic processes which result in co traction of the lungs. The liver is enlarged and engorged. The hepatic vei being situated in the center of the lobules, this central portion becomes dark pigmented, while the periphery of the lobules seems lighter colored. The peripheral cells may even appear distinctly yellow, from a fatty infiltration which is not infrequent. In this way the cut surface comes to present that variegated appearance which has led to the name of "nutmeg liver." If the venous stasis be persistent, there is considerable atrophy of the hepatic parenchyma, involving especially the cells near the center of each lobule. Thus the liver is somewhat reduced in size in spite of the secondary increase of connective tissue, and its surface may become slightly granular. This is the "atrophic nutmeg liver" or "contracted liver due to passive congestion."

The clinical phenomena are chiefly those caused by the hepatic enlargement. If chronic cardiac disease, emphysema, or some analogous trouble has occasioned congestion of the liver, the area of hepatic dullness is increased, and frequent we can feel the edge or even a portion of the anterior surface. In well-develop cases the liver reaches a hand's-breadth or more below the ribs on the right side. If there is also tricuspid regurgitation (*q. v.*), the liver is usually much e larged and can be felt to pulsate distinctly if the palm of the hand is laid upon Not infrequently in passive congestion of the liver there is jaundice, which m be slight or quite well-marked. This is perhaps due to the pressure exerted by t distended blood-vessels upon the small bile-ducts in the liver, and perhaps also the secretion of a particularly viscid bile containing less water than normal, a tending to stagnate. As we have already mentioned, the peculiar mixture jaundice and cyanosis in the complexion of many cardiac patients is very ch acteristic. The secondary cirrhosis of the congested liver leads to ascites. may suspect this condition of the liver in all cases of heart disease in whi ascites is particularly well marked, in comparison with the slight œdema in oth portions of the body.

Quite often the congestion, if great, produces subjective disturbances. The is a feeling of pressure and weight in the hepatic region; and if the capsule of t organ is tightly stretched, there may be actual pain.

The prognosis and treatment depend, of course, upon the primary disorder.

3. About active hyperæmia of the liver we have little definite informatic Formerly there was a great deal said about it, as one of the conditions in " dominal plethora." Active hyperæmia is most frequently assumed to exist case of those who are good livers and of sedentary habit. In such, we are to the temporary physiological hyperæmia which attends digestion passes on int permanent congestion of the liver. Thereby the organ is enlarged, there are pa ful sensations in the right hypochondrium, digestive disturbances, and occasio slight jaundice. The abnormal condition just described is certainly often with in practice, but it would seem hardly possible to draw a clear dividing-

between active hyperæmia of the liver and other disturbances which give rise to similar symptoms. Such are chronic gastric and intestinal catarrhs; cardiac hypertrophy and functional cardiac derangement, with passive congestion of the liver; fatty liver; and incipient cirrhosis.

A prominent factor in the production of active hyperæmia of the liver is also ascribed to the ingestion of such matters as are said to "irritate" the liver, like the various spices, coffee, and, above all, alcohol.

It should also be noted that the liver may be much engorged in many acute infectious diseases, particularly in pernicious malarial diseases and in typhus or typhoid fever.

It is also maintained that the hyperæmia may result from the cessation of hæmorrhages elsewhere, such as the catamenia or bleeding from hæmorrhoids. The facts that have been brought forward to sustain this view are none of them conclusive. We will mention that the "menstrual jaundice" which occasionally appears when the menses are scanty or absent has been referred to a vicarious hyperæmia of the liver.

It is, of course, impossible to make general statements about the course and duration of active hyperæmia of the liver. The treatment of the first variety mentioned—namely, that arising from an improper mode of life—demands careful regulation of the diet, abundant exercise in the open air, such as horseback-riding, and laxatives. We may order rhubarb, aloes, or a course of the waters at Carlsbad, Marienbad, Kissingen, or Homburg.

CHAPTER XIII

ATROPHY, HYPERTROPHY, AND DEGENERATIONS OF THE LIVER

1. **Simple Atrophy of the Liver.**—Simple atrophy is not of rare occurrence, being seen in senile marasmus, and in malnutrition from almost any cause. The degree of atrophy varies. The borders of the organ are much wrinkled. The lobules seem decidedly smaller than normal, and even the individual cells that still remain are atrophied and also usually deeply pigmented.

The condition does not of itself give rise to any special symptoms. The area of hepatic dullness is usually lessened, but this sign is too ambiguous ever to justify us in making from it a diagnosis of hepatic atrophy. Perhaps there is some value in the alleged lighter color of the stools, as indicating a diminished secretion of bile.

2. **Hypertrophy of the Liver.**—Even under normal circumstances the liver undergoes quite marked alterations in size. The exact point, therefore, where an abnormal hypertrophy begins can not be set. Sometimes the autopsy reveals an unusually large liver, of which there had been no indications during life, and for which no cause can be made out.

There are certain diseases in which enlargement of the liver is found with comparative frequency: diabetes mellitus, chronic malarial poisoning, leukæmia, and sometimes rachitis. Topers quite often have enlarged livers, which as a rule present simple hypertrophic changes. Occasionally a liver has been reported as showing spots of localized hyperplasia, which may form flattened prominences upon the surface of the organ.

Hypertrophy is to be diagnosticated only when palpation and percussion give proof of an enlargement, and yet amyloid, hypertrophic cirrhosis, and other diseases which cause an increase in the size of the liver, can be excluded. The ætiology of the case should also be considered.

3. **Fatty Liver.**—This name is applied to excessive, diffuse, fatty infiltration of the hepatic cells. The size of the organ is increased. It is firm, anæmic, and of a uniform yellow color, both externally and upon section. The microscope shows that the cells of the parenchyma are filled with large and small globules of fat. The fat is most abundant toward the periphery of the lobules.

The causes of fatty liver are by no means clear. Sometimes it is found in cases of general obesity, where we may assume that the amount of fat which the liver receives as nourishment is abnormally great; but often we find a liver that contains comparatively little fat in those who have a well-developed panniculus adiposus and much fat in other organs. Topers may have a decidedly fatty liver. The occurrence of fatty liver in the cachectic, and particularly in the consumptive, is remarkable; and individuals suffering from cancer, or marantic children, may also exhibit the same change. We have no intimate knowledge of the conditions that prevent, in such cases, the oxygenation of the fat which comes to the liver from the ingesta or from other organs.

We do not know that the fatty liver is in any way functionally impaired. The only clinical indication, therefore, of its existence is the increased bulk of the organ. In phthisis we may sometimes feel pretty certain that the liver is fatty, if an increase in bulk can be demonstrated, and if other causes for this enlargement, such as amyloid degeneration, appear improbable. If the anterior edge of a fatty liver can be felt, it is usually found to be noticeably thick and blunt.

The treatment of fatty liver is to combat the original disease.

4. **Amyloid Liver** (*Waxy Liver*).—Amyloid degeneration of the liver is almost invariably a part of extensive amyloid disease, involving also the spleen, kidneys, intestine, and other organs. The disease occurs chiefly in certain cachectic conditions, such as chronic suppuration, as in caries and persistent empyema, and also in chronic pulmonary tuberculosis, and constitutional syphilis.

The amyloid liver is usually increased in bulk. The organ may even become almost double its normal size. It feels very firm and hard, its surface is perfectly smooth, and its edge is slightly thickened. The cut surface presents a characteristic grayish-brown "waxy" appearance.

The microscope shows that the degenerative process attacks chiefly the walls of the hepatic capillaries, the hepatic cells proper showing infrequent and slight amyloid changes. Very often the cells of the parenchyma are atrophied and somewhat infiltrated with fat.

The diagnosis of amyloid liver requires (1) the demonstration by palpation and percussion of hepatic enlargement. We can often feel a large part of the anterior surface and the margin of the hard and firm organ. The liver may reach as low as the level of the umbilicus. The diagnosis further demands (2) that some disease which predisposes to amyloid be present, and (3) that there be evidence of the degenerative process in other organs: the spleen should be enlarged, and the kidneys secrete an abundance of albuminous urine.

The other symptoms, as well as the prognosis and treatment, are determined mainly by the nature of the causative affection. The use of iodide of iron, iodide of potassium, the alkaline carbonates, and ammonic chloride, respectively, has been recommended as particularly efficient, but therapeutic claims of this sort do not withstand a rigorous criticism. The chief point is the general improvement of nutrition by careful diet and nursing. Further particulars about amyloid disease in general will be found in the chapter on amyloid degeneration of the kidney.

CHAPTER XIV

ANOMALIES IN THE SHAPE AND POSITION OF THE LIVER

1. **Corset Liver.**—The constant pressure of the lower ribs against the liver, as a result of tight lacing, often produces an atrophy of the hepatic parenchyma from pressure, as shown by a deep furrow crossing transversely the anterior surface of the organ. This "corset furrow" lies chiefly in the right lobe. Its usual situation corresponds to the margin of the ribs, and the atrophy may be so extreme that the liver is divided into a large upper part and a small, usually roundish, lower portion, connected by a narrow isthmus of tissue. At the atrophic place, the connective-tissue capsule of the liver is almost always much thickened. Often the lower section can be bent upward as if attached by a hinge.

This deformity of the liver is found quite often in elderly females, and rarely in men, as in soldiers. Unless extreme, it can not be detected during life, and it causes no discomfort. Even the bad cases do not, as a rule, occasion any special symptoms; but they can be clearly made out if the abdominal walls are lax. The deep transverse furrow can be felt, and also the lower section, with its usually blunt edge. Particularly in the case of old women we must bear this condition in mind, else we might easily confound it with some enlargement of the liver, such as amyloid or passive congestion, or even new growths.

In some few cases a well-marked corset liver seems to cause especial symptoms. It occasions a constant feeling of pressure and tugging in the region of the liver. Sometimes there are attacks of violent pain, with peritoneal irritation and vomiting, and a mild degree of collapse. These have been regarded as the result of temporary congestion and swelling in the portion of the liver below the constriction; but we should view this explanation with reserve, and should bear in mind that in corset liver gall-stones are remarkably frequent (*vide supra*, page 544), and that consequently the symptoms mentioned may also be connected with cholelithiasis. The treatment of such attacks, even when the diagnosis is doubtful, will in every case consist chiefly in absolute rest in bed, the employment of poultices or perhaps of an ice-bag, and a restricted diet. If there is violent pain, we must resort to narcotics.

2. **Movable Liver.**—This name has been applied to a condition exceptionally seen in women with very lax abdominal walls, in which the liver sinks deep down into the lower portions of the abdominal cavity, probably as a result of an unnatural length of the suspensory ligament. The organ can be distinctly felt in its new situation, and can usually be brought back to a normal position with tolerable ease by means of external pressure. It is invariably very movable and it can be seen to change its place when the patient lies upon the side. In most cases there are at the same time the indications of universal enteroptosis, with which we have already become acquainted (see page 462). The interpretation of the symptoms present, such as pain and digestive disturbance, is consequently difficult in most cases, especially if the patient is also neurasthenic and hysterical, which is not infrequently the case. The reader may compare on this point the chapter on movable kidney. Treatment is governed by the same principles as in the other forms of enteroptosis. Above all, trial should be made of a suitably applied and well-fitting bandage.

CHAPTER XV

SUPPURATIVE PYLEPHLEBITIS

(Purulent Inflammation of the Portal Vein and its Branches)

Ætiology.—Purulent pylephlebitis is seldom a primary, idiopathic disease. In most instances it is due to the propagation of a suppurative inflammation of neighboring tissues to the walls of the vein. The main trunk of the portal vein is rarely directly attacked. Usually the process originates in the hepatic branches of the vein or in the veins of the portal system, and thence extends to the larger vessel.

Perityphlitic abscess is the most frequent source of suppurative pylephlebitis. The inflammation involves a mesenteric vein, and thence extends upward. Other causes are gastric ulcer, intestinal ulcers, as in dysentery, splenic abscess, and purulent inflammation at the porta hepatis or within the liver itself, as in abscess due to gall-stones. The mode of production in these cases is precisely analogous to that in perityphlitic abscess; but they are rare.

A special form of pylephlebitis is observed in the new-born. Here the inflammation originates in the umbilical vein, and we need hardly say that the cause is a suppurative infection through the navel.

In rare instances it has been found that pylephlebitis has resulted from the penetration into a vein of some foreign body that had been swallowed, such as a pin. Here, too, the true factors in producing the inflammation are, of course, the bacteria which adhere to the foreign body.

Pathology.—Where the inflammation has attacked the vascular walls, the vein is thickened, and often the surrounding connective tissue is infiltrated with pus-cells and mottled with minute ecchymoses. If the vein is cut open, the intima is seen to be opaque and often superficially ulcerated. The lumen of the vessel is filled with a thrombus, which is usually to a great extent in a state of purulent softening, so that offensive purulent fluid flows out. The course of events is as follows: First, the wall of the vein becomes inflamed. As a consequence of this, a thrombus forms at the same place. The bacteria penetrate this thrombus and occasion its purulent softening.

The extent of a pylephlebitis naturally varies in different cases. As a rule, little fragments become detached from the thrombus and enter the liver, producing metastatic abscesses. Secondary suppuration may occur also in the lungs, kidneys, brain, and joints, so that we have all the anatomical characteristics of a general pyæmia.

Clinical History.—Inasmuch as the primary, causative disease may be very different in different cases, it is impossible to delineate the disease comprehensively. It is, however, frequently ushered in by a number of symptoms, which render a diagnosis possible, at least in some cases, if the original disease has been recognized.

The symptoms of suppurative pylephlebitis are in part due directly to the local disease itself, and in part are occasioned by the general pyæmia. One of the local symptoms is pain in the epigastrium. This is rare. It may radiate downward or laterally, according to the starting-place and extent of the inflammation. An inevitable result of the portal thrombosis is portal obstruction. The spleen becomes considerably swollen, and, if the disease be not too quickly fatal, there is an evident effusion into the peritoneal cavity. The splenic enlargement can not be regarded as due merely to venous stasis, but is in part the "acute splenic tumor" of constitutional septic conditions. If the inflammation spreads from the branches of the portal vein to the neighboring bile-ducts, jaundice results.

This is seen quite often. Sometimes it is due also to the hepatic abscesses, or to a gall-stone which happens to cause trouble simultaneously. Now and then there is no jaundice whatever.

Of the pyæmic symptoms, hepatic abscesses come first. They are due, as we have said, to the conveyance of infectious matter directly into the liver by emboli. The one almost constant sign of their occurrence is a decided enlargement of the liver. When there are no hepatic abscesses, the organ usually retains its normal bulk.

The course of the fever is very characteristic. As in other pyæmic conditions, there are almost invariably great elevations, to 106° (41° C.), or higher, accompanied by rigors, and followed by marked remissions, with profuse perspiration. These onsets of fever occur at irregular intervals, either daily, or every two or three days.

There are at the same time indications of constitutional septic infection, which keep increasing in severity. The pulse grows rapid and small. Intelligence is impaired. Somnolence and delirium come on, and the strength rapidly fails.

There are other symptoms. Vomiting is frequently seen. The bowels are seldom constipated, but usually relaxed. The dejections may contain blood, because of the venous stasis. In some cases the inflammation extends so as to produce a fatal general peritonitis. It is noticeable that the urine is generally scanty, and the amount of urea is strikingly diminished.

The disease usually runs a rather acute course. On the average, it lasts about two weeks, but it may occupy three or four weeks, or even a longer period. It is invariably fatal. At least, no cases of recovery are known.

The diagnosis can sometimes be made with considerable positiveness. In other instances it is impossible to exclude other pyæmic conditions, or abscess due to gall-stones, etc. Important factors are the origin of the trouble—if it can be made out—the pyæmic rigors, the enlargement of the spleen and liver, jaundice, epigastric pain, and the evidences of general sepsis.

Treatment is unfortunately almost entirely useless. The fever is not affected even by large doses of quinine. All we can aim at is to support and relieve the sufferer as far as possible.

CHAPTER XVI

THROMBOSIS OF THE PORTAL VEIN

(Chronic Adhesive Pylephlebitis. Pylethrombosis)

Ætiology and Pathology.—Like suppurative pylephlebitis, chronic portal thrombosis is not an independent disease, but is the sequel of a great variety of pathological conditions. Marantic thrombosis is of rare occurrence, and is usually formed toward the close of life, so as not to be of practical interest. Apart from this, almost all cases of thrombosis of the portal vein are due to a compression and constriction of the trunk of that vessel or one of its main branches. This most often occurs in certain chronic hepatic diseases which involve a mechanical stenosis, either of the smaller branches of the portal vein within the liver, or of the vein itself, with resulting coagulation of the blood within it. Chief among these diseases are cirrhosis and syphilis of the liver, which have repeatedly been observed to entail portal thrombosis; but other diseases in the neighborhood of the vein may produce a similar effect. New growths of various kinds may press upon the vessel, or chronic inflammatory hyperplasia of the connective tissue at the porta hepatis may act in the same way. This is illustrated in chronic peritonitis, whether circumscribed or diffuse, an example of the former being sometimes seen as an effect of duodenal ulcer.

It was formerly held that many forms of so-called "lobulated liver" were due to a primary adhesive pylephlebitis. This is erroneous. These cases are probably all due to some primary hepatic disease, usually syphilitic. The size of the liver is little influenced by obstruction of the portal vein, even if long continued, for the hepatic artery suffices to supply all the blood required by the organ.

The anatomical changes in pylethrombosis do not differ essentially from those seen in thrombosis of any other vein. If fresh, the thrombus is still red; later it grows harder, paler, and more friable. If the thrombosis has existed a long while, the clot becomes completely organized. We have observed this even in the main trunk of the portal vein.

Clinical History.—The symptoms of portal thrombosis are those occasioned by the obstruction, and therefore such as we have already repeatedly met with, in connection with various hepatic diseases. The intensity and extent of these results, as well as the time occupied in their development, depend, of course, upon the place and size of the clot. If it is the portal vein itself which is attacked, and if the thrombus is extensive enough to obstruct the flow of blood, then the signs of venous stasis are evident throughout the portal system. The spleen becomes much enlarged, as can be easily demonstrated by percussion and palpation. Soon ascites appears, as a result of the passive congestion of the peritoneal veins; and from a similar condition of the gastro-intestinal veins arise catarrhal disorders, such as diarrhœa; or, not so very exceptionally, there is repeated gastric and intestinal hæmorrhage.

As we have seen, a collateral circulation may be developed (*vide* page 559), by which the venous blood of the portal system is enabled to reach the systemic veins. This explains why some of the symptoms of venous stasis may temporarily (perhaps permanently) vanish. We saw one case of portal thrombosis, the sequel to what was apparently a syphilitic disease of the liver, in which quite a large ascitic effusion appeared some six or seven times at intervals of three to six months, and under proper nursing and treatment, without aspiration, as often disappeared. The patient did not die till the illness had continued six years, and tapping had been required in all some fifteen times. At the autopsy the trunk of the portal vein was found to be converted into a fibrous cord, with a lumen which barely admitted a knitting-needle.

In simple pylethrombosis there are no local symptoms such as pain. The condition of the liver depends upon the primary disease. It is possible that a moderate atrophy of the entire organ might at length ensue if the portal blood were permanently cut off from it. But, as we have said, any cirrhotic changes, or any "lobulation," are not to be regarded as the result, but as the cause of the thrombosis, or at least as related to the cause.

The course and duration of the disease are according to the nature of the original, causative trouble. No general statements can be made.

The diagnosis of thrombosis of the portal vein is usually extremely difficult, and it can really hardly ever be made with absolute certainty. We may, indeed, recognize readily that there is some decided obstruction to the portal circulation; but whether this be due to a thrombus, or to compression of the portal vein, or to the obliteration of a large number of the smaller branches of that vein within the liver, we can very seldom determine. Pylethrombosis may be regarded as probable, if no other possible cause of the portal obstruction seems likely, and if we are able to discover a cause for thrombosis.

The prognosis is always unfavorable, although there may be, as we have said, great temporary improvement. Treatment must be symptomatic, and it follows in the main the principles set forth under cirrhosis of the liver.

APPENDIX

DISEASES OF THE PANCREAS

The few facts of clinical importance that are known about the pathology of the pancreas are given below.

1. **Hæmorrhages into the Pancreas.**—Small hæmorrhages occur in the pancreas in cases of general hæmorrhagic diathesis, marked passive congestion, severe acute constitutional infection, and trauma of the abdomen. These do not, as a rule, cause marked clinical symptoms. On the other hand, Klebs, Zenker, and others describe cases in which a more or less extensive pancreatic hæmorrhage has been found at autopsy as the only demonstrable cause of death. The patients were previously in apparently perfect health and were vigorous, although usually corpulent individuals; and they died suddenly. Perhaps it was the influence of the hæmorrhage upon the semilunar ganglia or the solar plexus which occasioned the speedy death. At any rate, we find in such cases the cavities of the heart of usual size and empty, while the abdominal vessels are distended with blood. The special causes which lead to the hæmorrhage are not aways clear. In general, we probably must surmise a primary disease of the blood-vessels (syphilis, atheroma). It is noteworthy that a comparatively large number of cases have occurred in hard drinkers. Many cases also seem to be similar to those first described by Balser. These have an uncertain ætiology, perhaps infectious, and exhibit a multiple necrosis of fat and of the pancreatic tissue—there being numerous minute necrotic foci in the pancreas and in the fat tissue of the mesentery. Extensive disorder of this kind is observed particularly in obese persons, but is not confined to them. Sometimes in cases of pancreatic hæmorrhage death is extremely sudden and apoplectiform. In other cases the symptoms last from twelve to twenty-four hours before the fatal termination. The patient suffers from violent abdominal pain, vomiting, abdominal distention, and general collapse; treatment is purely symptomatic.

2. **Atrophy of the Pancreas.**—The organ may share in a general marasmus. There is sometimes also extreme atrophy of the pancreas in those who have died of diabetes mellitus (*q. v.*).

3. **Pancreatitis.**—A few cases have been reported of what would seem to be a primary acute pancreatitis, usually hæmorrhagic. The disease is certainly very rare. It begins with violent colicky pains in the epigastrium. Vomiting and collapse soon follow. The pulse grows small, the extremities become cool, and death is speedy. At the autopsy the pancreas is found to be much enlarged, and mottled with ecchymoses, or it even presents scattered foci of suppuration. The ætiology is unknown. It is most likely that the germs which excite the inflammation originate in the intestines. Apparently there should be a sharp distinction made between primary pancreatic hæmorrhage and acute hæmorrhagic pancreatitis, but formerly these two conditions seem often to have been confounded. Secondary abscesses of the pancreas are not infrequent in pyæmia.

[Fitz, in the Middleton-Goldsmith lecture for 1889, threw much light on the ætiology and diagnosis of acute pancreatitis, which he subdivides into three anatomical forms—the hæmorrhagic, the suppurative, and the gangrenous. He shows that the affection is not so rare as has been supposed; that its victims are usually in middle life, fat, and good livers; that it commonly originates by the extension of a gastro-duodenal inflammation along the pancreatic duct.

"If the case does not end fatally in the course of a few days, recovery is possible, or a recurrence of the symptoms in a milder form takes place, and the characteristics of a subacute peritonitis are developed."

In the differential diagnosis irritant poisoning, perforation of the digestive or biliary tracts, and acute intestinal obstruction are to be considered. The location of peritonitic symptoms, their suddenness of onset, the absence of apparently sufficient cause, and the age and habit of the individual are important points.]

Chronic interstitial pancreatitis sometimes results from the extension of chronic inflammatory processes affecting neighboring parts. Friedreich states that it sometimes is a primary disease in topers. Syphilitic lesions of the pancreas have been observed, occasioning contraction and induration.

There are no characteristic clinical symptoms which correspond to these various changes. The symptoms observed in the cases belonging in this category are digestive disturbances, indefinite pain, bodily weakness, emaciation, and anæmia, and they are so ambiguous that a diagnosis of the true condition is scarcely ever possible. If there were a single distinctive symptom, it would be one common to all sorts of grave pancreatic disorder—namely, the appearance of a large amount of fat in the stools. As we know, the pancreatic juice is an important factor in the digestion of fat, so that it is very natural for any great derangement of the organ to have this result; and yet the bile alone may render the ingested fat capable of absorption, so that in repeated instances there have been no fatty stools when the pancreas has been completely atrophied or degenerated.

4. **Cysts of the Pancreas.**—After closure of Wirsung's duct by scars, concretions, or the like, cysts of the pancreas may form, as a result of the damming up of the secretion. These may become so large as to be felt as great tumors through the abdominal walls. In some instances they have been operated upon with success. Rarely, spaces are found in the neighborhood of the pancreas filled with a thick, slimy fluid. These seem to be associated with the above-mentioned peculiar multiple necrosis of fat and pancreatic tissue, but with regard to them further observations are desirable.

5. **Cancer of the Pancreas.**—Primary cancer is the most frequent, and therefore clinically the most important disease of this organ. As a rule, the ne growth is situated in the head of the pancreas. It is usually of the medullar variety, though occasionally colloid. It may involve neighboring parts by direc extension, and a great many organs by metastasis; for example, the liver, peritoneum, and lymph-glands.

The clinical symptoms of cancer of the pancreas are very seldom so decided a to justify a positive diagnosis. Sometimes the secondary nodules can be detecte in the liver, peritoneum, and elsewhere. Then we are left in doubt about the sea of the primary growth. Or the primary tumor may be plainly felt through th abdominal walls; but then we can hardly ever exclude cancer of the stomach o of the omentum, and neighboring parts.

The symptoms of pancreatic cancer, as a whole, resemble closely those occasioned by most cancers of abdominal organs. Usually the patient is elderly. Th first symptoms are weakness, emaciation, and indigestion, or they are the result o compression. Often there is complaint of a persistent dull pain in the epigastrium. If the portal vein is pressed upon by the tumor, ascites appears. If th common duct is compressed, there is jaundice. Icterus is an especially frequen symptom of pancreatic carcinoma. The dejections have repeatedly been observe to contain a large amount of fat (*vide supra*), but there may be none whatever Osler remarks the noticeable frequency and large amount of the dejections and the quantity of undigested muscular fiber which they contain, because the pancreas no longer performs its part in the digestion of albuminoids. Sugar is sometimes present in the urine, but by no means in every case. Marasmus increases, and usually at the end of six months or a year the patient dies.

The **diagnosis** is difficult. It can be made with a certain degree of probability when there is a slowly growing tumor in the region of the pancreas, without enlargement of the liver, but accompanied by jaundice, deeply-seated epigastric pain, glycosuria, and the above-mentioned abnormalities in the fæces. Usually, however, the clinical picture is by no means so characteristic, and it is often impossible to avoid confusing cancer of the pancreas with cancer of the stomach, duodenum, gall-bladder, or other organs.

The **prognosis** is absolutely bad. The **treatment** is merely symptomatic, with the aim of lessening the patient's suffering.

6. **Pancreatic Calculi.**—In the excretory ducts of the pancreas are sometimes found calculi of small or moderate size, consisting essentially of carbonate and phosphate of lime. The cause of their formation is as little settled as that of gall-stones (which see). Because of the obstruction caused by these calculi we have secondary dilatation of the ducts, secondary contraction and atrophy, and in some cases secondary inflammation and suppuration; these probably result from processes similar to those with which we have become acquainted, while considering biliary calculi. There may be no symptoms. Sometimes, however, there is violent pain, either continuous or paroxysmal, also digestive disturbances, and occasionally the above-mentioned changes in the dejecta, and glycosuria. In only a very few cases has it been possible to find pancreatic calculi in the fæces. An absolute diagnosis can rarely be made. As a therapeutic measure Eichhorst has recommended the stimulation of the pancreatic secretion by means of injections of pilocarpine; as a rule, treatment will be purely symptomatic.

DISEASES OF THE URINARY ORGANS

SECTION I

DISEASES OF THE KIDNEYS

CHAPTER I

GENERAL PRELIMINARY REMARKS UPON THE PATHOLOGY OF RENAL DISEASE

ALTHOUGH some knowledge of the occurrence and significance of renal affections had been acquired even by the older physicians, still the service of having pointed out the frequency of these diseases, and of having clearly recognized their most important anatomical forms and their chief clinical symptoms, belongs undoubtedly to the English physician Richard Bright, who was born in 1788 and died in 1858, as physicion in ordinary to Queen Victoria. Bright's first work on this subject appeared in the year 1827. In this he brought forward the special discovery that, in many cases of general dropsy, which are associated with the secretion of an albuminous urine, a primary affection of the kidneys must be regarded as the true cause of the disease. Since then, the disease described by him has been almost universally called "Bright's disease" ("*Morbus Brightii*"), a name still much employed, but in whose stead the anatomical terms would be more proper, since many forms were previously classed under it which, according to our more accurate present knowledge, must be separated.

Bright's statements were either confirmed or expanded in subsequent times by many other observers. Christison, Osborne, and R. Willis in England, and Rayer and M. Solon in France, were the chief students of renal diseases. Frerichs published the first great work in Germany in the year 1851. His division of Bright's disease into three different "stages," based on Reinhardt's histological investigations, was for a long time quite generally accepted, until gradually further clinical experience showed that it was untenable. A more accurate division of renal diseases was substituted for it first in England (Johnson, S. Wilks, and others), and then in Germany (Traube, Bartels). However admirable these labors, especially the work of Bartels in 1871, renal pathology fell into subjection to theories, with which the facts of experience could be harmonized only by force. Only of late years has a natural theory of renal diseases, derived from general pathological observations, at last become accepted—a theory which is based chiefly upon the anatomical work of Weigert.

The following presentation of renal pathology is intended to show that, from a clinical standpoint as well, the renal diseases may be viewed comprehensively as a unit, and this not in a schematic way, but with due regard to actual conditions.

The chief reason why the kidneys are so often diseased, either alone or in conjunction with other organs, is to be found in the fact that the body must eliminate all forms of injurious matter, which circulate in the blood, in great part by the kidneys. Consequently the action of any injurious substance is often manifested chiefly in the kidneys, and they must, in a certain measure, suffer for the service which they do the rest of the body. According to their nature and character, the injurious substances, which are here to be considered, are divided chiefly into two great groups—the chemico-toxic and the organized infectious substances. At the same time, it should be noted that in case of infection it is comparatively exceptional for the germs themselves to reach the kidneys and there fix themselves. Most cases are referable to toxines, which are formed in the body as the result of the infectious process, and, being excreted through the kidneys, produce a nephritis. In this way the kidneys may be involved sympathetically after the ngestion of many poisons, both organic and inorganic, and also in the great majority of all the infectious diseases. In these cases, of course, as we shall see later, certain chemical and infectious poisons exert their action in a particularly frequent and in a particularly severe or definitely characterized fashion. Besides these forms of origin for many renal diseases, which are the chief ones to be considered, we must consider other causes of disease which are much rarer. One way in which the morbific agents may also enter is especially important—namely, from the lower urinary passages, the bladder, and pelvis of the kidney, upward into the kidney. In this way those renal diseases arise which come on secondarily to cystitis, pyelitis, etc. Finally, of course, disturbances of circulation and mechanical injuries may also make themselves manifest in the kidneys.

The clinical symptoms which are caused by the different forms of renal disease are referable only in very small part directly to the diseased organ itself. In renal diseases characteristic subjective local symptoms—such as local pain—are rare, and the anatomical position and the physiological conditions of the kidneys make it almost impossible to discover any changes in their size, their physical consistency, etc., by a direct objective examination. In the diagnosis of renal diseases we are therefore confined chiefly to the investigation of two groups of symptoms: in the first place, to the examination of the secretion from the kidneys, the urine, whose character, as we know by experience, may be materially altered when there is renal disease; and, in the second place, to the discovery of certain phenomena in other portions of the body, which are immediately dependent upon the renal affection. Since both the pathological changes in the urine, and the symptoms in other organs occurring in renal affections, have much in common in almost all the forms of renal disease, it is advisable first to describe the main features, at least, of the general symptomatology of renal diseases. We shall then be obliged, in the following chapters, to mention only the precise circumstances of the occurrence and onset of each symptom—the general significance of the symptoms being already known.

1. Albuminuria

The most constant symptom, which in many cases, even by itself, renders the diagnosis of a renal affection possible with complete certainty, is albuminuria—that is, the appearance of albumen, and especially of serum albumen and serum globuline (paraglobuline), in the urine. From recent investigations (Leube, Fürbringer, and others) we know that in some cases the urine may contain a very slight amount of albumen even in healthy persons, especially after physical exertion, emotional disturbance, a cold bath, a hearty meal, etc. Indeed, we find occasionally in some few individuals, particularly in early years, marked albuminuria, especially in the urine of the daytime, while the night urine never contains a trace of albumen; and yet these persons are apparently in perfect health. This has been called intermittent or functional albuminuria (*vide infra*). Yet it is at least questionable if such a condition, even if no symptoms are associated with it, can be regarded as physiological, and these facts do not impair the justice of the dictum that every case of persistent excretion of albumen in the urine, which can be demonstrated clearly by ordinary methods, is to be regarded as somewhat pathological.

The detection of albumen in the urine for clinical purposes, wherein no regard need be paid to the separation of serum albumen and serum globuline, is performed almost exclusively by means of the so-called heat test. If the urine is cloudy, it must be filtered before heating. The reaction of the urine must always be tested first. If the reaction of the urine is acid, as it ordinarily is, it is heated in the test-tube without any further addition. If the urine is neutral or alkaline, then, and only then, we acidify it slightly before boiling, by means of a few drops of dilute acetic acid. If the urine contains albumen there will appear, upon boiling, a distinct flocculent precipitate of coagulated albumen. The possibility of an error lies in the fact that sometimes, with a neutral or faintly acid urine, upon heating there appears a cloudiness because of the phosphates and carbonates which are precipitated. These are salts of lime and magnesia. In order to avoid confounding such phosphatic deposit with a deposit of albumen, we must in every case after the urine has boiled for a short time, if there is any deposit formed, add a few drops of nitric or acetic acid. By this means the deposit of phosphate or carbonate is immediately dissolved, while a deposit of albumen is not affected. The change in the color of the urine, which is sometimes caused by the addition of nitric acid, is due to the action of the acid upon the urinary pigments. We can measure the amount of albumen contained in the urine approximately by the height of the settled precipitate in the test-tube. We often speak of "one-half or one-fourth of the volume being albumen," but we can not state any definite relation between this estimate of the volume and the precise amount of albumen. Approximately, however, a deposit of albumen, which upon settling occupies about half the volume of the urine, corresponds to about one per cent. by weight of albumen; a deposit of about one-third of the volume to about one-half per cent. of albumen, and so on.

Besides the test by boiling there is a very distinct test which can be highly recommended to practitioners, in which acetic acid and ferrocyanide of potassium are employed. A rather large amount, perhaps one-tenth of the volume, of acetic acid is added to the urine, and then a few drops of a ten-per-cent. solution of ferrocyanide of potassium are added to the mixture. If the urine contains albumen there is formed a distinct deposit. This reaction usually takes place at once, but if the amount of albumen is very small, it may be somewhat delayed.

If we have found out that the urine certainly contains albumen, we must then decide whether we have really a true renal albuminuria—that is, whether a urine already albuminous is secreted in the kidneys, or whether the albumen is not mixed with a perfectly normal or at least non-albuminous urine later, in the kidneys themselves or in the urinary passages, the pelvis of the kidney, or the bladder (spurious, accidental albuminuria). Such a spurious albuminuria occurs when the urine is contaminated with blood (as in hæmorrhages from the kidneys, the pelvis of the kidney, the bladder, or the urethra), or with pus (in pyelitis, cystitis, etc.). In these cases, of course, the albumen contained in the serum of the blood or pus is found in the urine. Spurious albuminuria is usually easily recognized, since the presence of pus or blood in the urine, which is shown by the appearance of the urine or upon microscopic examination (red blood-corpuscles, pus-corpuscles), points with immediate certainty to the origin of the albuminuria. Moreover, the amount of albumen in these cases is usually but slight, and corresponds to the amount of pus or blood in the urine. A disproportion in this respect must excite the suspicion whether, beside the spurious albuminuria, there is not perhaps at the same time an affection of the kidneys causing a true renal albuminuria. The determination of this point is not always perfectly easy, but we can usually come to a decision by finding abnormal morphological constituents ine, the so-called urinary casts (*vide infra*), which give indubitable evi- the existence of a disease of the kidneys.

general pathological significance has the true renal albuminuria, and the causes of its origin? According to our present theories, the answer questions is simply this: In every case of genuine albuminuria there is rmal transudation of the albumen of the blood into the urine. The this transudation is chiefly the glomeruli, and its immediate cause is the l permeability of the walls of the glomeruli, due to disease of those struc- id particularly of their epithelium. The fact that the easily filtrated lbumen of the blood, as well as the water, does not pass through the loops of the glomeruli, even under normal conditions, is due entirely to imstance that the capillaries of the Malpighian bodies are not inserted the beginning of the uriniferous tubules, but that they are covered with m. This epithelium of the glomeruli has the physiological task and the providing for the retention of the albumen in the blood. If it suffers a ical change in any way, it loses this power, and then the albumen passes urine (Heidenhain). The inflammatory changes which the walls of the ssels themselves undergo probably facilitate the transudation of serum , as is suggested by what we know about inflammatory exudations in rts of the body. The best experimental proof of the theory that the in the walls of the glomeruli are the chief cause of the appearance of in the urine is furnished by the albuminuria which appears whenever ly of arterial blood to the kidney is checked by a temporary constriction enal artery. The epithelium of the glomeruli thereby suffers a visible pic change. If the kidneys in this condition are removed as rapidly as and boiled, according to Posner's suggestion, we can discover under the pe in the capsules of the glomeruli the albumen that is thus coagulated)—a most certain sign that the passage of the albumen from the blood- nto the urinary passages has in fact taken place in the glomeruli.

st all cases of albuminuria may readily be referred to analogous disturb- nutrition in the epithelium of the glomeruli, whether they be excited by s of the circulation, such as arterial anæmia or venous stasis, by toxic or s influences which have reached the glomeruli, or by any other circum- In these cases the changes in the glomeruli need not always be of a ere or irreparable nature; for we often see a slight albuminuria appear e most different conditions, and rapidly pass off again. This is the so- transitory albuminuria," which is seen, for example, in various febrile s, after slight intoxications, after epileptic attacks or in other severe conditions, in lead-colic, etc. We will show later how the anatomical in the glomerular epithelium explain the occurrence of albuminuria in nal diseases.

other factors, which have also been made answerable for the origin of uria, are without doubt quite subordinate to the changes in the epithe- the glomeruli, and at most they can affect merely the amount of albumen ed. The changes in the composition of the blood, on which formerly, and some investigators recently, great stress has been laid, especially the hy- and hypalbuminosis (the diminished amount of albumen) of the blood, bably only an indirect significance, since the nutrition of the walls of eruli suffers from such a faulty condition of the blood, and this circum- gain is the true cause of elimination of the albumen.

significance of the blood-pressure with regard to the occurrence of albu- was also formerly very much overrated. According to the older hypoth- was believed that, in an increase of the blood-pressure, the molecules of in the blood could be pressed through the filter formed by the membrane can be demonstrated clearly by ordinary methods, is to be regarded as somewhat pathological.

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Second, of white blood-corpuscles. These are often considerably swollen, so that we must guard against mistaking them for epithelium. Third, of renal epithelium, which may be recognized by its size, its more angular shape, and its nuclei. Of course, we often find the epithelium cloudy and granular, or shriveled and atrophied. Fourth, of fatty granular globules—that is, both fatty-degenerated epithelium and also white blood-corpuscles which are filled with fat-drops from the fatty-degenerated cells. Fifth, of little granular masses whose nature can not always be easily recognized. They are either coagulated granules of albumen, or fat-drops, or urates, or bacteria, or, finally, granules of hæmatoidine, which have come from the destruction of red blood-corpuscles, and are usually easily recognized by their dark, brownish-yellow color. Sixth, we rarely find in the casts drops like myeline, as to whose precise significance nothing is known.

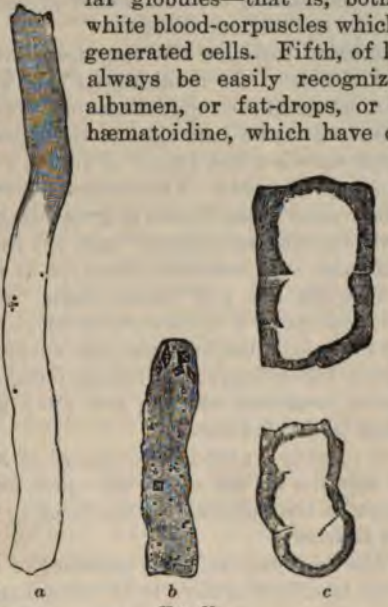

FIG. 69.

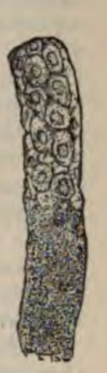

FIG. 70.

FIG. 69.—*a*. Waxy cast. *b*. Waxy cast bearing crystals of calcic oxalate. *c*. Fragments of waxy casts. (From von Jaksch.)

FIG. 70.—Epithelial cast, granular in its lower half. (From von Jaksch.)

As to the origin of hyaline casts, despite many investigations the question has not been fully answered. Probably they are due to the coagulation of the excreted albumen occasioned by the dying epithelial cells and the escaped leucocytes. It may be questionable whether, in this process of coagulation, the albumen excreted with the urine by the glomeruli is involved, or the albumen due to the inflammatory exudation and originating from the other renal blood-vessels. From analogy with other inflammations which lead to coagulable exudates,we regard the latter as the more probable supposition. Furthermore, many investigators assume that the desquamated and disintegrating epithelium is transformed into hyaline casts.

2. The *epithelial casts* are composed exclusively of renal epithelium, although probably hyaline casts not infrequently form a basis to which the epithelial cells adhere. Epithelial casts are usually easily recognized, and always indicate an excessive desquamation of epithelium in the diseased kidneys. One must be on one's guard, as already mentioned, not to confound renal epithelium with swollen white blood-corpuscles. The separate epithelial cells and the epithelial casts may present various changes, such as granular opacity, fatty degeneration, and atrophy.

3. The so-called *waxy casts* are almost always rather broad and usually yellowish-colored, opaque casts, of evidently a much tougher consistency than hyaline casts. We have found them most frequently in severe acute nephritis, either primary, or secondary to scarlet fever, but they also appear in the severer forms of chronic diffuse nephritis. As to their origin, we are convinced that they certainly in many cases are formed out of epithelial casts. The closely apposed renal epithelial cells degenerate into waxy flakes, and gradually coalesce. One can, in fact, observe all stages of transition between the epithelial casts and the almost completely homogeneous waxy casts. Fürbringer, therefore, terms waxy casts "metamorphosed"; and he insists, correctly, that they always indicate severe disease of the kidney.

of the glomeruli. This hypothesis has been disproved, especially by the experiments of Runeberg; these experiments showed that, in the filtration of solutions of albumen through animal membranes, a rise in the filtration pressure was followed by a decrease, and a fall in the pressure by an increase of the percentage of albumen in the filtrate.

Although in the preceding paragraphs only the Malpighian bodies have been regarded as the spot where the transudation of the albumen of the blood into the urine takes place, we must also note that, under some circumstances, we may admit the possibility of a passage of albumen directly into the tubules from the capillaries that encircle the uriniferous tubules; but we must also necessarily assume in such cases that there is a disturbance of nutrition in the membranæ propriæ, or at least in the epithelium of the uriniferous tubules. Such an assumption seems to explain the albuminuria, according to Senator's experiments, in venous stasis in the kidneys, although in these cases the epithelium of the glomeruli also suffers soon, and then becomes pervious to albumen.

2. Casts and other Abnormal Morphological Constituents of the Urine in Renal Disease

Beside albuminuria, certain peculiar morphological constituents of the urine, visible under the microscope, are of especial importance for the diagnosis of renal affections—the urinary casts, whose significance was first correctly recognized by Henle in 1842. These are cylindrical bodies, whose breadth corresponds to the width of the uriniferous tubule, and whose length only exceptionally reaches a millimetre, which must be regarded in their chemical nature as consisting mainly of a coagulated albuminous substance. To the latter circumstance we owe their old name of "fibrine casts," or "fibrous casts," a name which is obsolete, and properly so, since the coagulated albuminous substance of casts is certainly not identical with fibrine.

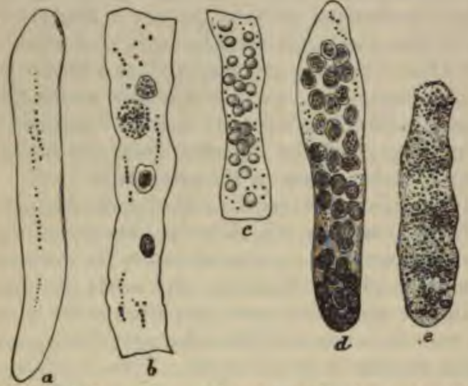

Fig. 68.—Different forms of casts. *a.* Hyaline cast with occasional granules. *b.* Hyaline cast with fat-drops and granular cells. *c.* Hyaline cast with red blood-corpuscles attached. *d.* Hyaline cast with white blood-corpuscles attached. *e.* Cast with a large number of fat-drops.

Since the precise conditions of the occurrence and the character of the renal casts, in the different diseases of the kidneys, will be spoken of later, we need discuss here only the general properties, the origin, and the significance of casts (see Fig. 68).

1. *Hyaline Casts.*—The hyaline casts are the commonest and most important form of casts, and, to a certain extent, are the ground-form for different varieties. They are perfectly homogeneous, clear as glass, colorless, soft, and flexible. We find them either wide or narrow, sometimes broken off short, sometimes quite long, usually straight, but in many cases partly curved. They are easily stained with carmine or gentian-violet. On heating the urine, they are dissolved, but they are quite resistant to acids.

The hyaline casts are very often covered to a greater or less extent with all sorts of deposits, which are usually affixed to the soft substance of the cast in the kidney itself, but which may often be attached to it later. These deposits are as follows: First, red blood-corpuscles. This condition is important, because it points with certainty to the existence of hæmorrhages in the kidneys themselves.

Second, of white blood-corpuscles. These are often considerably swollen, so that we must guard against mistaking them for epithelium. Third, of renal epithelium, which may be recognized by its size, its more angular shape, and its nuclei. Of course, we often find the epithelium cloudy and granular, or shriveled and atrophied. Fourth, of fatty granular globules—that is, both fatty-degenerated epithelium and also white blood-corpuscles which are filled with fat-drops from the fatty-degenerated cells. Fifth, of little granular masses whose nature can not always be easily recognized. They are either coagulated granules of albumen, or fat-drops, or urates, or bacteria, or, finally, granules of hæmatoidine, which have come from the destruction of red blood-corpuscles, and are usually easily recognized by their dark, brownish-yellow color. Sixth, we rarely find in the casts drops like myeline, as to whose precise significance nothing is known.

As to the origin of hyaline casts, despite many investigations the question has not been fully answered. Probably they are due to the coagulation of the excreted albumen occasioned by the dying epithelial cells and the escaped leucocytes. It may be questionable whether, in this process of coagulation, the albumen excreted with the urine by the glomeruli is involved, or the albumen due to the inflammatory exudation and originating from the other renal blood-vessels. From analogy with other inflammations which lead to coagulable exudates,we regard the latter as the more probable supposition. Furthermore, many investigators assume that the desquamated and disintegrating epithelium is transformed into hyaline casts.

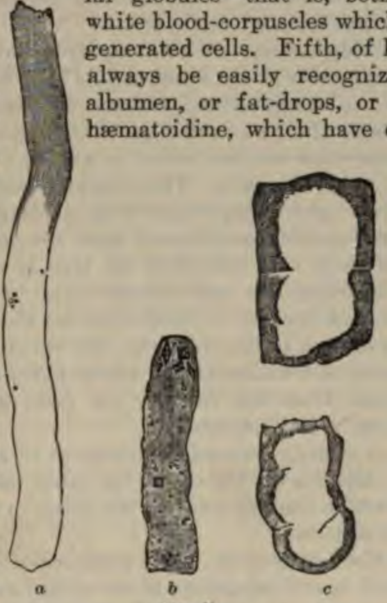

FIG. 69.

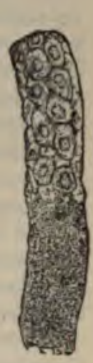

FIG. 70.

FIG. 69.—*a*. Waxy cast. *b*. Waxy cast bearing crystals of calcic oxalate. *c*. Fragments of waxy casts. (From von Jaksch.)

FIG. 70.—Epithelial cast, granular in its lower half. (From von Jaksch.)

2. The *epithelial casts* are composed exclusively of renal epithelium, although probably hyaline casts not infrequently form a basis to which the epithelial cells adhere. Epithelial casts are usually easily recognized, and always indicate an excessive desquamation of epithelium in the diseased kidneys. One must be on one's guard, as already mentioned, not to confound renal epithelium with swollen white blood-corpuscles. The separate epithelial cells and the epithelial casts may present various changes, such as granular opacity, fatty degeneration, and atrophy.

3. The so-called *waxy casts* are almost always rather broad and usually yellowish-colored, opaque casts, of evidently a much tougher consistency than hyaline casts. We have found them most frequently in severe acute nephritis, either primary, or secondary to scarlet fever, but they also appear in the severer forms of chronic diffuse nephritis. As to their origin, we are convinced that they certainly in many cases are formed out of epithelial casts. The closely apposed renal epithelial cells degenerate into waxy flakes, and gradually coalesce. One can, in fact, observe all stages of transition between the epithelial casts and the almost completely homogeneous waxy casts. Fürbringer, therefore, terms waxy casts "metamorphosed"; and he insists, correctly, that they always indicate severe disease of the kidney.

4. *Granular Casts.*—A kind of cast which is composed of coarse, yellowish, flaky granules represents the just-mentioned transition of epithelial casts into waxy casts. In other cases the waxy casts are nothing but hyaline casts completely covered with the above-mentioned granules of various kinds. Sometimes, also, coagulated masses of albumen or granules of hæmatoidine may themselves be formed into cylindrical shapes.

Genuine blood casts are not very frequent. They consist of coagulated blood and represent casts of the renal canals into which hæmorrhage has taken place.

The clinical diagnostic significance of renal casts is very great. They are, in the first place, always a sure sign of the existence of some renal disease, since in normal urine casts are not found at all, or, at most, they are exceptional and are present in small numbers. The consideration of the special forms of casts, and of the deposit upon them, is also of great diagnostic importance, although from it we can never decide immediately upon the general form of the renal disease, but we can recognize with certainty the type of special pathological processes in the kidneys. Frerichs has well named casts "messengers from the kidneys." The blood-casts and the red blood-corpuscles sticking to the cylinders point to the occurrence of renal hæmorrhages; the epithelial casts to a desquamation of the epithelium in the kidneys; the white blood-corpuscles to an emigration of the colorless cells from the vessels; the fatty granular cells to processes of fatty degeneration in the kidneys.

We have already learned to recognize in a great measure in the preceding, as occasional deposits on the casts, the other morphological constituents found, beside the casts, in the sediment of the urine in renal disease. Briefly recapitulated, they are as follows:

1. *Red blood-corpuscles.* The presence of a large amount of blood in the urine (hæmaturia) is almost always to be recognized by its blood-red color. The blood may be made out with certainty by the microscope, or by Heller's blood-test. The latter is performed by heating the urine in a test-tube with sodic or potassic hydrate. The blood-corpuscles are thus dissolved, and the hæmatine formed from the blood-pigment is precipitated with the phosphates, giving to the precipitate of the latter a very characteristic blood-red color. Van Deen's test is very distinct and easily performed. It requires a freshly prepared mixture of equal parts of old oil of turpentine and newly made tincture of guaiac. This mixture is poured upon the urine to be examined, and slightly shaken. At the place of contact of the two fluids there will immediately form a beautiful blue ring, if there is the slightest trace of blood in the urine. Finally, of course, the spectroscope may serve for the detection of hæmaturia. Hæmoglobinuria will be described in a special chapter later.
2. *White blood-corpuscles.* Only when they are also attached to the casts can we assume with certainty that these come from the kidneys, and not from the lower portions of the urinary tract.
3. *Renal epithelium.*
4. *Fat-drops and fatty granular cells.*
5. *Uric-acid crystals, urates and calcic oxalate, bacteria,* etc.

For microscopic examination of the urinary sediment, we let the urine settle in a tall beaker; it is more convenient, and, if the urine contains few morphological constituents, far more reliable to employ a centrifugal machine.

3. THE DROPSY OF RENAL DISEASE

Although the changes in the urine must be alone decisive in the diagnosis of any renal disease, there are yet certain other symptoms which are also due immediately to the renal affection, and which may first direct our suspicions to the existence of a disease of the kidneys, and consequently lead to a careful examination of the urine. Among these symptoms the dropsy of renal disease is one of the commonest and most important. This may, indeed, quite frequently be entirely absent, both in acute and chronic nephritis, and in other diseases of the kidneys; but in many cases it is decidedly prominent in the whole clinical picture.

If we ask what is the reason of the frequent occurrence of dropsy in renal disease, the answer at first does not seem difficult. Since the main function of the kidneys is to excrete water from the body, and since, as we shall see later, in many cases the diseased kidney can no longer fulfill this task, or can fulfill it only to a slight degree, we are not, in fact, very much out of the way in considering the retention of water in the body as the main cause of the consequent œdema. Clinical observation seems in general to agree completely with this assumption. The œdema in renal disease seldom appears until the daily amount of urine has been below the normal for some time, while, on the other hand, in those cases where the amount of urine passed is normal, or even abnormally great, in spite of the existing renal disease, œdema is usually wholly absent. In individual cases, too, we very often see a decrease of the œdema associated with an increase in the amount of urine, and an increase of the œdema associated with a corresponding diminution in the excretion of urine. The pathological process accordingly seems to consist of an accumulation in the body of the water which can not be excreted from it, and which transudes from the vessels and thus gives rise to the development of œdema.

On more careful consideration, however, there are some objections to this theory, which is apparently so simple. In the first place, it might be supposed that, when there is retention of water, the body must get rid of the surplus water by employing to a greater degree the other channels of elimination which are at its service—the skin and the intestines. Since we can never determine accurately the time when the water first begins to be retained in the body, the clinical experience just mentioned may also be thus interpreted, that the lessened excretion of urine is not the cause of the œdema, but that, on the contrary, the appearance of œdema is rather the cause of the diminished elimination of water by the kidneys. The other clinical facts, also, are inconsistent with the view that the œdema depends upon the simple accumulation of water in the blood. In many forms of nephritis, and particularly in the nephritis of scarlet fever, we very often see extreme œdema appear suddenly, while, on the other hand, many severe varieties of nephritis—e. g., those associated with diphtheria, septic diseases, and pneumonia, run their course with little or no œdema, although they are characterized by a marked diminution in the amount of urine. Moreover, cases have been repeatedly observed in which, as a result of obstruction of the ureters, or from pressure on these canals, there has been complete anuria for several days without a trace of œdema developing; and experimental investigations have given corresponding results. The bilateral ligation of the ureters in animals does not lead to the development of œdema even after several days. Cohnheim and Lichtheim introduced large amounts of a one-half to one-per-cent. solution of common salt into the vascular system of an animal, and despite this great artificial "hydræmic plethora," observed no œdema, even when the renal arteries were tied. Nor can œdema be provoked by slow and continuous infusion (Gärtner, Francotte).

Consequently, we must seek some other cause to explain the œdema of nephritis, and this is in all probability the peculiar change in the vascular walls, which renders them more permeable and permits the water accumulated in the blood to transude into the tissues. Just what this change in the vascular walls may be and what produces it we do not yet know; probably there are chemical agents which change the vascular walls, either the same substances which excite the nephritis, or matters secondarily formed, or certain products of metamorphosis retained in the blood because of the inefficiency of the kidneys. That dropsy develops because of direct damage to the blood-vessels is rendered probable from the cases of so-called "acute essential dropsy," in which there develops a considerable general dropsy, just as in acute nephritis, often preceded by mild gastro-intestinal symptoms, and yet without a trace of albumen in the urine or any other demonstrable cause for this dropsy, which usually soon abates.

Many clinical peculiarities of nephritic œdema harmonize with this conception of its origin. In general, we may say that the œdema of nephritic patients appears first in the skin ("anasarca"), and has a striking tendency to show first in the face, in contrast with the œdema due to the passive congestion of heart disease, which usually begins in the ankles. The puffy and noticeably pale countenance of the patient will often suggest the existence of kidney trouble at the first glance. In severe cases the dropsy often develops over the whole body in an extreme degree, involving the skin of the trunk, and particularly the dependent parts, the extremities, and scrotum, so that the patient is a pitiable object. In such cases there are also apt to be dropsical transudations into the internal cavities of the body, causing hydrothorax, ascites, and hydropericardium; and these aggravate the patient's distress. It is, however, a striking fact that often there will be marked dropsical effusions (ascites, hydrothorax), and yet no marked œdema of the skin; and hydrothorax is sometimes much greater on one side than on the other. All these facts indicate special local conditions—viz., the changes in the blood-vessels, which we have surmised. Still more is this the case with regard to the œdema of mucous membranes which sometimes occurs. There may be œdema of the conjunctiva, of the soft palate, of the ary-epiglottic ligaments (œdema of the glottis), and other parts. Œdema of this sort is apt to resemble a mild local inflammation, and in general it is impossible to deny a certain relationship between "inflammatory" œdema and nephritic œdema. The nephritic œdema of the skin may in places have suggestions of an inflammatory nature, such as slight redness and tenderness. Œdema may affect the internal organs, and nephritic pulmonary œdema is of great practical importance. The question of the existence of cerebral œdema and of its possible importance will be discussed below (see uræmia).

The preceding remarks relate only to genuine nephritic œdema. We shall see later that œdema may have an entirely different origin, particularly in cases of chronic nephritis—that is, if there is a cardiac hypertrophy and the powers of the heart become impaired, dropsy may at last develop. This œdema is of course a genuine congestive œdema, and exactly similar to that seen in failing compensation in heart disease.

In its chemical composition, the dropsical fluid corresponds to a very thin blood-serum. The amount of water is usually 97 to 98 per cent., the amount of salts one to one and a half per cent. The amount of albumen is usually very slight. Urea has been repeatedly found in the fluid.

4. Uræmia

If the diseased kidneys can no longer perform their secretory functions in a satisfactory way, not only does the elimination of water from the body thereby suffer, but the soluble constituents of the urine, the salts, the urea, and the other final products of tissue metamorphosis may also be retained in the blood and accumulate there. Hence we often find the blood, in patients with renal disease, not only more watery than under normal conditions, so that the specific gravity of the serum may fall from 1030 to 1020, or even lower, but, in almost all cases where there is a diminished excretion of urine, it is also richer in urea, as many experiments have shown, and under corresponding conditions it is probably also frequently richer in the other constituents of the urine, or in substances corresponding to them but not completely metamorphosed.

This accumulation of the urinary constituents in the blood, and further, perhaps, in the tissues themselves, is the cause of a class of symptoms which are often seen in diseases of the kidneys, and which are termed uræmic symptoms or uræmia.

Probably no one to-day doubts that uræmia must be regarded as essentially an intoxication of the body by the retained products of tissue metamorphosis. Numerous experimental investigations have proved that in animals extirpation of the kidneys, or ligation of the ureters, will produce a symptom-complex, characterized by vomiting, convulsions, and coma—almost completely analogous to the uræmia of Bright's disease; but if we inquire what constituents of the urine are the particular occasion of the uræmic phenomena, we can not as yet obtain any definite answer. For a long time it was believed that urea played a chief part in the development of uræmia, but the result of experiments upon animals does not support this view. It is possible to inject enormous amounts of urea into the circulation or into the peritoneal cavity of animals without any symptoms of poisoning. Voit did, indeed, show that the healthy kidneys remove from the blood the excessive amount of urea with extreme rapidity, and that accordingly uræmic symptoms do really appear if, while we are feeding an animal with large amounts of urea, we impede the excretion of the urea by a simultaneous withholding of water. Yet the amount of urea necessary for the success of this experiment is greater than can possibly exist in the ordinary uræmia of Bright's disease; and, moreover, the withholding of water might also prevent the excretion of other matters; hence we must seek for other poisonous substances as factors in the production of uræmia. Many experiments seem to indicate that the potassium salts are especially poisonous, while some authors have laid the blame mainly on the extractive matters, such as creatinine. Bouchard has sought to prove that certain alkaloid substances (urotoxines), which are probably developed during the digestion of albumens and are always demonstrable in normal urine, occasion the phenomena of uræmia; but to all these suppositions there are serious objections, so that really the substances which occasion uræmic intoxication are as yet unidentified. It might be possible that the poison corresponding to uræmia is not, in every case, the same.

The mode of action of the toxic material is as unsettled as is its chemical nature. This much only is certain, that in uræmia the disturbances are almost exclusively cerebral, and in the main located in the cortex of the brain; but whether there is actually a direct injury of the nervous elements, or whether the immediate action is upon the blood-vessels, is uncertain. The occurrence of uræmic focal symptoms, to which we shall refer below, renders the former supposition the more likely. Fleischer produced uræmia experimentally in animals, and in every case observed an extreme anæmia of the brain and spinal cord, so that he concludes that there is a spasm of the blood-vessels.

Clinical experience also agrees perfectly with the theory that uræmia is caused by a retention of urinary constituents in the body. In most cases the uræmic symptoms appear only when the daily amount of urine has fallen to a very low figure, or when the secretion of urine has wholly ceased for several days. That in these cases not only the elimination of water, but also the elimination of an amount of urea corresponding to the food taken, and also the elimination of the other urinary constituents, is very much diminished, is shown by the experiments in regard to this point made by Fleischer and others. Furthermore, a great increase of the amount of urea in the blood in uræmic patients has been found in many if not in all cases.

Of course there can be no question that some clinical facts can not be brought into exact harmony with what has been previously said. If cases are repeatedly reported in which no uræmic symptoms have appeared in spite of anuria lasting several days, it does not prove very much, since we can never make an exact estimate of the matter accumulated in the blood which ought to have been eliminated; for the organism can certainly get rid of the final products of tissue metamorphosis in other ways than through the kidneys—for instance, through the skin or the intestines—and we must also bear in mind that different individuals show a great diversity in tolerating the action of any poison in the body, particularly as regards the nervous system. It is harder to explain those cases which are sometimes seen, in which uræmic symptoms suddenly appear in patients with renal disease, although these symptoms are not preceded by any noticeable diminution of the secretion of urine. We may, however, assume that, despite the abundant excretion of water and the normal amount of urine, there has been a slight retention of solid matters. Analogy with other kinds of poisoning makes the supposition very plausible that the long persistent retention of even extremely small portions of toxic matter may occasion an absolutely sudden explosion of the severest symptoms. In chronic lead and mercurial poisoning the symptoms often appear with great abruptness, although the poisoning has taken place very slowly and gradually. In the same way we explain to ourselves the not very infrequent cases of the sudden development of severe uræmic symptoms, as seen particularly in patients with contracted kidney (*vide infra*), in whom it may seem as if no single prodromal symptom had indicated the impending outburst of intoxication. Often, also, peculiar circumstances may favor the occurrence of uræmia—for example, the development of cardiac weakness, so that the blood tension is diminished and the excretion of urine impeded. In some cases again it is observed that the development of uræmia coincides with the absorption of previously existing œdema. This is explained by the supposition that the rapid absorption of the œdema introduces into the blood a comparatively large amount of the poisonous products of metabolism, which had not been excreted, but had been contained in the œdematous fluid.

While we are firmly of the opinion that uræmia is to be regarded as a poisoning of the body by the retained constituents of the urine, yet we should not fail to mention that attempts have been made to explain in other ways the uræmic phenomena. In particular, Traube has propounded the theory that the so-called uræmic symptoms are dependent upon an acutely developing cerebral œdema, and consequent cerebral anæmia. This theory can be regarded as, on the whole, untenable. Still, it may contain a certain amount of truth, for we can not wholly deny that sometimes there may be actual anatomical lesions, such as inflammatory œdema of the brain, in the course of a nephritis, sufficient to occasion severe cerebral symptoms. During nephritis, secondary inflammation may appear in almost all the internal organs, and often suddenly; and this sort of nephritic inflammation is especially frequent in the retina, a structure composed of nervous elements, so that the possibility of similar disease in the brain is very great. If we consider that cases of uræmic hemiplegia and monoplegia have been repeatedly observed, and that likewise cases have been described of uræmic Jacksonian epilepsy, hemianopsia and aphasia, it will be seen that the supposition of an actual local lesion as the explanation of such well-marked focal symptoms is very plausible. Yet we must also consider that in the last analysis the cause of such limited œdematous—or inflammatory-œdematous—changes in the brain is to be sought in the action of some retained products of metamorphosis.

Finally, we may mention here the theory advanced by Frerichs in the year 1851, which at first found much favor, but which at present is almost universally abandoned. According to this, the urea retained in the blood was not in itself the cause of the uræmic symptoms, but it was changed into carbonate of ammonium by the action of a ferment in the blood, and from this the severe nervous symptoms arose. This theory is untenable, because carbonate of ammonium is scarcely ever found in the blood of uræmic patients. It is much more probably formed first in the stomach and intestinal canal of uræmic patients from the urea there excreted (*vide infra*), as Claude Bernard, Treitz, Voit, and others have shown.

In regard to the clinical symptoms of uræmia in the individual case, they show all possible transitions from the mildest symptoms, which are only intimated, up to the severest nervous symptoms, which may be the immediate cause of death. The severe forms of uræmia may sometimes come on quite suddenly, while in other cases they may be preceded for a long time by milder uræmic symptoms, which are then termed prodromata. The severest symptoms may not appear at all, and the milder symptoms may exist alone for a longer or shorter time. This latter condition is called chronic uræmia.

The milder uræmic symptoms, which are observed either alone or as precursors or as sequelæ of severe uræmia, consist of headache, somnolence, and mental stupor, of a peculiar uneasiness, or of a feeling of anxiety and constraint (sometimes associated with hurried respiration), and very often of nausea, spasmodic eructations, and repeated vomiting; and, finally, not infrequently, of various symptoms of motor irritation, of slight twitchings or temporary tonic rigidity of the face or the extremities, etc.

Among the characteristic symptoms of this milder form of uræmia, besides the vomiting, we would place the peculiar restlessness and præcordial distress of patients as especially important. Not infrequently, observers use the term uræmic asthma (*vide infra*).

The most characteristic symptom of severe uræmia is the uræmic convulsion, or the so-called uræmic eclampsia. It corresponds almost exactly in its details to an epileptic attack; it usually begins with a short tonic stage, in which the whole body is generally in a position of extension in opisthotonos, and then follow vigorous clonic contractions in the face and extremities. The face becomes cyanotic, a bloody froth comes from the mouth, the pupils are usually dilated and almost without reaction, the respiration is accelerated (but at times it is intermittent from spasm of the respiratory muscles), the pulse is small and accelerated, and it can scarcely be felt in the radial artery, and the temperature is sometimes raised. In other cases the spasm begins with short jerky contractions in one extremity, as in the arm, and then invades the trunk muscles, the face, and the legs. One half the body is often more affected in the attacks than the other. The spasms usually cease in a few minutes, and are followed by deep coma and stertor, which last for several hours or more. There is only rarely a single attack. The attacks are much oftener repeated after longer or shorter intervals, so that there may even be twenty or more in the twenty-four hours, during the whole of which time a complete loss of consciousness persists. Severe and fully developed epileptiform attacks often alternate with slighter convulsions.

Some other uræmic symptoms, besides the convulsions, which have already been briefly mentioned, merit a somewhat fuller description.

The uræmic amaurosis occasionally seen is especially interesting. It is usually left after recovery from the convulsions. Only rarely does it precede them or appear without them. It always develops quite rapidly, so that the first disturbance of vision soon passes into complete blindness. The reaction of the pupils to light is almost always retained, and the ophthalmoscope shows a perfectly normal retinal image. At what spot in the visual apparatus the lesion is situated is not yet known. Many investigators assume that there is an œdema of the sheath of the optic nerve; while others, including the author, think it more probable that there is disturbance of the cerebral visual centers, and particularly of the occipital cortex.

Its prognosis is on the whole favorable, since the disturbance of vision usually disappears completely in a day or two, though sometimes not until after a longer time. Anomalies are only rarely seen in the domain of the other nerves of special sense, the most frequent, comparatively, being a difficulty in hearing, or even complete deafness.

Other motor disturbances, except twitchings and convulsions, are rare. Only in a few cases have hemiplegic or monoplegic paralyses, contractures, trembling, etc., been observed. Mental symptoms are more common. Delirium, and maniacal or sometimes melancholic states, occasionally follow uræmic coma.

Those uræmic symptoms have also a great interest which are to be regarded as a sort of self-help on the part of the organism, since they often lead to a vicarious elimination of urea and presumably of the other products of tissue metamorphosis. The first of these is uræmic vomiting, which is a frequent and often an extremely obstinate symptom both in acute and chronic uræmia. In many cases it is of central origin, and is to be regarded as analogous to the vomiting so frequent in different forms of cerebral disease; but it is often produced by the irritation which the gastric mucous membrane suffers from the urea eliminated, or rather from the carbonate of ammonium arising from it. The latter is always first formed from the urea in the stomach itself, and we find in the vomitus of uræmic patients either the still undecomposed urea or the carbonate of ammonium in considerable quantities. Sometimes there is quite a violent hiccough besides the vomiting.

Uræmic diarrhœa has the same significance as uræmic vomiting. It is usually provoked by the carbonate of ammonium arising from the urea in the intestines. The latter often causes quite a severe catarrhal, and even at times a diphtheritic, inflammation of the intestinal mucous membrane.

Another way in which the organism sometimes tries to get rid of the large amount of urea accumulated in it is by the sweat-glands. Schottin first described the remarkable discovery of a coating of urea on the skin in the uræmia of cholera, an observation which since then has been repeatedly confirmed in other cases of uræmia. This coating is most frequently seen on the face, especially on the sides of the nose, to which little faintly lustrous scales are seen sticking after the evaporation of a clammy sweat. Chemical examination shows that these scales are urea. The excretion of urea is much more rare in other parts of the skin, but perhaps the occasional severe uræmic itching of the skin is due to an irritation of the cutaneous nerves by some of the constituents of the urine that are excreted.

Other organs besides the skin and the digestive tract are but rarely to be considered as a means of the vicarious elimination of urea, but Fleischer was once able to discover considerable amounts of urea in the saliva and sputum of a uræmic patient.

In conclusion, we must describe the condition of the pulse, of the temperature, and of the respiration in uræmia. The pulse is often very slow before the appearance of severe symptoms, sometimes 48 or 40, but it is almost always tense and hard. In chronic uræmia, also, a moderate slowness of the pulse is not infrequent. When uræmic convulsions appear, however, the pulse usually becomes small and very frequent, especially in cases that terminate unfavorably. The temperature but rarely remains unchanged in severe uræmia. If there are convulsions, it usually rises several degrees, in severe cases even to 106° or 108° (41°–42° C.). We have seen these high temperatures, especially, as a terminal rise with an unfavorable issue, although there may sometimes be an improvement even in such cases. On the other hand, there are also great declines in temperature, down to 93° or 91° (34°–33° C.), most frequently again as a terminal temperature of collapse, in cases which end in deep coma without marked symptoms of motor irritation. We might also mention the "uræmic chills" which we have seen several times—that is, a chill coming on suddenly along with other uræmic symptoms, with a great increase of temperature, and followed by a rapid fall in the temperature. The respiration in uræmic patients is sometimes very much accelerated, and is especially deep—a symptom which recalls the peculiar breathing in diabetic coma (*vide infra*). Certain severe attacks of dyspnœa in patients with renal disease have been described as "uræmic dyspnœa" or "uræmic asthma"; but it is not always easy to decide whether this is really a uræmic nervous symptom in these cases, since similar conditions of sudden dyspnœa may depend upon coincident heart disease and insufficiency of the left ventricle or upon inflammatory affections of the lungs.

In regard to the duration of uræmic symptoms and to the different forms and ways in which the various uræmic symptoms may be combined in the clinical picture, we can give only a few general statements. The division of uræmia into an acute and a chronic form, already mentioned, is generally very useful practically. In the acute form we usually have the severe uræmic symptoms, especially uræmic convulsions and uræmic coma. This condition usually lasts some days, while chronic uræmia, in which the milder cerebral symptoms—uræmic vomiting, difficulty in breathing, etc.—are most prominent, may last as many weeks. The severe acute form of uræmia may, as has been already stated, begin with almost absolute abruptness. Quite often, however, there are first mild uræmic symptoms, such as headache, vomiting, general restlessness, and an occasional slight twitching of the muscles, and then suddenly uræmic convulsions or other uræmic symptoms.

The termination of uræmia is always doubtful in every severe case, but it is by no means always unfavorable. Even after coma lasting for several days, with very severe and often-repeated convulsions, the uræmic symptoms may wholly disappear, while on the other hand, of course, uræmia is by no means a rare cause of death in the most diverse forms of acute and chronic renal disease. In judging of the individual case, the most stress is to be laid on the condition of the pulse, the respiration, and the temperature; we must also consider, of course, the character of the urinary secretion, and especially the other morbid symptoms dependent upon the primary disease.

5. The Changes in the Circulatory Apparatus in Renal Disease

Although it had not escaped Bright's observation that changes in the heart are also present in diseases of the kidney, this condition was first generally known when Traube, in 1856, in a treatise which has become famous, explained that a change in the heart was very common in certain renal affections, and thus gave the chief impulse to the numerous clinical and experimental investigations that have been made since then as to the connection between cardiac and renal disease.

This connection, generally considered, may be accounted for in three ways:

First, the heart disease may without doubt be the primary disease, and only secondarily lead to a disease of the kidneys. In this way develop the kidney of passive congestion (*vide infra*, and page 331), acute nephritis secondary to acute primary or recurrent endocarditis, and the embolic processes in the kidney (*vide infra*).

Secondly, heart disease and renal affections may also develop independently of each other, as a result of an injurious influence that affects both organs at the same time. Thus, for example, a general arterio-sclerosis leads to cardiac hypertrophy or to myocarditis, and also to a granular kidney (*vide infra*), as a result of an implication of the renal vessels. Certain other injurious influences, such as toxic and constitutional influences, alcohol, syphilis, or improper living, may also cause a disease of the heart and the kidneys at the same time. Later on, if both affections have developed, their influence upon each other is often, of course, considerable—a circumstance which may render our judgment as to the condition decidedly difficult.

In the third place—and this is the point with which we are here chiefly concerned—the renal affection may be the primary disease, and of itself the cause of a change in the heart, and especially of a secondary hypertrophy of the left ventricle. At present there can no longer be any doubt of the fact of this dependence. We also know now that the secondary development of cardiac hypertrophy is not confined to one form of chronic nephritis, the so-called contracted kidney, as was at first believed, but that it is almost as constant in all other forms of chronic, and also even in prolonged acute, nephritis. Opinions are at present still much divided as to the precise nature of this connection, and as to the causal factors.

The theory which Traube himself advanced for the explanation of the cardiac hypertrophy in nephritis rested on the assumptions that, in the first place, less water is withdrawn from the blood in nephritis for the formation of the renal secretion, and that, in the second place, the flow of arterial blood into the venous system is hindered by the changes in the kidneys, particularly by the destruction of many of the smaller blood-vessels. Both circumstances must raise the pressure in the arterial system, and therefore gradually lead to cardiac hypertrophy. Traube's theory can not be maintained. The first claim especially is untenable, because in many cases of chronic contraction of the kidney with co-existing cardiac hypertrophy there is never a diminution of the elimination of water by the kidneys, and, besides, this can never of itself cause an increase of the arterial pressure. The second supposition, that the destruction or the narrowing of a large number of the small blood-vessels of the kidneys must produce a general rise in arterial tension, is disproved by the fact that even the complete ligation of both renal arteries does not raise the tension in the arterial system, because the blood at once passes off into other vessels which dilate.

In place of the "mechanical theory," therefore, there have been of late many supporters of the "chemical theory" of cardiac hypertrophy, which was in a certain sense propounded by Bright himself, and later by Johnson and others. According to this view which, with some modifications, is held by the author, the retention of urinary constituents in the blood is the cause of the cardiac hypertrophy because the retained material occasions a rise in arterial tension, and the increased tension, if it persists for a sufficiently long time, must occasion a hypertrophy of the left ventricle. Clinical experience shows beyond a doubt that any severe case of acute nephritis occasions in a few days an increase in the arterial tension, which can usually be easily perceived in the pulse. This increased tension, which certainly precedes the cardiac hypertrophy, is best explained by the supposition that the matters which ought to be but are not excreted occasion a contraction of the smaller arteries. It is also possible to conceive that there may be a direct irritation of the cardiac muscle. At any rate, the occurrence of a rise in arterial tension is of decided benefit to the body, as it promotes the excretion of urine. If, in the course of a nephritis, the normal conditions return after a few days or weeks, the increased tension is relaxed and the heart suffers no noticeable change; but if the nephritis and consequent impairment in urinary secretion and increased arterial pressure persist for a considerable time, we often see, even after six or eight weeks, a hypertrophy of the left ventricle develop, under our own eyes, in a way that can be demonstrated most distinctly at the bedside. This is the necessary consequence of the increased effort which the heart is obliged to make in order to overcome the abnormal resistance in the systemic arteries.

We have precisely the same conditions in the chronic forms of nephritis, except that they develop more slowly and insidiously. In these, also, the first factor is the insufficiency of the kidneys, occasioned by the disease—that is, failure to excrete all the products of metabolism. To this the body responds immediately by a rise in arterial tension, which is intended to serve, and does actually serve, as a compensation of the impairment; then, thirdly, hypertrophy of the left ventricle develops, and enables the heart for a long while to supply the demands put upon it. This hypertrophy is consequently the most important and indispensable compensatory arrangement by means of which the body is protected from the onset of uræmic intoxication. Just as any patient with valvular heart disease would invariably fail much earlier if his heart did not become hypertrophied in portions corresponding to the lesion, so, also, in chronic nephritis, the unfavorable termination would occur much earlier if the body were not in a position to effect and maintain an increase in the arterial tension, and thus for a time, at least, ward off the threatening enemy. Viewed in this light, the chemical theory of cardiac hypertrophy not only gives us an insight into the clinical phenomenon as such, but also enables us to perceive its true significance.

The question naturally arises here, just as when we were considering the theory of uræmia, what substances are the chief occasion of the rise in arterial tension. This question, however, can not at present be answered. We can merely say that the results of experiment indicate that urea probably does not play the most essential, and certainly not the sole, part in the matter. The question whether the hypertrophy of the left ventricle associated with nephritis is mainly simple (concentric), or whether it may not sometimes be accompanied by dilatation of the cardiac cavities (eccentric), we do not regard as of any special importance. It should, however, be noted that while the hypertrophy always affects the left ventricle chiefly, and often it alone, yet not infrequently the right ventricle is also found to be hypertrophied. If we assume that there is a direct irritation of the myocardium (*vide supra*), this condition will not seem remarkable. Otherwise, the hypertrophy of the right ventricle might be regarded as a compensatory hypertrophy, supplementing failure of the left ventricle.

The relation between certain diseases of the vessels and diseases of the kidneys will be spoken of in the chapter on contracted kidney.

CHAPTER II

ACUTE NEPHRITIS

(Acute Bright's Disease)

Ætiology.—Acute nephritis, like most of the other forms of nephritis, is not a disease whose ætiology is uniform. The same anatomical change, which we term "nephritis," and which is attended by about the same morbid phenomena, may be excited by influences of very different kinds. Almost all these influences have one thing in common, namely, that, as we have stated in the preceding chapter, they reach the kidneys by way of the circulation and are here in part eliminated, and thus exert their specific injurious action upon the parenchyma of the kidneys; but they differ considerably from one another in their precise chemical nature. Since the pathological change in the kidneys depends upon the amount of the noxious material, upon the intensity of its action and the duration of its influence, we see that the cases of nephritis that arise in this way must present a perfectly continuous series from the mildest to the severest, from those that pass off rapidly to those that last perhaps for years and years. The history of renal pathology teaches us in the plainest way that all attempts to divide the forms of nephritis into different clinical and pathological "varieties" can not be strictly carried out. The more scientific experience increases, the more numerous must be the forms established, and still we only too frequently have to assume all sorts of "transitional forms" merely to bring the reality into harmony with the scheme. It therefore corresponds merely to our practical needs if we take certain types from this whole list and divide nephritis into various groups; for, from the nature of the case, there can be no question of a sharp separation of the various forms.

We accordingly call those inflammatory renal affections acute nephritis which arise comparatively rapidly from any of the injurious influences soon to be enumerated, and which terminate, after a few days or a few weeks, either fatally or with recovery; or occasionally, after a rapid onset of this sort, pass gradually into a chronic form. Acute nephritis, on the one hand, follows immediately, without any fixed boundary, the mildest morbid changes in the kidney, which are usually not termed actual nephritis, but simple "parenchymatous degeneration"; while on the other hand it shows a continuous transition to those forms which last for several weeks or months, or longer, and hence are called subacute or subchronic nephritis.

Let us now consider more minutely the conditions under which acute nephritis develops. In the first place, it occurs with very great frequency as a sequel or complication of all sorts of infectious diseases. We may assert that there is really no acute infectious disease in which a secondary acute nephritis may not occasionally appear. There are, however, many diseases in which this complication is peculiarly frequent and characteristic. The exact causes of its development are as yet imperfectly known. Formerly, authorities were inclined to assume that the original germs had directly invaded the kidney itself, but probably this is true in only a few cases, if we except genuine metastatic pyonephritis. According to the views of the present day, it is much more likely that abnormal chemical substances, toxines, which develop in the body under the influence of the infection, reach the kidneys and excite disease in them. The amount and character of these substances determine, of course, the severity of the renal disease. Since we have already dwelt upon the occurrence, the frequency, and certain peculiarities of secondary nephritis in the description of the different infectious diseases, a brief recapitulation of the facts, which have already been for the most part discussed, will suffice here.

The infectious disease which most frequently gives rise to an acute nephritis is scarlet fever. As has been shown previously (see page 43), the renal affection appears but rarely at the beginning of the disease, and then in a very mild form, while the special severe scarlatinous nephritis usually attains its development only toward the end of the third week of the disease. In measles, secondary nephritis is very much rarer than in scarlet fever; in rötheln it is only of very exceptional occurrence. It is commoner again in small-pox, especially in the severe hæmorrhagic forms. In varicella, renal affections are very rare, but they have been occasionally observed. They are always of but slight intensity. In typhoid fever a slight albuminuria is very common, but genuine acute nephritis is quite rare. There are some cases, however, where a nephritis appears very early, and where the other typhoid symptoms are so crowded into the background by it that we have decided difficulty in making the diagnosis of typhoid; this is called the "renal form of typhoid fever." In typhus and recurrent fevers severe cases of nephritis are not especially common, but they are seen more frequently than in typhoid fever.

The nephritis that often comes on in cholera is of great practical importance. This is seen in the earlier stages, and is especially one of the most frequent causes of the so-called cholera typhoid (see page 89). Of course it may appear questionable whether the renal affection here is always of a genuine inflammatory nature, or develops only in consequence of the disturbance of circulation.

Nephritis develops quite frequently in the course of diphtheria, especially in severe cases of this disease; but the renal affection only rarely reaches a high degree. We sometimes see, however, very severe forms of nephritis in all the so-called septic diseases (septic nephritis, see page 120), in acute ulcerative endocarditis and endocarditis verrucosa, and allied affections, such as puerperal fever, septic wounds, etc.

Erysipelas also is very often associated with nephritis, and likewise croupous pneumonia; while, on the other hand, acute articular rheumatism is seldom accompanied by albuminuria. It is not very exceptional for sore throat, particularly follicular tonsillitis, to occasion acute nephritis, and the same is true of acute intestinal diseases. It is of great practical importance to know that a severe secondary nephritis may follow primary diseases of this sort, even if the latter seem to be very mild.

We ought also to speak of the acute nephritis sometimes seen in patients with pustular eruptions, such as impetigo, pustular eczema, severe scabies, etc. Probably we have to do in most of these cases with a mild septic infection, which has found ingress through one of the many minute scratches or other lesions of the skin. We must also bear in mind the possibility (*vide infra*) of damage done by external applications which are absorbed by the skin.

There are many chronic infectious diseases besides these acute ones, in the course of which acute nephritis may appear. We see this complication most frequently in the case of primary tuberculosis, but it seems to us highly probable that the nephritis in this instance is not directly connected with the tuberculosis, but is the result of absorption of septic matters from the pulmonary cavities and similar places. Syphilis should also be mentioned in this connection. We have ourselves repeatedly seen mild and even severe acute nephritis develop in the secondary stage of syphilis. Finally, malaria might be mentioned, although the nephritis which occurs in this connection usually takes a chronic form.

Besides the infectious forms of nephritis just described, there is a second great group, which may be classed under the general heading of toxic nephritis. In these cases we are dealing with the deleterious action of definite chemical substances which enter the body from without and are excreted from it by the kidneys. It is wholly impossible to enumerate all the substances which have this injurious effect; we will therefore confine ourselves to mentioning those of the greatest practical importance. Among the poisons proper we may mention the mineral acids, sulphuric, hydrochloric, and nitric acids, oxalic acid, phosphorus, arsenic, lead, mercury (corrosive sublimate), and chromate of potassium. Among remedies used internally, which may excite nephritis when given in too great doses, we may mention cantharides, squills, balsam of copaiba, turpentine, salicylic acid, and chlorate of potassium. This last causes hæmoglobinuria as well as nephritis. It is also very important to know that many remedies applied externally are absorbed by the skin, and that in this way they may reach the kidneys and excite severe changes there. Among these are cantharidal plaster, preparations of tar, petroleum, styrax, naphthol, and pyrogallic acid. We must mention, in addition to these, the nephritis which may arise from the too abundant use of carbolic acid or iodoform on the surface of open wounds. Under some circumstances renal affections may even arise in individual cases from taking excessive amounts of certain foods and drinks, such as spices, alcohol, horse-radish, or very acid foods.

The many cases of nephritis thus far discussed are directly referable to some definite cause, but in a comparatively small number of instances acute nephritis appears as an apparently primary disease in persons previously healthy. In many such cases we are absolutely unable to make out any causative factor, while in others, again, we do find certain unfavorable influences affecting the patient, such as hard drinking or severe labor in the open air. It can not be doubted that exposure to severe cold and thorough wetting promote the development of acute nephritis. The author's experience has convinced him of this fact. Thus, the disease may occur when a man works in the snow or in cold water. We possess as yet, however, scarcely any knowledge of the conditions which bring about this result. Many investigators assume that cold interferes with the excretory function of the skin; others imagine that the blood is damaged as it circulates in the cutaneous vessels. We have repeatedly seen cases of apparently primary acute nephritis in great beer drinkers. We suppose that in such cases the effect of the long-continued chronic intoxication with alcohol is cumulative, and finally leads to the apparently spontaneous acute development of nephritis (acute alcoholic nephritis). Sometimes, however, even in such cases there seems to be some other factor, such as catching cold, which gives the final impulse to the development of the disease. A considerable number of cases of apparently primary acute nephritis must, however, be regarded in the last analysis as the results of a septic infection of some sort. If we question the patient closely, we learn in such cases that the appearance of the nephritic phenomena has often been preceded by symptoms of a mild sore throat which scarcely attracted the patient's attention, or an insignificant gastro-intestinal disturbance, etc. Such statements are indicative of the possible place of infection. Again, slight purulent affections of the skin, such as furunculosis, phlegmon of a finger or toe, and eczema may sometimes furnish a gate of entrance for infection. In considering all cases of apparently primary idiopathic and acute nephritis, it is very important to remember that not infrequently a chronic nephritis may exist for a long while, perhaps without any symptoms whatever, and suddenly flame up into an acute disease (acute recurrent nephritis of Wagner), and thus simulate a primary acute nephritis. We can not get at the correct interpretation of such cases without careful inquiry into the previous history, and sometimes only after observing the subsequent course of the disease.

In conclusion, we have an especial form of acute renal disease to mention in this connection—viz., the nephritis of pregnancy (*nephritis gravidarum*). This does not usually appear until the last months of pregnancy. It may attack women whose health was previously perfect, and it is much more frequent in primiparæ than in multiparæ. The precise causes of the nephritis of pregnancy are very obscure. The earlier explanations, according to which the renal disturbance was occasioned by pressure of the pregnant uterus upon the renal arteries, the renal veins, or the ureters, are entirely unsatisfactory. Perhaps there are special toxic influences, the origin and nature of which are as yet entirely unknown.

Pathological Anatomy.—The anatomical changes of acute nephritis show a continuous series from the mildest to the severest degrees, according to the intensity of the injurious action. The mildest changes, which, as we have said, are not called actual "inflammation," but usually simple parenchymatous degeneration, affect exclusively the parenchyma of the kidney—that is, the epithelium—while the interstitial tissue, the connective tissue, and the vessels remain perfectly normal. This fact is of prime importance, since it implies that, in almost all the injurious influences acting on the kidneys, the specific renal parenchyma itself is diseased first and before any other. On macroscopic examination, the kidneys of "parenchymatous degeneration" may show scarcely any plainly perceptible changes, but it sometimes strikes the practiced eye that the kidneys are a little enlarged, that the cortex on section shows either a more reddish-gray, dimmed coloring (cloudy swelling), or a more grayish-white, yellowish hue (fatty degeneration). The microscopic examination gives more accurate information as to the degree and the extent of the disease. We distinguish different conditions according to the form of change in the epithelium, of which the three following are most important: 1. *Cloudy Swelling:* It is most easily made out in the epithelium of the cortical tubules, but it may also be seen in the epithelium of the glomeruli. The cells swell, their contents become uniformly granular and cloudy, the nuclei swell, and finally disappear. Such changes are often found in acute infectious diseases, such as typhoid, small-pox, and diphtheria. 2. *Fatty Degeneration:* This may proceed from the cloudy swelling, or may develop independently. Many fat-drops appear both in the cells of the uriniferous tubules and also in the epithelium of the glomeruli, and they may finally lead to the disintegration of the cells. Simple fatty degeneration of the kidneys is sometimes found in acute infectious diseases, after certain poisons, such as phosphorus, and finally in anæmic conditions. 3. *Necrosis of the Renal Epithelium:* The nuclei of the cells disappear, and the cells are changed to clear homogeneous flakes, while in some cases they are greatly swollen ("dropsical degeneration" of Nauwerck-Ziegler). Genuine epithelial necrosis is found in the kidneys, chiefly after the action of toxic substances—cantharides, the chromic and chloric salts, etc. —but sometimes also in infectious diseases. Combinations of simple necrosis with granular cloudiness and fatty degeneration are not infrequent. Both the last-named states may undergo resolution if they have not reached a high degree. Otherwise all the degenerations mentioned lead to the destruction and disintegration of the cells; nevertheless, a complete restoration is possible, from the regeneration of new epithelial cells from epithelium that is still present.

We term those changes in the kidneys genuine acute nephritis, in which not only the renal parenchyma proper, the epithelium, but also the interstitial tissue, especially the vessels, is affected; so that we can make out the exudative changes characteristic of all inflammatory processes—the escape of fluid and cells from the vessels. In these cases the different histological processes may be combined in the most varied ways, so that the anatomical picture presents quite great variations, although it is almost always seen on analysis to display the same processes.

If we begin with the histological lesions in acute nephritis, in order to learn to recognize at once the essential changes, we have first precisely the same processes of degeneration in the epithelium which have been already described, but they are usually present here in a more marked degree. In some cases the simple necrotic processes predominate; in others, the fatty degeneration. We often find degenerated cells, and not infrequently a more or less marked desquamation of epithelium. We see also the special inflammatory changes. We find a fluid inflammatory exudation, rich in fibrine, and therefore soon coagulating in the interstitial connective tissue, which is dilated and swollen by it—inflammatory œdema. The same exudation is also found in the uriniferous tubules, and, by the proper methods, by alcohol or by boiling the fresh kidney, the albuminous effusion can be made out both in the capsules of the glomeruli and in the uriniferous tubules. The interpretation of the exudation is, of course, made very difficult, or often wholly impossible, by the presence of albuminous urine in the uriniferous tubules. The second characteristic of inflammation, the "cellular exudation"—that is, the emigration of white blood-corpuscles—is also present. In the interstitial tissue we find accumulations of round cells, usually distributed in foci, and white blood-corpuscles in greater or less numbers also enter the interior of the uriniferous tubules. We often find many hyaline casts in the lumen of the straight tubules or of Henle's loops, whose origin is, in all probability, connected with the albuminous exudation and the emigrated white blood-corpuscles (see page 593). The vessels themselves are often hyperæmic and dilated, but in some cases they are compressed by the interstitial inflammatory œdema. It is of special significance that in very many cases there are hæmorrhages, either into the interstitial tissue or into the interior of the uriniferous tubules, or even into Malpighi's capsules.

Special care has been recently devoted to the study of the minute changes in the glomeruli, although at present there is a conflict in the views regarding them. Klebs called attention to the fact that in scarlatinal nephritis the lesions may be almost exclusively confined to the glomeruli (glomerulo-nephritis). Sometimes kidneys of this sort seem almost normal to the naked eye, although during the life of the patient there have been the severest symptoms, such as anuria and uræmia. Friedländer, Ribbert, Langhans, and others have pursued the study of the histological changes in the glomeruli. In part there are degeneration, hyperplasia, and desquamation of the glomerular epithelium; in part changes in the walls of the blood-vessels themselves. With regard to the secretion of urine, and, in particular, the development of albuminuria, these changes in the glomeruli are certainly of the greatest importance, and it is not improbable that in many cases of acute nephritis the disease begins chiefly in the glomeruli; but, on the other hand, it is not proper to suppress too completely the other changes in the renal parenchyma as compared with the glomerulo-nephritis.

In regard to the extent of all these changes which have been described, it should be noted that not infrequently certain portions of the kidney are more severely diseased than others, so that sometimes the nephritis tends to develop in separate foci. In general, however, acute nephritis is diffuse.

If the histological processes have been made clear, the understanding of the macroscopic appearance of the inflamed kidney is very simple. We can understand that either this or that "form" of acute nephritis must be present according to the predominance of this or that histological process. If an abundant interstitial exudation is present, the kidney is much enlarged; if this exudation is slight, the kidney varies but little, or not at all, from its normal size, notwithstanding any other severe changes. In the first case it usually feels soft, from inflammatory œdema; in the second case, it is comparatively firm. If there is a marked hyperæmia of the kidney, it appears much reddened; if the kidney is anæmic, it is paler; and if an extensive fatty degeneration is also present, it is yellowish-white or yellow. If hæmorrhages are present, they can easily be recognized with the naked eye on the outer surface beneath the capsule as dark-red points that can not be wiped away. We speak then of an "acute hæmorrhagic nephritis." On section, the medullary substance is more or less dilated, often projecting somewhat above the general level of the cut surface, its normal striated appearance is almost always obliterated, and its color shows the same variations as the outer surface of the kidney. Not infrequently the diseased Malpighian corpuscles can be recognized by the naked eye as grayish-red or whitish points. Since, as we have said, the nephritic changes often show not a uniform and diffuse, but a nodular arrangement, we can understand that the kidneys sometimes have quite a mottled appearance, since hyperæmic or hæmorrhagic red spots alternate with the lighter anæmic and the yellow fatty-degenerated parts.

There are, accordingly, cases of nephritis which show almost nothing abnormal to the naked eye, while, on the other hand, there are hæmorrhagic and non-hæmorrhagic forms, appearing pale, yellow, red, or variegated, none of which can in the essential features be separated from one another, but which are combined with one another in all conceivable ways. The forms of nephritis that differ in ætiology have, to a certain degree, definite and characteristic anatomical types, but strict rules can not be laid down in regard to this.

Clinical History.—The most essential symptom of acute nephritis is the abnormal character of the urine. In most of the milder, and even in many of the severer cases of nephritis, the change in the urine is the sole objective clinical symptom which renders the diagnosis possible. The physician must, therefore, make it his practice to submit the urine to repeated examinations in every case of disease where there is any possibility of the presence of a nephritis.

The simple parenchymatous degeneration of the kidneys, cloudy swelling, fatty degeneration, etc., may probably sometimes exist without being followed by any discoverable change in the urine; but they often lead to a slight albuminuria, which is easily explained from the change in the epithelium of the glomeruli. If, then, the urine contains a slight amount of albumen (which usually soon passes off) in the course of any febrile infectious disease or other affection (the so-called febrile albuminuria, etc.), we are justified in assuming some of these mild conditions of degeneration in the kidneys. Usually the urine shows no other peculiarities, but sometimes we find in the sediment a few hyaline casts, a few white blood-corpuscles, etc. As we have repeatedly stated, these conditions pass into nephritis proper without any sharp limitations.

Character of the Urine in Acute Nephritis.—In almost every case of severe nephritis the amount of urine for the twenty-four hours is more or less diminished. This is caused directly either by the lessened elimination of water by the kidneys, or by the plugging of many uriniferous tubules by casts, desquamated epithelium, etc. The amount evacuated daily is often only fifteen or twenty ounces (400–700 cubic centimetres), but it sometimes falls to a much lower figure, two or three ounces (100–50 cubic centimetres), and there may finally be even complete anuria. In general, though not without exceptions, the diminution of the amount of urine runs parallel to the severity of the anatomical changes in the kidney. Improvement in the disease is very often first seen in an increase of the amount of urine. If there was a previous œdema, and this is absorbed, the daily amount of urine often rises during convalescence to a very considerable quantity, eighty to a hundred ounces (2,500–3,000 cubic centimetres). Even in cases in which there had been no decided œdema, we have often seen during recovery from acute nephritis very abundant diuresis. In so far as this symptom did not relate to the excretion of water that had been retained in the system, it might perhaps be referable to an abnormal permeability of the glomeruli, similar to the condition sometimes seen after a severe attack of typhoid fever.

The specific gravity of the urine is at first usually increased, since the urine is poor in water, but comparatively rich in solid constituents, especially in albumen (*vide infra*). Of course there are great differences here, and a urine secreted in an abnormally small amount may show a specific gravity of only 1010 or 1015, while, on the other hand, urines with a specific gravity of 1020 to 1030, or even more, have been observed. If during convalescence a very abundant, watery urine is passed, it of course usually has a low specific gravity, about 1005 to 1008.

In many cases, but of course not in all, we may suspect the abnormal character of the urine from its appearance. This depends chiefly upon an admixture of abnormal morphological constituents. If these are present in large numbers, as is usually the case, the freshly passed urine is cloudy, and deposits a more or less abundant floccular sediment. The appearance of the urine is most altered if blood be mixed with it (hæmorrhagic urine). According to the amount of blood, the urine is a light or dark red, or even a dark black-red, and often has a greenish reflection when the light falls on it.

The microscopic examination of the sediment gives more accurate information upon the different morphological constituents. We can not, of course, enumerate all the possibilities that may exist (see page 592 *et seq.*). In general, it may be said that in most of the severe cases of acute nephritis the urine contains numerous casts of the hyaline and other varieties. As has been already mentioned, the epithelial casts and the firm, waxy casts are especially common in the severer forms of acute nephritis. The hyaline casts have usually adherent to them red and white blood-corpuscles, as well as epithelium, fat-drops, and detritus. These morphological constituents are also found free in greater or less amount. The sediment is apt to contain also numerous beautiful crystals of uric acid.

Individual cases are often characterized by a striking predominance of some one constituent—epithelium, white blood-corpuscles, or red blood-corpuscles—but no special rules in regard to this can be given. We have spoken previously (page 594) of the special conclusions we can draw from the different objects found in the sediment. We can accordingly distinguish an acute hæmorrhagic, or non-hæmorrhagic, an acute desquamative, and a fatty degenerative nephritis, but we must always bear in mind that all these forms pass into one another without sharp boundaries.

The chemical examination of the urine gives, as the most important and constant result, usually a considerable amount of albumen. Since the reaction of the urine is almost invariably acid, the albumen is immediately precipitated on heating the urine, and sinks to the bottom of the test-tube, where it usually takes up about one half or three fourths of the volume of urine used for the heat-test. More accurate quantitative determinations of albumen give most frequently in acute nephritis an amount of albumen of from three tenths to one per cent.; higher percentages are rare. The daily total amount of albumen eliminated amounts to about one or two drachms (grammes 5–8), or sometimes more, but the daily loss of albumen from the body hardly ever exceeds the amount of five drachms (grammes 20). The variation in the amount of albumen eliminated in different cases is quite noticeable.

The examination of the other solid constituents, which is not generally employed in practice, usually gives a diminished secretion of urea, phosphoric acid, etc., corresponding to the diminution in the whole amount of urine.

THE OTHER SYMPTOMS OF ACUTE NEPHRITIS.—Local symptoms in the kidneys themselves are only rarely present. There is, of course, a certain tenderness in the region of the kidneys, which, however, is too ambiguous to have very great symptomatic importance. It is more often the case that the abnormally concentrated urine causes frequent micturition, associated with a disagreeable burning sensation—a sort of vesical tenesmus.

The subsequent symptoms of acute nephritis, which appear in the rest of the body, and among which dropsy takes the first place, are far more important than the local symptoms. Œdema may be entirely wanting in acute nephritis, particularly in cases of secondary nephritis after pneumonia, erysipelas, diphtheria, and usually in cases of septic nephritis. On the other hand, œdema is especially characteristic of many cases of acute nephritis, including cases due to scarlet fever, to catching cold, to alcoholic excess, and to pregnancy, as well as the so-called primary nephritis and the nephritis which follows cutaneous diseases, such as scabies or pustulous eczema. This œdema is not infrequently the most prominent clinical symptom. We must always be prepared for its appearance, especially when the amount of urine shows a considerable and persistent diminution.

The œdema is usually discovered first in the face, which has a bloated, and often a pale and somewhat shiny appearance. The eyelids are usually most swollen at first. Besides the face, the ankles, the legs, the scrotum, and the dependent parts of the trunk may be the chief seat of the œdema, the severity and extent of which may of course vary greatly in different cases. If a high degree of general dropsy develops, this is a source of great distress to the patient. The movements of the body are much restrained, and all changes of position are difficult, associated with great exertion, and painful. In the severest degrees of dropsy small fissures may form here and there in the excessively tense skin, from which the dropsical fluid oozes. Such little wounds are sometimes the starting-point for disagreeable erysipelatous inflammations, etc.

If great œdema of the skin is present, we usually find at the same time a more or less marked dropsy of the serous cavities. It is often hard, however, to make out ascites or hydrothorax on physical examination, owing to the œdema of the skin that is present. The symptoms mentioned acquire their chief clinical significance from the difficulty of respiration necessarily associated with them, since the diaphragm is pressed upward by ascites, and the lungs are compressed by hydrothorax. If a hydrothorax is more marked on the left, or especially if hydropericardium sets in, the activity of the heart is materially impaired.

A marked œdema of the mucous membranes develops but rarely; in a few cases we have seen œdema of the conjunctivæ, œdema of the soft palate, and œdema of the glottis. Of the œdemas of internal organs, œdema of the brain has already been mentioned as a possible cause of severe nervous uræmic symptoms. Œdema of the lungs, which often comes on toward the end of the disease, when it terminates unfavorably, is usually not to be regarded as a part of the general œdema, but as a result of the final cardiac weakness.

In regard to the other symptoms in the different organs, the symptoms on the part of the circulatory apparatus must first be mentioned. The pulse is often abnormally tense, hard, and full (see page 600). In the beginning of the disease it is often somewhat slow; later it is usually accelerated. A beginning cardiac hypertrophy can often be made out post mortem, and sometimes clinically, in cases which have lasted a rather long time, four to six weeks. It seems to develop most rapidly in children who were previously well and strong. We pay especial regard to the condition of the apex-beat, and to the accentuation of the aortic second sound. The occasional nose-bleeds are probably connected with the increased arterial tension. Pericarditis is seen as a rare complication—a complication which is connected with the general fact that in all forms of nephritis the different internal organs, especially the serous membranes, have a tendency to inflammation. Whether this circumstance is connected with the retention of urinary constituents, as has been repeatedly surmised, can not at present be decided with certainty.

Of the symptoms in the respiratory apparatus, we have mentioned above the dyspnœa consequent upon the dropsical symptoms. In severe cases the lungs themselves are often drawn into sympathy, as shown by the development of a diffuse bronchitis or a peculiar form of pneumonia. The latter stands midway between a catarrhal and a croupous inflammation. It exhibits, to a certain degree, a form of stiff inflammatory œdema, and occurs in just the same way in the chronic forms of nephritis as in acute nephritis. When it involves both lungs to a great extent, it may be the immediate cause of death. The development of general pulmonary œdema is usually a sign of beginning weakness of the left ventricle, as we have said above, although here, too, inflammatory factors may have some influence.

Vomiting is the most important symptom in the digestive apparatus. If it appears in a marked degree, it may almost always be considered as a uræmic symptom, and then is often the precursor of severe nervous symptoms. The appetite is almost always diminished in acute nephritis; the bowels are usually constipated, but there may be quite severe diarrhœa (see page 600). We may mention peritonitis, which is sometimes purulent, as a very rare complication (*vide supra*).

The temperature is markedly influenced by acute nephritis only in those cases where the disease develops in previously healthy persons, or at least in those free from fever. Then we see quite frequently a moderate fever, with an irregular rise of temperature of about 100° to 102° (38°–39° C.). It is quite rare that an apparently primary acute nephritis begins suddenly with a chill and high fever, 104° (40° C.). The condition of the temperature on the onset of uræmic symptoms has already been described (page 600).

The state of the general nutrition suffers in quite a noticeable degree in most of the severe cases of acute nephritis. The emaciation is often concealed by the œdema; but the anæmia is the more prominent, and often lends to the bloated face a peculiar aspect.

Uræmic symptoms may come on at any time in the course of acute nephritis. We are often prepared for the onset of uræmia by a previous marked decrease in the secretion of urine, or by the well-known prodromal symptoms, but in other cases it begins very suddenly with severe symptoms of eclampsia. In regard to all further details we may refer to what was said on page 596 *et seq.*

THE COURSE AND DIFFERENT FORMS OF ACUTE NEPHRITIS.—The whole clinical picture of acute nephritis depends very materially upon the form of its development. If an acute nephritis comes on in the course of a severe infectious general disease, as in the course of a septic affection, of ulcerative endocarditis, or of severe typhoid, the changes in the urine are often the sole indication of the occurrence of the complication. The type of the severe febrile general disease is in no way materially modified by the added renal affection; œdema and uræmic symptoms do not usually appear, often because the primary disease soon ends in death.

Also when nephritis comes on in previously healthy persons or in chronic invalids, the tuberculous, etc., in many cases the changes in the urine are the chief symptom, while the other general and secondary symptoms are scarcely evident at all, or at least only in a very slight degree. Such mild cases are associated only with more or less general dullness and loss of appetite. Œdema is entirely absent, or present only to a very slight degree. Of course such cases demand great caution, since even in them we may have a sudden outbreak of severe uræmic symptoms.

The fully developed type of severe acute nephritis is seen especially in scarlatinous nephritis (*q. v.*), which comes on in children who are fully convalescent or apparently wholly well; it is also seen in many cases of apparently idiopathic nephritis (*vide supra*), or nephritis coming on after exposure to cold, etc. In these cases there is often the development of a general dropsy, secondary pulmonary affections, uræmic symptoms, the symptoms mentioned in the circulatory apparatus, etc. In these cases, too, the examination of the urine affords the only certain means of judging accurately of the condition, but the other morbid symptoms which appear early—œdema, anæmia, and vomiting—may direct our suspicions to the developing renal affection.

Scarcely any general statements can be made as to the course and the duration of acute nephritis, since the variations in this respect are too great. To describe here in particular all the different forms of nephritis according to the ætiological conditions in question would lead us too far. We will therefore refer to the description of the different primary diseases, in which the characteristic marks of any renal complication are always stated. A few remarks must be added about some of the simple forms of acute nephritis.

The so-called PRIMARY IDIOPATHIC NEPHRITIS usually appears rather suddenly without any demonstrable cause, or it may follow a marked chill ("nephritis due to catching cold"). The first symptoms of the disease are sometimes insignificant, but at other times they are quite severe—chills, fever, renal pain, etc. Sometimes other "rheumatic symptoms," such as angina or articular pains, are also present. Often the first thing which attracts the attention of the patient is the occurrence of œdema, the puffiness of the face, or slight shortness of breath due to hydrothorax. Sometimes the first symptom is vomiting. The further course of the illness may be mild or severe. In the former case the œdema that has appeared is but slight, the changes in the urine (albuminuria, hæmaturia, etc.), do not attain a very high degree, and after a few weeks complete recovery ensues. In other cases, however, the type of a severe, acute, and very often hæmorrhagic nephritis develops, with great general dropsy, uræmia, etc., which in three or four weeks, or sooner, may lead to death; but improvement may follow in spite of the severest symptoms. Then the amount of urine gradually increases, and the abnormal constituents of the urine, the œdema, and the other morbid symptoms, gradually disappear. Of course, it is often a long time before complete recovery ensues, since, even when the patient feels perfectly well subjectively, the urine may still sometimes contain some albumen, a few casts, or a few red blood-corpuscles. In such cases one must think of the possibility of a transition of acute into chronic nephritis. Not infrequently there is apparently complete recovery, but several weeks or months later there appears suddenly, perhaps after some special cause, such as catching cold, or over-exertion, or some febrile disease, a fresh attack of acute nephritis ("acute recurrent nephritis," *vide supra*).

The NEPHRITIS OF PREGNANCY usually begins gradually. Frequent micturition and œdema of the lower extremities make their appearance, and beside these there are often nausea and even vomiting. If we examine the urine, we usually find it quite rich in albumen, but comparatively poor in morphological elements. The slight sediment consists of hyaline casts, a few white blood-corpuscles, and some epithelium. Only rarely does the urine assume a hæmorrhagic character.

The condition described almost always lasts to the end of pregnancy. In the cases that proceed favorably a very rapid recovery often follows after the birth of the child; but the onset of *eclampsia gravidarum* is to be dreaded as a not infrequent and a dangerous complication. This is to be regarded as entirely analogous to uræmia. It begins after mild prodromal symptoms, or even quite suddenly, with violent general convulsions, during which the child is usually born. A more or less persistent coma follows the convulsions. The convulsions may be very frequently repeated. Death ensues in about one third of the cases; the other cases usually recover, only rarely passing into chronic nephritis. A slight albuminuria may, however, persist for months after delivery. The prognosis is still more unfavorable for the child than for the mother, inasmuch as the child dies in nearly one half of the cases.

Moreover, it should be noted that eclampsia sometimes occurs without any preceding albuminuria, and perhaps of itself leads to a mild albuminuria because of the circulatory disturbances incident to the convulsive seizures.

The anatomical changes in the nephritis of pregnancy are hardly ever very striking to the eye. The kidneys are usually pale and but little enlarged. Under the microscope we usually find a slight interstitial œdema and degenerative changes in the epithelium. Only rarely are more marked nephritic appearances present.

ACUTE ALCOHOLIC NEPHRITIS is a term which we apply to that form of the disease which we have repeatedly observed in excessive beer drinkers—for instance, in brewers. The appearance of the disease seems to be favored by coincident exciting causes, such as catching cold, but beyond this it is to be regarded as the sudden development of a renal disease, resulting from the toxic influences of alcohol habitually ingested. There is a rapid and great development of general dropsy. The amount of urine is moderately diminished. The urine contains much albumen, but usually remains rather clear and contains no blood. As a rule, the patients are obese, and their discomfort is great. A favorable termination is possible under suitable treatment, but yet there is always danger of a transition into chronic nephritis.

Diagnosis.—Acute nephritis can be overlooked only when the examination of the urine is neglected or when it is impracticable. The latter sometimes happens, for example, when the patient does not come under observation until after the onset of severe uræmic symptoms. Otherwise, however, the changes in the urine always furnish evidence enough to recognize the existence of the affection of the kidneys. We can, of course, decide that the nephritis is acute only by consideration of the history, the ætiological conditions, and the whole course of the disease. We must also bear in mind the possibility that there may be an acute exacerbation in a chronic nephritis that has already existed for a long time, and has been perhaps without symptoms—acute recurrent nephritis, usually hæmorrhagic.

Prognosis.—The prognosis of acute nephritis depends in many cases not only upon the renal affection, but also upon the underlying primary disease. We can not here describe in detail the numerous conditions that must be considered, but they are to be found in the appropriate chapters.

Many cases of primary nephritis from toxic action, or exposure to cold, and also many cases of secondary nephritis after scarlet fever, in pneumonia, typhoid fever, or syphilis, during pregnancy, etc., recover perfectly in a short time or after several weeks, according to the severity of the individual case. On the other hand, however, it must be said that every case of nephritis must be viewed with great caution, partly because it may be the starting-point of a subsequent chronic renal disease, and partly because dangerous sequelæ may sometimes develop in cases that at first are apparently mild. The dangers of acute nephritis are chiefly: First, the appearance of severe general dropsy, especially in the internal cavities of the body. Of the forms of dropsy hydrothorax is the most dangerous, as it may produce suffocation by compression of the lungs. Second, uræmia, especially in its severe convulsive forms, with high temperature and finally cardiac paralysis. Third, the inflammation of internal organs, among which secondary pneumonia, in particular, is a frequent cause of death, while secondary pericarditis and peritonitis, as we have said, are seen in but very few cases. We must bear in mind, however, that in individuals otherwise healthy the severe sequelæ just mentioned may also be recovered from. The most extreme dropsy may be re-absorbed, and we sometimes see recovery, especially in children, after the worst uræmic symptoms.

Treatment.—With regard to prophylaxis, the reader should be reminded that in all diseases which experience has shown to be prone to lead to secondary nephritis—for instance, scarlet fever—the kidneys should be guarded as much as possible, from the start, by suitable diet (milk); by promoting the secretion of the kidneys ("flushing the kidneys"), by administering an abundance of liquids, such as mineral waters, milk, and lemonade; and by stimulating the cardiac activity by warm clothing, warm baths, and rubbing with alcohol.

If nephritis has already developed we can scarcely hope to influence directly the inflammatory process. The remedies recommended for this purpose are tannin, uva ursi, fuchsine, and methyl blue, but to an unprejudiced observer they seem absolutely useless.

We expect as little result at present from "external antiphlogosis" as from the internal remedies mentioned—that is, from local blood-letting, applications of ice to the region of the kidneys, etc. Only in the rare cases where severe pain in the region of the kidneys comes on at the beginning of nephritis, in an otherwise robust individual, are we at present justified in trying leeches or a few dry cups. The warm baths, to be described more fully below, have perhaps an indirect favorable action on the process in the kidneys, since they produce a hyperæmia of the skin, and thus lessen the flow of blood to the kidneys.

Although we must accordingly admit that there is scarcely any remedy at our service which has a direct therapeutic influence upon the diseased kidneys, the treatment of nephritis may nevertheless produce very significant results, since both a number of hygienic measures and the fulfillment of certain symptomatic indications are of the greatest importance.

Among the general hygienic measures we must mention first strict confinement to bed. In the severe cases its necessity is self-evident; but, even in the milder cases, which run their course without any severe subjective symptoms, constant rest in bed is necessary throughout. In this way we not only avoid the unfavorable action of cold upon the external skin, but the activity of the skin, which must act vicariously for the kidneys, is also excited by the uniform warmth of the bed; while any useless muscular exertion, which would tax the heart's capacity for work, is also avoided by staying in bed. In general it is advisable to cover the patient quite warmly, so as to keep him in a constant slight perspiration.

The regulation of the diet is very important. All those foods and drinks which may irritate the kidneys are to be strictly avoided, especially spices, very sour substances, strong tea and coffee, or alcoholic drinks. Milk has for a long time proved itself to be by far the most suitable and best food. It has won for itself the reputation of a remedy in renal disease, and the best results have often been seen from a methodical "milk-cure"—that is, from feeding the patient almost exclusively with milk. The great aversion of some patients toward milk, however, is sometimes an obstacle to its use. We may often be aided, then, by making the milk more acceptable to the patient by the addition of a little coffee, salt, a little cognac, or soda-water. Among other foods to be recommended are buttermilk, milk-gruel with rice or groats, and flour-gruel. We should be very cautious about giving meat as long as there are severe symptoms.

Less harmful are light broths made from pigeons, fowls, or veal, with eggs. In every case of acute nephritis, even when there is dropsy, we regard the abundant ingestion of liquids as advantageous. For beverages, besides milk we may give Seltzer, Wildinger, or Fachinger water, tea, and lemonade. The last is particularly to be recommended. Alcoholic beverages must be entirely withheld, or, at the most, we may allow a little claret. The stronger wines are given only when the heart becomes weak, and even then their usefulness is dubious.

The chief object in the symptomatic treatment consists in preventing the injurious results of the defective elimination of the water and the solid constituents of the urine by the kidneys, or in remedying these results if they have already occurred. This purpose can be attained only by exciting, as far as possible, the activity of other organs which in this respect may act vicariously for the kidneys. The skin deserves the first attention here, through which, by means of the sweat-glands, large amounts of water, and also, to a certain extent, the solid constituents of the urine, which have been retained, may be eliminated. The diaphoretic treatment of renal diseases has, therefore, been generally in vogue for a long time. If the patient's general condition permits, we always begin with it as early as possible, even before there have been any signs of œdema, uræmic symptoms, etc. Formerly we employed, like most other physicians, hot baths from 95° to 105° (36°–40° C.). The patient stays about half an hour or an hour in the bath, is then rapidly dried somewhat, wrapped up in bed in a previously warmed sheet, and well covered up to the neck with blankets. In order to make the procedure somewhat easier for the patient, it is a good plan to cover the forehead with a cold compress, always to wipe the sweat carefully from the face, and frequently to give a little swallow of fresh cold water. The production of sweat is, of course, better excited during the pack if the patient takes some hot drink, hot milk with soda-water, or hot elder-tea. It sometimes seems to aid diaphoresis if an internal diaphoretic be given at the same time, the best being five to ten grains of Dover's powder (gramme 0.3–0.5), or a teaspoonful or two (grammes 5–10) of liquor ammonii acetatis [U. S. P.] (*spiritus Mindereri*) in a cup of elder-tea. We have also found a good rubbing of the whole body with dilute warm French brandy of service before the pack. The pack may last two or three hours. Of late years, particularly in severe cases with marked dropsy, we have almost entirely abandoned baths and in their stead employed with the best results the hot, wet pack, or dry heat (*vide infra*). For the pack, the patient is completely wrapped up in a hot, wet sheet; on both sides are arranged several jugs filled with hot water, and likewise enveloped in moist cloths; then the patient is covered with several dry blankets and is given two or three cups of some hot drink. The duration of a pack of this sort is about two or three hours. Instead of moist heat, dry heat may be applied with the purpose of sweating the patient. There are complicated arrangements, such as sweat-boxes and a patent sweat-bed, but these can seldom be employed outside of hospitals, and we have for a considerable time adopted with good results the arrangement made by Quincke. A tin pipe is bent at an angle and fastened upon a board, and its upper end is introduced under the bed-clothes, which should be, if possible, somewhat raised up, while under its lower open end a lighted alcohol lamp is placed upon the floor; the warm air rises and is conducted beneath the bed-clothes, where the heat becomes so great that the upper end of the pipe must always be wrapped in a cloth wet in cold water.

In this way we succeed in many cases in causing a considerable sweat, so that the patient loses several pounds in weight at each pack, and an existing dropsy may sometimes be made to disappear completely in a comparatively short time. On the other hand, however, we can not deny that it is very hard sometimes to make patients sweat, even when there is œdema of the skin, and also that many patients do not bear hot baths and packs at all. The latter is especially true if the patient has dyspnœa, and if signs of cardiac weakness have already set in. In such cases we must be very cautious about sweating the patient, and we should employ merely warm baths; or if we put the patient in a pack, it should be for a brief time.

Of internal diaphoretics there is one remedy which is particularly useful in renal disease—viz., the hydrochlorate of pilocarpine, prepared from jaborandi. This is best employed subcutaneously, one sixth to one third of a grain at one dose (gramme 0.01–0.02), but the drug may also be given internally in pills in the same dose. Its effect consists in the production of a rather profuse perspiration, and likewise a marked increase in the salivary secretion, which latter is often a great annoyance to the patient. For this reason, and because it sometimes induces cardiac weakness, pilocarpine is, on the whole, scarcely to be recommended. We ourselves employ it only occasionally when the hot baths are not used or when they do not have a satisfactory effect.

Next to the skin, the intestinal mucous membrane is the organ from which we may soonest expect to produce a vicarious elimination of water, and also of urea. It is sometimes, therefore, of distinct service to prescribe drastic cathartics in nephritis with a diminished secretion of urine, especially if there is a tendency to constipation besides the dropsy, dyspnœa, etc. The drastic cathartics chiefly used are infusion of senna, extract of colocynth, gamboge in two-grain powders (gramme 0.1), tamarinds, Epsom salts, etc.

Finally, it may be asked whether we should not excite the secretory function of the kidneys themselves by the exhibition of diuretics. Certainly the employment of vigorous diuretics is opposed by the consideration that we might still further irritate and consequently damage the renal epithelium. The milder diuretics, particularly the acetates of potash and of sodium, in doses of about one to two and a half drachms (grammes 5–10) in the course of twenty-four hours, and diuretine, one to one and a half drachms (grammes 4–6) a day, are usually well borne and are often efficient. The use of calomel in renal disease has been repeatedly commended, but we would warn against its employment because very disagreeable symptoms of mercurial poisoning, such as stomatitis, are apt to occur.

The therapeutic measures so far spoken of are intended to prevent as far as possible the retention of urinary constituents in the body. They are also very much employed when the signs of this retention have already appeared. The dropsy especially is best treated by methodical sweating, with the aid, eventually, of drastic and diuretic remedies. If there is persistent and extreme dropsy we may determine to remove the dropsical fluid by direct mechanical means from the subcutaneous cellular tissue. Better than the so-called capillary trocars and similar apparatus (such as aspiration through a funnel placed like a wet cup upon the incised skin) are simple scarifications of the skin made longitudinally in the dependent parts (scrotum and posterior surface of the thigh and calf). From these the dropsical fluid is allowed to escape into pads made of peat, [gauze], or similar substances. Absolute antisepsis and asepsis are demanded, for there is a great tendency to erysipelatous inflammation.

When uræmia is threatening, and often even when it has broken out, we may try to produce an elimination of the injurious products of tissue metamorphosis from the body in the well-known ways above described, by sweating or drastic purgatives. Beside this, the uræmic symptoms, however, often demand a special symptomatic treatment. If very violent and frequent uræmic convulsions appear, we consider it advisable to try to suppress the attacks by chloroforming the patient. At any rate, it seems to us to be better to use chloroform in uræmia than to give narcotics internally, because with this we can watch the action of the remedy better, especially with regard to the pulse and respiration. Chloroform is also generally used by the obstetricians as the main remedy in the eclampsia of pregnancy. If the attacks are not very frequent, but if there is marked somnolence or coma, tepid baths with cold shower-baths are often employed with distinct advantage. Cool baths are also serviceable when there is a great increase of the temperature. If we are treating a robust individual with a full pulse, and during severe uræmia there is a decided redness or cyanosis of the face, venesection may be indicated. This sometimes has a striking and instant effect, as has lately been confirmed by various observers. Great attention is to be paid to the condition of the heart. As soon as the pulse becomes small and weak, energetic stimulants, such as strophanthus or subcutaneous injections of camphor, must be used. If the signs of cardiac weakness appear before the beginning of severe uræmic symptoms, infusion of digitalis must be used, two or three parts to three hundred of water. Through its action in raising the blood-pressure—it being advisable under some circumstances to combine with it acetate of potassium—free diuresis sometimes comes on, and with it a disappearance of the danger from uræmia; but meanwhile we must watch for symptoms of the toxic action of digitalis. The tinctura nervina Bestuscheffii [nearly equivalent to the tincture of the chloride of iron, U. S. P.] may also be sometimes used to advantage in uræmia. We are not apt to interfere with uræmic vomiting or uræmic diarrhœa, because these symptoms, as we have said, are to be regarded as a form of self-help by the organism. Only when such symptoms are very distressing do we give cracked ice, morphine, opium, cocaine, chloroform, etc. If the vomitus contains ammonia, it is a good plan to give ten or fifteen drops of dilute hydrochloric acid in water several times a day. An abundance of liquids is always to be prescribed upon the appearance of uræmic symptoms, because in this way it is probable that the poisonous blood is diluted and also the excretion of the toxines promoted.

[A method of treatment of uræmic convulsions, whether post partum or connected in no way with parturition, which gives excellent and prompt results, is the administration of pilocarpine hypodermically—grain ¼, and repeat in twenty minutes—followed by the hot-air bath, to maintain the action of the skin after it has once been started. Pilocarpine sometimes acts as a decided cardiac depressant, so one must be ready to administer stimulants—brandy or ether—under the skin if, as is apt to be the case, there is doubt as to the readiness with which they will be absorbed from the stomach or rectum.]

In severe cases, the patient's dyspnœa often demands prompt relief. If the dyspnœa be caused, or at least increased, by hydrothorax, and we do not succeed in removing the hydrothorax in any other way, it is necessary to evacuate it by puncture. In acute nephritis, indeed, we may hope by this means sometimes to preserve the patient's life until improvement sets in. Great ascites must also be relieved by tapping. Against "renal pneumonia" our remedies are powerless. Tepid baths, shower-baths, and wet packs sometimes procure relief. In "uræmic asthma," morphine injections may act beneficially. If pulmonary œdema ensues, the heart again is chiefly to be considered. We may try, besides the remedies already mentioned, large mustard plasters, baths, and acetate of lead.

We accordingly see that many remedies are at our service in the treatment of nephritis, the choice of which in the individual case must be committed to the personal judgment of the physician. In the main, we should always begin with the necessary hygienic measures, and, if possible, with a methodical diaphoretic treatment, and govern ourselves otherwise by symptomatic indications. After recovery has set in, great caution is still necessary for a long time. The patient must guard against physical over-exertion, errors in diet, and exposure to cold. Preparations of iron are to be prescribed when there is a secondary anæmia.

In regard to the influence of the onset of an acute nephritis on the treatment of the primary disease, we may mention that cold baths are in general not to be freely used, as in typhoid fever with nephritis, but still they may be tried if they are otherwise urgently desirable. We would also suggest that certain internal remedies, especially calomel and salicylic acid, must be used only with great caution when there is nephritis. In the eclampsia of lying-in women the induction of premature labor is only rarely indicated, since the child is usually born during the paroxysms without interference.

CHAPTER III

THE SUBCHRONIC AND CHRONIC FORMS OF NEPHRITIS, WITH THE EXCEPTION OF THE GENUINE CONTRACTED KIDNEY

(*Chronic Diffuse Nephritis, Chronic Parenchymatous Nephritis, Chronic Hæmorrhagic Nephritis, Large White Kidney, Secondary Contracted Kidney*)

Ætiology.—While the acute nephritis described in the preceding chapter runs its course in several days or weeks, and only rarely extends over some months, we will now speak of inflammatory degenerative affections of the kidneys which last at least several months, and often go on for a year or two. The term "subacute" or "subchronic" is chosen for the cases that last a comparatively short time. As we must once more repeat, there is no sharp limit in this respect.

In regard to the ætiology of these forms of nephritis, they do arise from an acute nephritis, but this is quite rare. Formerly such an origin was erroneously regarded as the rule, and this is the reason why the changes in the kidney in these cases were described as the "second stage of Bright's disease" (Frerichs). The English clinical observers Wilks and Johnson, whom Bartels followed in Germany, first pointed out the fact that in most cases the disease shows a chronic character from the start, and that we can only exceptionally, as after scarlet fever, recognize an acute "first stage." The name "chronic parenchymatous nephritis," since frequently used, is chosen entirely from practical reasons, inasmuch as it briefly states the distinction from the genuine contracted kidney; but it is incorrect in principle, as will be shown from the description of the anatomical conditions later. It is more correct, therefore, to employ the term "chronic diffuse nephritis," because we have, in fact, to deal with what is essentially a chronic universal or diffuse disease of the kidneys, as contrasted with the "contracted kidney" in which there are always bits of normal tissue interspersed between the numerous separate foci of disease.

If we look for the ætiological conditions in cases that have a chronic course from the beginning, we can often discover nothing definite at all. The disease seems to have developed "of itself" in previously healthy persons. Most probably we have here some toxic or infectious agency that acts on the kidneys, whose detection, however, is at present impossible. In malarial regions the malarial poison may often lead to chronic nephritis. It is also attributed to syphilis and tuberculosis, but the cases met with are usually combinations of these diseases with amyloid kidney (*vide infra*). Frequent exposure to wet and cold, damp dwellings, etc., seem sometimes to be of more material significance. That hard drinkers, particularly beer drinkers, are especially liable to nephritis seems to the author indubitable, because of his personal observations (*vide supra*); but it is, of course, difficult to form a definite opinion on all these points.

Persons in early middle life are most frequently affected by the disease, and men more often than women. In children and old people the disease is quite rare.

Pathological Anatomy.—There is no essential distinction between the anatomical lesions of the kidney in acute and in chronic nephritis. The changes that are seen in both are essentially the same, only they develop and extend more slowly in the chronic forms; and they also, during their longer duration, lead to certain sequelæ in the kidney, which can not develop at all in acute nephritis, owing in part to the lack of time. Even in chronic nephritis the individual cases differ from one another in many respects. First this and then that histological process is especially prominent, and thus lends certain peculiarities to the macroscopic appearance of the kidneys. Certain sequelæ—such as contractions—have also developed but little in many cases that soon end fatally, but they develop far more in other cases of longer duration. Hence it happens that we can quite well regard certain anatomical forms that are more frequently observed as types, although we must never lose from sight the important fact that there is a pathological unity in all these forms and types. Then we shall not lose the clew to the understanding of the morbid process if the individual case does not always harmonize with the scheme of the text-books.

We distinguish the three following chief anatomical types of subchronic and chronic nephritis:

1. CHRONIC HÆMORRHAGIC NEPHRITIS in the form of the LARGE RED or VARIEGATED KIDNEY.—The kidney is at least of normal size, and often a little or a good deal enlarged. It feels firmer than normal; its capsule is often adherent to the surface in some places. The surface looks either uniformly a more gray red or more mottled, while dark-red spots alternate with lighter gray or even yellow spots. The red spots on the surface can not be wholly wiped off, and thus they prove to be hæmorrhages. The gray or yellow parts correspond to the anæmic or fatty-degenerated portions. On section, the cortical substance is usually broader, its normal boundary is obliterated, and its color is a uniform gray red, or mottled and striated.

Under the microscope we find in part the same changes as in acute nephritis—parenchymatous and fatty degeneration of the epithelium, casts or hæmorrhages in the uriniferous tubules, inflammatory œdema or granular infiltration of the interstitial tissue, the capsules of the glomeruli sometimes thickened, the epithelium of the glomeruli sometimes proliferated or desquamated, etc. The special characteristic of this chronic form, in contrast with acute nephritis, is that in many places a complete destruction of the uriniferous tubules has occurred, and that a genuine interstitial connective tissue, more or less rich in cells, has taken their place. In this lies the anatomical evidence of the longer duration of the disease, since the two processes—both the complete atrophy of the epithelium, and especially the secondary proliferation of connective tissue—of course need a certain time for their development. The atrophy and the consequent proliferation of connective tissue usually predominate in some parts, while in others nothing but fresher inflammatory and degenerative changes are perceived.

2. THE INFLAMMATORY FATTY KIDNEY, or the LARGE WHITE KIDNEY (*yellow* would be more exact).—In this form of chronic nephritis the kidney is usually enlarged, or at least of normal size. Its outer surface is smooth and of a yellow or an alternating yellow and gray-yellow color throughout. The broader cortical substance shows a yellow and usually somewhat mottled appearance, while the pyramids almost always appear considerably reddened. Hæmorrhages are also almost always present in this form, usually, of course, in smaller numbers than in the variegated kidney, but they are sometimes quite abundant, as in the hæmorrhagic fatty kidney.

The microscope shows the great affinity between this form of nephritis and the preceding. We have almost precisely the same changes, including always a partial destruction of renal tissue with a subsequent increase of interstitial connective tissue. The macroscopic appearance of the kidney is due to the fact that it is anæmic, and that the fatty degeneration preponderates in the epithelium. It is worthy of note that in these kidneys marked changes in the glomeruli are usually present.

3. THE SECONDARY CONTRACTED KIDNEY.—While in the two forms of nephritis thus far described the outer surface of the kidney is still smooth, and the kidney, on the whole, is somewhat enlarged, we have to do here with kidneys of about normal size, on whose surface there are granulations, which as yet are slight, but which still are already plain. This granulation signifies nothing more than that the destruction of the renal tissue has here advanced farther, and that the newly formed connective tissue has in part undergone cicatricial contraction. These kidneys, therefore, represent a later stage of the two forms first named. They usually come under observation when the nephritis has lasted about a year and a half or two years, or even somewhat longer. The first beginnings of granulation may, of course, show themselves earlier, while, on the other hand, when the process lasts a longer time, a completely contracted kidney may develop.

The color of these kidneys is usually reddish or mottled, the red spots corresponding to the sunken atrophic parts, and the gray or yellowish spots to the elevated parts. Yellow kidneys, however, may also show at times decided granulations. Microscopically, we find already marked atrophy of the renal parenchyma, with a corresponding increase of the interstitial connective tissue.

Formerly these kidneys were called the "transition between the second and third stages of Bright's disease." As follows from the above, they are to be regarded only as a more advanced form of chronic nephritis. Since the kidneys, in spite of their granulation, have on the whole a normal size, we can infer from this, and from the clinical course, that they were previously enlarged. Therefore the name of "secondary contracted kidney" is quite suitable, in opposition to the genuine contracted kidney, which represents a much more chronic form of renal atrophy. In the latter the contraction takes place in an extremely chronic manner in a kidney which was previously normal, while in the "secondary contracted kidney" the cicatricial process develops in a kidney which was previously the subject of diffuse disease.

Of other pathological lesions, apart from the changes in the kidney, we will mention here only the hypertrophy of the left ventricle, which is found (*vide infra*) in all the above-mentioned forms of nephritis. The chronic parenchymatous nephritis without cardiac hypertrophy, assumed by Bartels in his time, does not exist. Such cases were probably instances of unrecognized amyloid disease. Exceptionally it may happen that when a patient with chronic nephritis is very much debilitated and enfeebled, the cardiac hypertrophy is not developed.

Clinical History.—Only in the comparatively rare cases when the renal affection begins acutely, do the symptoms of chronic nephritis follow immediately on the first acute stage. In most cases, however, the disease develops slowly and gradually from the start, as we have said, so that it is usually impossible to determine accurately the moment when the disease begins. Still we may assume that the onset is decidedly more rapid in chronic diffuse nephritis than in genuine contracted kidney.

The first signs of the disease consist of certain general symptoms, pallor, dullness, loss of appetite, nausea and headache, and later of œdema. The latter is often the first symptom which sends the patient to the physician, since in the beginning he is apt to pay little attention to the symptoms first named. The œdema usually appears first in the ankles and legs, more rarely at an early period in the face. It often disappears at first after a night's rest, but always develops afresh during the day, gradually increasing in intensity. The patient himself now sometimes notices a change in the urine, either an abnormal color or cloudiness or a diminished amount. The accurate examination of the urine by the physician first establishes the diagnosis.

In regard to the more special symptomatology of chronic nephritis, we meet exactly the same symptoms as have been described in the preceding chapter on acute nephritis. The characteristic distinction is based merely upon the whole course of the affection and the order of development of the different symptoms, and not upon the symptoms themselves.

The urine almost always is diminished. Of course the figures vary considerably both in different cases and at different times in the same case. A small amount of urine, ten to twenty-five ounces (300–700 cubic centimetres) a day, is almost always an unfavorable sign, while a free diuresis signifies an absorption of the dropsy and an improvement of the condition. There is also a persistent increase in the amount of urine when the chronic nephritis passes into a secondary contracted kidney. Under such circumstances the daily amount of urine may rise to 1,500 or 2,000 cubic centimetres or more.

The specific gravity of the urine is often increased to about 1015 to 1025, corresponding to the amount of albumen and of other solid constituents. It is of course correspondingly lower when there is a more abundant elimination of water by the kidneys.

The amount of albumen in the urine is quite marked in all severe cases, being one third to three fourths of its volume. It amounts to about 1.5–3 per cent. by weight, so that the patient's daily loss of albumen may reach half an ounce to an ounce (grammes 15–30).

The examination of the sediment, which is usually abundant, is of the greatest importance for the accurate determination of the form of the anatomical changes in the kidneys. Above all, the question arises as to the presence or absence of blood in the urine. If abundant, hæmaturia may be recognized by the naked eye from the color of the urine. The detection of smaller amounts of blood can be made only by the aid of the microscope. It goes without saying that the amount of blood in the urine varies considerably in the different cases, and in the same case the urine often contains much more blood during certain periods in the course of the disease than at other times. The portions of urine passed at different times taken separately often show quite marked variation in this respect;

the day's urine usually contains more blood than the night's. From the detection of renal hæmorrhages, of course in connection with other symptoms, we can always make with certainty the diagnosis of a "chronic hæmorrhagic" nephritis.

In most cases casts are quite abundant in the sediment of the urine, but naturally their amount and variety undergo quite great variations in different cases and at different times in the same case. They are the direct sign of the presence of an inflammatory exudative process in the kidneys, although the deposits on the casts are more important for the diagnosis of the special form of renal disease than are the casts themselves. Those formed constituents of the sediment are most characteristic in this respect which point directly to the processes of fatty degeneration in the kidneys: the fatty granules and fatty granular cells, free or attached to the casts. The number of these elements is especially great in the chronic inflammatory fatty kidney, the "large white kidney." The usually clear, non-hæmorrhagic urine may in some cases have even a fatty lustrous surface. Renal epithelium is, on the whole, more rarely present in the sediment in chronic nephritis than in acute, but it occurs in some cases.

Of the other symptoms, the one that usually most strikes the eye is dropsy. It usually come on, as we have said, in the beginning of the disease, and slowly or rapidly reaches a great extent and intensity. A medium or even a high degree of general dropsy may often persist almost unchanged for months. In other cases it shows either spontaneous variations or variations influenced by treatment; it decreases for a time only to increase anew. The severer and more comparatively acute the case, the greater in general is the dropsy. In the more chronic cases, in secondary contracted kidney, its intensity may be slight for a time or even permanently. The dropsy may even be absent in some cases, as we learn especially from the observations reported by Wagner under the name of "chronic hæmorrhagic Bright's disease without œdema" (*vide infra*). In regard to the different localizations of the dropsy, and to dropsy of the internal cavities, hydrothorax, ascites, and hydropericardium, and their results, the same holds true as in acute nephritis.

Of the internal organs, the condition of the heart lays claim to the most interest. In all cases of chronic nephritis, in which we do not have to do with especially weak and run-down patients, who can not save the necessary nutritive material for the formation of a cardiac hypertrophy, we find a pronounced and often a very marked hypertrophy of the left ventricle, either with or without a co-existing dilatation of its cavity. The detection of cardiac hypertrophy during the patient's life is sometimes difficult, especially when there is general dropsy, but the diagnosis can usually be correctly made with proper attention to the abnormally tense radial pulse, the accentuated, valvular aortic second sound, and the displacement outward of the apex-beat, or at least its increased strength. We often find in the cadaver, and can sometimes make out during life, a hypertrophy of the right ventricle (*vide supra*, page 603).

A second important sequel of chronic nephritis consists of the changes in the retina—albuminuric retinitis. Although very rare in acute nephritis, these changes are present in the majority of the cases of this class. Sometimes the patient's subjective visual disturbance (dimness of vision, defects in the field of vision) points to a disease of the retina, but the existence of disease can be established with certainty only by ophthalmoscopic examination. In these cases we find two kinds of changes, in varying numbers and combinations: first, retinal hæmorrhages; and, second, white spots and streaks, especially in the vicinity of the optic nerves. The origin of the spots, which may appear and disappear again, is not yet entirely clear. At any rate, they are circumscribed fatty degenerations of the special retinal elements. The degree of amblyopia depends, of course, chiefly upon the localization of the changes, whether in the macula lutea, or other parts.

We need say little in regard to the other symptoms, since they agree essentially with those of acute nephritis. The general anæmia is very pronounced in many cases, but it is less marked in the very chronic forms. The cerebral symptoms, especially the headache and the mild vertigo, may depend in part upon the cerebral anæmia; otherwise, they are due to uræmia (*vide infra*). Cerebral hæmorrhages have been observed in a very few cases. Hæmorrhages on the inner surface of the dura mater are more frequent, but they are usually without clinical significance. The mouth, larynx, and pharynx usually show nothing particular, except accidentally complicating inflammations. We must, however, remember the occasional occurrence of a very distressing or even dangerous œdema of the soft palate, or of the arytæno-epiglottic ligaments—œdema of the glottis. Forms of bronchitis and pneumonia occur, similar to those seen in acute nephritis. Bronchitis and chronic œdema of the lungs also make their appearance in the more advanced stages of the disease, as a result of cardiac insufficiency. Finally, we must remember the hindrance to respiration from hydrothorax, and also from uræmic dyspnœa. The changes in the heart have already been spoken of. Endocarditis or pericarditis may occur, but they are very rare.

As to digestive disturbances, loss of appetite is a very common symptom. Very persistent vomiting is usually to be regarded as a chronic uræmic symptom. The bowels, as a rule, are constipated, but there may also be severe diarrhœa, as in acute nephritis. In severe cases, especially in the last stages of the disease, ulcerative and dysenteric processes have repeatedly been observed in the large intestine and the ileum. Peritonitis may occur, but it is at all events extremely rare. The liver and spleen usually show no peculiarities.

Uræmic symptoms, both of the milder chronic variety and also in their severest acute form, may come on at any time, although they do not by any means attain their full development in all cases, and are somewhat rarer than in genuine contracted kidney.

The temperature remains normal, as a rule, as long as it is not influenced by complicating inflammations, or by the appearance of uræmia.

Course, Duration, and Termination of Chronic Nephritis.—In general, the whole course of chronic nephritis presents quite a great uniformity. The different symptoms may show certain variations within long periods, but the patient often presents almost the same appearance day after day for months. The duration of the disease varies greatly, from three to six months, in the subacute cases, to two or three years, or even more, in the very chronic cases. The cases of long duration are almost all cases of secondary contracted kidney. They sometimes show in their clinical relations the transition from the enlarged to the granular kidney, since the picture in many of its details is more like that in the genuine contracted kidney: the œdema decreases, disappears completely, or, at least, continues in a lesser degree; the amount of urine becomes more abundant, and the specific gravity and the amount of albumen become correspondingly less. The condition thus lasts for a long time until it grows worse again, through uræmia, or disturbance of the compensation in the heart.

The final termination of chronic nephritis is in most cases unfavorable. In the severe forms death ensues in from three months to a year, either in consequence of general dropsy or from uræmia, from complicating inflammations, etc. The conditions when the nephritis goes on to secondary contraction are comparatively more favorable, inasmuch as the patient may then find himself in a tolerable state, for a time at least. Complete recoveries doubtless occur in chronic nephritis, but they are rare. The longer the disease lasts beyond the first six months the less likely is recovery. The development of secondary contraction may simulate recovery or, at any rate, the subjective condition of the patient may be almost perfect. Even after signal improvement, however, relapses are always to be feared. There are even genuine acute attacks in the course of chronic nephritis.

Different Forms of Chronic Diffuse Nephritis.—Diagnosis.—By means of careful examination of the urine in all suspicious cases—for example, those presenting œdema, anæmia, and similar symptoms—we may in general be certain of making a correct diagnosis of chronic nephritis, but all experienced observers will acknowledge that the diagnosis of the particular form of chronic nephritis is difficult and uncertain, even when the examination of the case is most painstaking. Still, the following schematic *résumé* may at least furnish some indications:

Chronic Hæmorrhagic Nephritis (large variegated or mottled kidney).—Duration from six to eighteen months. Urine hæmorrhagic; usually quite rich in red blood-corpuscles and casts. Œdema. Cardiac hypertrophy. Retinal changes. Quite frequently uræmia.

Inflammatory Fatty Kidney (large white kidney).—Duration also six to eighteen months, but usually somewhat shorter than in the preceding form. Urine not at all, or only slightly, hæmorrhagic. Frequently many white blood-corpuscles, and especially signs of fatty degeneration in the kidneys, fatty granular cells, fat-drops in the urine, etc. Large amount of albumen in the urine. Marked œdema. Cardiac hypertrophy. Very often retinal changes. Death by uræmia frequent.

Secondary Contracted Kidney.—Longer duration of the disease, from a year and a half to three years. At first the symptoms of the preceding forms; later, urine more abundant, less œdema, etc. Death from an increase of the dropsical symptoms due to cardiac insufficiency, uræmia, etc.

Chronic Hæmorrhagic Nephritis without Œdema.—This form has been already mentioned (page 622), but it should receive somewhat further consideration. Up to this time it has been too little regarded, although we ourselves have observed not a few cases of this variety of chronic nephritis. The disease has a rather chronic course. For a long time the patient is but slightly inconvenienced, particularly if he has good care. The urine is usually quite abundant, and contains very little albumen, but has always, or almost always, small amounts of blood mixed with it, and this continues for many months or even longer. From time to time there will appear a more or less considerable hæmaturia, so that the urine has an abundant dark, brownish-red sediment. This is especially likely to occur if the mode of life has been indiscreet, but it may also happen without exciting cause. The urinary sediment is composed of red blood-corpuscles, some of which are entire and some already disintegrated, together with a moderate number of hyaline casts. The latter usually have red blood-globules or granules of hæmatoidine and detritus adherent to them. The amount of urine temporarily diminishes, but it soon increases again. Little, if any, increase in the size of the heart can be demonstrated; œdema is entirely absent, nor have we yet observed retinitis in association with this form of nephritis. Uræmia does occur, but it is rare. The course of the disease is very tedious, as we have said. Whether recovery may take place is uncertain. Usually apparent recovery is followed by the sudden appearance of a fresh hæmorrhage. It is probable that in most cases the final result is a secondary contracted kidney.

With regard to the ætiology of this form of chronic nephritis, it is our opinion that in most cases there is a chronic septic infection or intoxication; at any rate, it is noticeable that such patients not very infrequently give a history of some suppurative process.

Treatment.—The treatment of chronic nephritis corresponds in all its details so closely to that of acute nephritis that we can refer almost entirely to the preceding chapter.

The main thing here also is regimen and symptomatic treatment. The patient must always keep himself warm, wear flannels, or stay in bed. The diet must be unirritating. At first, for a time, we may try giving milk alone; it is always advantageous to give an abundance of liquids, except that alcoholic beverages should be avoided so far as possible. It is well to pay careful attention to the skin—bathing, or sponging with warm water and alcohol. Under some circumstances climatic treatment, in Italy, Egypt, etc., are indicated in the more chronic forms.

The treatment of dropsy follows entirely the methods previously described, and so does the treatment of any uræmic symptoms. If there is persistent hæmaturia, ergotine may be tried, but it seldom seems to do any good.

In the more chronic cases with great anæmia, preparations of iron, such as iodide of iron, are often to be used, and also frequently stomachics, cathartics, etc. The condition of the heart always deserves careful attention (digitalis!). The retinitis rarely demands special treatment.

CHAPTER IV

CONTRACTED KIDNEY

(Genuine Contracted Kidney. Granular Atrophy of the Kidney. Granular Kidney. Chronic Interstitial Nephritis)

Definition and Ætiology.—The genuine contracted kidney is the result of an extremely chronic and very slowly but constantly progressive atrophy of the renal tissue. The term "chronic nephritis" is also used for contracted kidney, but the special inflammatory processes are very subordinate here, for the anatomical change consists essentially in nothing but a simple degenerative atrophy of the renal parenchyma, and in a corresponding gradual increase of the interstitial connective tissue. From a general pathological point of view the process is to be regarded as wholly analogous to the corresponding changes in the liver in cirrhosis of that organ, in the spinal cord in the chronic degenerations of the different systems of fibers, etc. In all these cases we have a primary destruction of the special tissue-elements as a result of some deleterious action, and, following a general pathological law (Weigert), a partial replacement of the parts destroyed by a newly formed cicatricial connective tissue.

In the "genuine" contracted kidney the atrophy of the renal parenchyma begins in a previously healthy kidney. Cell after cell of epithelium, islet after islet of tissue, are slowly attacked, while other parts still remain intact. It was therefore an error of the older pathologists to regard the contracted kidney as the "third stage of Bright's disease," as if every granular kidney were first found in the stage of acute inflammation, and then passed into the stage of chronic enlargement, and lastly into that of contraction. This theory, of course, suits certain cases in part, for chronic nephritis at least may often finally pass into contraction, but these "secondary contracted kidneys" (*vide supra*) can clinically, and almost always anatomically, be differentiated from the genuine contracted kidneys. The contracted kidney may arise from an acute nephritis in some cases, and careful investigation indicates that such cases are not very rare; but then the process hardly ever passes through the three stages mentioned above, for the acute nephritis apparently recovers. A slight remnant of it is left—a little fire, as it were, smouldering under the ashes; its work of destruction advances, wholly in secret, and perhaps only after many years do the symptoms of a pronounced renal contraction appear.

If we inquire into the causes which produce the atrophy of the renal tissue in the ordinary cases of contracted kidney, which are chronic from the first, we are very often unable to make out any special etiology. Of course, one of the first things to consider in this disease is chemical or toxic agents, whether these are introduced directly as such into the body, or whether they are manufactured in the body because of abnormalities in metabolism, or as a result of infectious processes.

Experience teaches us that there are three chemical substances to be mentioned which may favor the development of contracted kidney: alcohol, lead, and uric acid. Chronic alcoholism is often to be regarded as the most probable cause of renal contraction, especially in people who have "lived well" otherwise, and have become corpulent. In these cases contracted kidney is to be viewed as a sort of atrophy, due to wearing out, or to strain. It is difficult to decide how much influence should be ascribed to the alcohol itself and how much to the excessive ingestion of food. The specific effect of alcohol is almost indubitable in those cases in which contracted kidney and hepatic cirrhosis are both present, a combination repeatedly observed.

We are, therefore, strongly inclined to assume that the chief cause of interstitial nephritis is an improper mode of life, but yet it can not be denied that the disease is also frequently observed in individuals whose lives are not open to criticism. There are cases of genuine contracted kidney in youthful patients, twenty-five to thirty-five years old, and of both sexes, for which not the slightest cause can be discovered. One might suppose, under such circumstances, that there are kidneys which are peculiarly vulnerable and which can not permanently meet even the ordinary demands of life. The connection between contracted kidney and chronic lead-poisoning, in type-setters, painters, etc., is also incontestable. It is a remarkable circumstance, and one not yet fully explained, that in these cases we very often see at the same time genuine gout (*arthritis uratica saturnina*). Gout, however, alone, without any co-existing chronic lead-poisoning, often leads to the development of contracted kidney, "gouty kidney," in which we probably have to do with the noxious action of an abnormal amount of uric acid on the renal parenchyma; but it always seems to be especially unfavorable to have an accumulation of several injurious factors—for instance, lead-poisoning associated with chronic alcoholism.

Infectious influences are, probably, first to be considered in those cases where the contracted kidney can be referred to a former acute infectious nephritis, as after scarlet fever. We may also mention here the appearance of contracted kidney sometimes observed after severe acute articular rheumatism. We may perhaps imagine a similar connection in the cases where contracted kidney is found combined with chronic endocarditis (valvular heart disease), or with chronic arthritis not of gouty origin. Of the chronic infectious diseases, which sometimes have a connection with the origin of contracted kidney, we may mention malaria and syphilis. The latter ought especially to be considered more than it is at present, because we may have either an immediate action of syphilitic toxines, or a renal atrophy as the result of a specific disease of the renal arteries.

We must here devote a little time to a consideration of the connection between renal contraction and primary disease of the vessels, which has been much discussed. It is true that we often find general arterio-sclerosis, and also atheroma, especially in the renal arteries, in the bodies of persons who have died from contracted kidney, but this frequent coincidence can not be remarkable in such cases, because contracted kidney is seen chiefly in elderly persons, and those in whom atheroma of the arteries is also a very common symptom. The theory advanced by the English authors, Gull and Sutton and others, that the vascular disease, "arterio-capillary fibrosis," always represents the primary process, to which the renal atrophy is only secondary, is, however, utterly untenable. We often find the most pronounced contraction of the kidneys without any vascular changes sufficient to explain the atrophy; and where the latter can be found in the small renal arteries, we usually have not a primary but a secondary process—namely, the well-known obliterating arteritis, which is seen in almost all chronic inflammations and degenerative atrophies of various organs.

Of course, it can not be denied that primary disease of the arteries in the kidney may check the flow of blood to certain portions of the tissue, and thus occasion secondary atrophy in limited areas (arterio-sclerotic contracted kidney), just as, for example, interstitial myocarditis results from primary arterio-sclerosis of the coronary arteries. This is particularly true of the so-called senile kidney—that is, the granulated kidney of old persons, due to atheromatous changes in the blood-vessels, and perhaps also certain other cases of nephritis subsequent to syphilis, and especially the rare cases of unilateral contracted kidney, which have been seen chiefly in association with syphilis.

With relation to the age and sex of patients with contracted kidney, it has already been repeatedly stated that the disease occurs mainly in later life. The explanation of this fact lies in the peculiar circumstances which occasion the renal process. For the same reason, also, the male sex is much oftener attacked than the female; yet the causes which lead to contracted kidney are so numerous that it is easy to understand why the disease is not infrequently seen even in younger individuals and in women. Contracted kidney does occur in children, though rarely. In all cases in youthful persons we must make careful inquiry as to any previous attack of acute nephritis, subsequent perhaps to scarlet fever, diphtheria, or measles.

The relations of contracted kidney to amyloid disease of the kidney (so-called amyloid-contracted kidney), and to chronic disease of the urinary passages, particularly of the pelvis of the kidney, will be discussed later in the appropriate chapters.

Pathological Anatomy.—In the genuine contraction of the kidney, both kidneys are always diminished in about the same degree. Their size is sometimes reduced to one half or even one third of the normal, so that it is almost difficult to find the little kidney in the very abundant and thick fatty capsule that is often present. The kidneys feel firm and dense, and show on their surface a very plain, coarse or fine, uniform or irregular, granulation. On pulling off the somewhat thickened fibrous capsule, these granulations become more prominent, and the capsule usually adheres quite firmly to the depressed portions. The raised portions are almost always darker and redder—that is, richer in blood—than the lighter and grayer depressions. Whether the whole kidney appears more red or more white depends only upon the amount of blood in the organ, and there is no reason for separating the " small red " from the " small white " contracted kidney.

On section of the contracted kidney, we find the cortex very thin, and pale atrophic streaks alternating with the darker portions. The pyramids are also rather small, and, as a rule, are darker than the cortex. In the pelvis of the kidney, which is often somewhat dilated, there are sometimes a number of uric-acid concretions. Striated uric-acid infarctions in the pyramids are a very characteristic mark of the gouty contracted kidney. The microscope shows an advanced destruction of the renal parenchyma, which is replaced by a cicatricial connective tissue in which the nuclei are still numerous or else have already become scanty. We can always make out signs of degeneration and atrophy of the epithelium, and the formation of casts in the uriniferous tubules which still remain, but which are already diseased. Atrophy, thickening of the capsule, etc., are found in many of the glomeruli. The uriniferous tubules that are still preserved in some places are often in part dilated. We can not here go more fully into the manifold histological details, especially the formation of cysts, the changes in the vessels (*vide supra*), the deposition of lime-salts, etc. Hæmorrhages are only very rarely present.

Thus the contracted kidney may be regarded as the form of chronic nephritis with by far the longest course (lasting from three to five years, and even much longer), and also the form with the widest extent. Its essential nature can in no way be contrasted with "chronic parenchymatous nephritis" as a "chronic interstitial nephritis"; for we always find interstitial processes in the former, which have reached a far higher degree in the contracted kidney only because the slow atrophy of tissue is compatible with a much longer duration of life, and hence can attain a much greater extent.

The anatomical changes in the other organs of the body beside the kidneys will be spoken of in connection with the symptomatology of contracted kidney.

Clinical Symptoms.—Except in the comparatively rare cases where we can refer the origin of a contracted kidney to a previous acute or chronic nephritis, the clinical symptoms of contracted kidney develop as gradually and insidiously as the anatomical process itself. There is no doubt but that a contraction of the kidney may exist for years, without calling the patient's attention to his disease by a single serious subjective symptom. This is evident from the chance discoveries on autopsy of a contraction of the kidney in people who have lost their lives in some other way, but especially from the cases where the severest symptoms, such as uræmia, cerebral hæmorrhage, etc., which often lead immediately to death, suddenly come on in persons previously regarded as perfectly healthy, while the autopsy shows a quite far advanced contraction of the kidney as the special cause of these symptoms. The less prominent the subjective symptoms of renal contraction are in the earlier stages of the disease, the more we should consider the objective changes, which in fact usually permit the diagnosis of the disease quite early on careful examination of the patient.

The condition of the urine is most important in this respect. As soon as changes have taken place in the epithelium in different parts of the kidneys, the results previously spoken of must make themselves manifest in the secretion of the urine, although still in a slight degree, and the diseased portions will secrete a urine diminished in amount and in solid constituents, but containing albumen. Since, however, many normal uriniferous tubules and glomeruli are still present, and since the whole process, as we have seen, develops only very slowly, the body gains time for the development of one of those apt compensatory arrangements which we recognize in so many pathological processes, and which we must regard in a teleological sense. This compensatory process consists of an increase in the arterial pressure, coming on as gradually as the renal contraction itself, and constantly increasing, and of a hypertrophy of the left ventricle dependent upon it. The blood therefore courses through the many normal glomeruli of the contracting kidney under an increased pressure, and the consequence is that in these portions the secretion of the urine, especially of the water, is much more abundant. It is possible, also, that the incipient disease of the glomerular walls which tends to increase their permeability contributes to the polyuria. At any rate, it is a fact that in cases of contracted kidney there is usually an abnormally large amount of pale, watery urine of low specific gravity, containing merely a trace of albumen (originating from the diseased portions of the organ). The daily amount of urine is often seventy to a hundred and twenty ounces (2,000–3,500 cubic centimetres) or more; the urine looks light-yellow and clear, contains scarcely any morphological constituents, has a specific gravity of 1010 to 1005 or even lower, and gives, on heating, only a slight precipitate of albumen, the amount excreted in the twenty-four hours being about half a drachm to a drachm (two to five grammes). On careful microscopic examination of the urine, we usually succeed in finding a few hyaline casts, which only exceptionally may be more abundant. The urine also frequently contains some white, and more rarely a few red blood-corpuscles. In rare but definitely attested cases it may happen that for a time, or even during the main part of the disease, the urine contains no albumen at all, or only a trace of it. This is probably explained by the fact that the diseased glomeruli have wholly ceased secreting, and that therefore the urine is secreted only by the healthy portions of the kidney. As to the character of the urine in arterio-sclerotic contracted kidney, *vide supra.*

It is apparent of how great significance this abundant secretion of water, as a result of the abnormally high blood-pressure, must be for the whole morbid process; for, in spite of the renal disease, there is now absolutely no retention of water in the body, and we therefore understand why there is often no œdema in contracted kidney, even after a course of years. The secretion of the solid constituents of the urine is not quite in such a favorable condition as the secretion of water. It is self-evident that the percentage of the former decreases with the increased amount of urine, but the whole amount of urea, uric acid, phosphoric acid, etc., eliminated is also at times somewhat less than normal in relation to the food. This diminution, however, is not very great, as long as the work of the heart is sufficient, and at certain times, especially in the earlier periods of the disease, a normal amount may be secreted. We accordingly see that the symptoms dependent upon an accumulation of the urinary constituents in the blood do not appear at all for a long time. Thus it happens that the patient may still feel perfectly well up to a time when the objective examination of the urine indicates marked pathological changes. Many patients, of course, notice the polyuria, but often no special attention is paid to it, and it is attributed to drinking a good deal of fluid. The patient gets accustomed to it, even if, as often happens, he has to pass his urine much more frequently than formerly, and even during the night.

We need not go into detail here in regard to the special causes of cardiac hypertrophy (compare page 602). It was with regard to contracted kidney that Traube advanced his mechanical theory of cardiac hypertrophy, which, however, rested upon the considerations previously mentioned, and therefore was properly replaced by the chemical theory, which was also very applicable to this form of renal disease. In its clinical relations it is an important fact that the cardiac hypertrophy causes no subjective symptoms at all, as long as the heart can suffice for the work put upon it without strain, a condition which is perfectly analogous to that of any fully compensated valvular disease. We can usually recognize the condition correctly only by a careful physical examination of the heart and the vascular apparatus, although in contracted kidney the percussion and palpation of the heart are often rendered difficult by a co-existing pulmonary emphysema. We can often perceive, however, the displacement and the increased strength of the apex-beat, the extension of the cardiac dullness to the left, and almost invariably the abnormal tension of the radial pulse, and the accentuation of the aortic second sound. In the later stages of the disease a hypertrophy of the right ventricle is often added to that of the left (compare page 603). Complete, or almost complete, absence of the cardiac hypertrophy is observed, as we have said, only in weak and cachectic patients.

As long, therefore, as the high arterial pressure kept up by the cardiac hypertrophy regulates the renal secretion in the way above described, the condition of the patient as a rule shows no special abnormality. At most it happens that certain cerebral symptoms now appear, especially attacks of headache and occasional vertigo, which, unless they are uræmic, are probably to be referred to active cerebral hyperæmia. Frequent nose-bleed also sometimes results from the abnormally high blood-pressure.

The clinical picture is completely transformed, however, upon the appearance of the first signs of incipient cardiac insufficiency—that is, the moment that the hypertrophy of the left ventricle ceases to be sufficient to overcome the obstacles to the secretion of urine occasioned by the deficiency in the renal parenchyma. The disturbance is evident either when the left ventricle itself grows weak, or when the gradual advance of the diseased process has reached such a point that even the most vigorous efforts of the heart are no longer sufficient to bring about compensation. In the latter case the symptoms which appear are to be regarded as uræmic, in the broadest sense of that word. The pulse is frequent, but it still remains unusually full and hard. On the other hand, in case there is a diminution in the cardiac energy the pulse becomes more compressible, smaller, more frequent, and sometimes, toward the termination of the disease, irregular. The heart-sounds remain pure, but the first sound often becomes indistinct. When there is a decided disturbance of compensation we hear a well-marked *bruit de galop*. In all these cases the symptoms result from the disturbance to the circulation, and from the consequent retention of urinary constituents.

Ordinarily, the subjective symptoms of contracted kidney begin very gradually, then disappear for a time, then appear again, and grow worse slowly but steadily. Apart from the feeling of general languor and weariness, it is usually the dyspnœa which first calls attention to the disease. The patient grows short of breath, is much disturbed by even slight physical exertion, and is perhaps subject to attacks of palpitation. Not infrequently the dyspnœa occurs, in the later stages of the disease, in distinct paroxysms which suggest asthma. This nephritic asthma has long been recognized. It does not have the same origin in every case; often it is undoubtedly the result of the attacks of cardiac weakness, and is then a merely cardiac asthma and corresponds in its individual symptoms to angina pectoris (see page 355). In other cases the asthma seems to be connected with the retention of the products of metabolism (uræmic asthma, *vide supra*). The clinical picture is most peculiar in those cases in which the dyspnœa is associated with the signs of acute pulmonary œdema, and is attended with the expectoration of a large amount of foamy, serous sputum, which is often tinged with blood. These conditions, which may pass off and reappear at stated intervals, are mainly those to which the name of humid asthma was formerly given. It may seem questionable whether we should regard the pulmonary œdema in such cases as a purely congestive transudation due to cardiac weakness, or as an œdema occasioned by nephritis and in a certain degree inflammatory. In the last stage of the disease there is often constant dyspnœa, and this may be the chief complaint of the patient. It is in such cases often referable to various causes acting simultaneously—for instance, pulmonary congestion, diffuse bronchitis, pneumonia (*vide infra*), and hydrothorax.

Another result of beginning cardiac failure in the later course of the disease may be œdema of various portions of the body. This is certainly in many cases to be regarded as a purely congestive œdema, particularly when associated with contracted kidney; but, on the other hand, we can not always exclude conditions which occasion nephritic œdema (*vide supra*). It has, indeed, been repeatedly observed that dropsy may be entirely absent in contracted kidney; but this is the case only when death ensues from some intercurrent attack before the pronounced cardiac insufficiency. Otherwise œdema is by no means rare in contracted kidney. It usually appears first in the ankles, the eyelids, or the prepuce, disappears again when the patient remains at rest, and, after a longer or a shorter pause, comes on anew, until finally, in the last period of the disease, a high degree of general dropsy may develop.

Among the disturbances of the internal organs we must mention first the cerebral symptoms. While at first, as we have said, these have more of an active hyperæmic character, the frequent and very violent headaches that come on later in part are of a uræmic nature and in part depend upon the passive hyperæmia, or the arterial anæmia of the brain. The pain sometimes shoots into the back of the neck, and sometimes is localized chiefly in one half of the head; it is often associated with symptoms of vertigo, with a gloomy or morose mood, with troubled sleep, etc. The stasis is usually also apparent in the abdominal organs. Chronic dyspeptic disturbances appear, the appetite fails, the bowels become irregular, and we can even make out a moderate enlargement of the liver. The influence which the altered activity of the heart exerts upon the function of the kidneys themselves is, however, particularly important. From what has been previously said of the dependence of the secretion of urine upon the arterial pressure, it follows directly that any compensatory activity of the still normal renal territory must at once experience a reduction, as soon as the blood-pressure falls. Corresponding to this we see, in fact, that the secretion of urine also usually suffers a decline at the same time with the other symptoms of stasis. The amount of urine is less abundant: it drops to forty or fifty ounces (1,500–1,000 cubic centimetres), and even lower; the specific gravity rises, rarely to a high figure, but still up to 1010 or 1012, or over. The urine often retains its light color for quite a long time, but it may finally more and more resemble the genuine urine of stasis. The point, however, which is especially to be considered, is the simultaneous and increasing retention of the solid constituents of the urine in the blood, and the consequent possibility of the onset of uræmic symptoms.

It must be stated that, in contracted kidney, the immediate exciting causes of uræmia are not always clear. Thus, it is a well-known and very important fact, clinically, that very severe and often fatal uræmic convulsions may sometimes attack the patient quite suddenly, apparently when in the best of health. Cases have been repeatedly seen, by other observers and by ourselves, where the daily amount of urine has shown no discoverable diminution in the days preceding the uræmia. Probably the explanation of this is that for a long time very minute amounts of toxic material are daily retained in the blood, and that these of themselves occasion no noticeable disturbance until suddenly the severe symptoms of uræmia develop as a cumulative result of long-continued, though slight, injurious influences. These cases of suddenly developing uræmia always remind us of the similar phenomena in chronic poisoning from lead or mercury. In these cases, also, after a long-continued absorption of trifling amounts of poison, the symptoms of intoxication may finally appear abruptly. In other cases of uræmia associated with contracted kidney, the condition of the heart is of great importance. The uræmic symptoms develop in different ways, according to the slowness or rapidity with which the cardiac insufficiency causes diminution in the urinary secretion. If the amount of urine is slowly diminished, we observe the symptoms of chronic uræmia (page 596), consisting of headache, vomiting, diarrhœa, severe pruritus of the skin, etc., but these symptoms are, of course, often combined with the immediate symptoms of stasis, and are not always to be easily and distinctly separated from them. Such a condition of chronic uræmia, in patients with contracted kidney, often presents a very mournful picture, since the unrestrainable and constantly recurring vomiting, the headache, and the general mental anxiety may last for weeks. The severe acute uræmia either is preceded by chronic uræmic symptoms, or comes on at once in the severest form, with general and often-recurring convulsions, and coma. The uræmia may pass off again, even in contracted kidney, but quite frequently it is the immediate cause of death (*vide infra*).

Beside the symptoms so far described, we must now mention a set of anatomical complications which may appear in the course of contraction of the kidney.

From its diagnostic and clinical importance the albuminuric retinitis, already known to us from the preceding chapter, takes the first place. It may come on at any time in the course of the disease; but it often develops so early that the patient, up to this time, knows nothing at all of his other disease. He merely consults an oculist, who often first recognizes, from the ophthalmoscopic image (see page 622), the special seat of the primary disease. Even in the cases where no subjective visual disturbance is present, the retinal examination sometimes discloses retinitis. In general, the contracted kidney is that form of renal disease in which retinal changes are decidedly most frequent.

Another rarer but clinically important complication consists of the hæmorrhages in internal organs, whose cause is to be found either in the increased arterial pressure, or in an abnormal weakness of the walls of the vessels—arterio-sclerosis in older persons, defective nutrition of the vascular walls in young and anæmic patients. Hæmorrhages into the brain are comparatively the most frequent. They cause both mild and severe apoplectic attacks, which pass off completely or leave a hemiplegia behind, and sometimes they are the direct cause of death. Beside the hæmorrhages into the brain itself, there may also be hæmorrhages on the inner surface of the dura mater—hæmatoma. Nose-bleed is also of significance; in many patients it is frequent and very stubborn; we have ourselves seen two cases where the fatal termination was caused directly by an unrestrainable nose-bleed. Hæmorrhages into the other organs are more rare, but they have been observed in the skin, the stomach, the intestines, and the lungs. In a few cases, indeed, a sort of hæmorrhagic diathesis seems to develop. We must remind the reader again, in this connection, of the general truth that in renal disease all the internal organs display a somewhat increased tendency to secondary inflammation; thus the mucous membranes often present an accompanying catarrhal inflammation, such as chronic bronchitis, gastritis, gastric catarrh, and intestinal catarrh. These catarrhal troubles are in part to be regarded as due to congestion, but in part they certainly result from the retention of the products of metabolism. In serous membranes we often observe pleurisy, pericarditis, etc. The surface of the body sometimes displays a tendency to obstinate eczema. Of the inflammations of internal organs, pneumonia is the most frequent and important. Some cases are croupous or lobar, and some are of that diffuse lobular variety which is peculiar to all sorts of nephritis. We have already mentioned that the kidney itself may suffer from inflammatory exacerbations, in addition to the chronic disease.

Quite great variations appear in regard to the general nutrition. In most cases where the disease develops quite gradually in persons in middle or advanced life, the general nutrition for a long time shows no striking anomaly. The patient is often very well nourished, and even corpulent, at the period when the first cardiac symptoms begin. To the more practiced and attentive eye, of course, he shows a certain appearance of suffering, which later becomes more pronounced. He becomes emaciated, and has a sallow and often cyanotic complexion. Marked anæmia usually develops only in younger individuals, who then show the pallor characteristic of so many patients with renal disease.

We desire to append a few remarks with regard to arterio-sclerotic contracted kidney. This may pursue precisely the characteristic course of the ordinary disease, but in many cases its symptoms are very obscure. If we examine carefully, from day to day, the urine of old persons who present other signs of general arterio-sclerosis, we shall be likely to find at many times a small trace of albumen, and at other times none at all. In such cases we may be almost certain that there is an arterio-sclerotic contracted kidney. Still, the renal symptoms are usually of minor importance in the clinical picture of senile marasmus, senile emphysema, senile dementia, or the other changes of old age.

General Course, Duration, and Termination.—The most important peculiarities in the course of renal contraction have already been spoken of above. We have stated that the disease may be latent for a long time; that the severest symptoms—such as uræmia or apoplexy—sometimes come on suddenly and unexpectedly; that in other cases the disturbances of compensation in the heart, dyspnœa, palpitation, or slight œdema, are the first symptoms; that, under some circumstances, certain complicating conditions, such as retinitis, or frequent nose-bleed, first direct suspicion to a renal disease, and suggest an examination of the urine; while, finally, in a last class of cases, only general disturbances, loss of appetite, pallor, general physical weakness, and similar symptoms induce the patient to consult a physician. It is usually hard to decide how long the disease has lasted before a diagnosis is made. We must especially inquire into the existence of polyuria, which patients may not notice, however, even if it exists.

The further course may vary according to the onset of complications, the external conditions under which the patient lives, etc. In general, as we must repeatedly emphasize, much depends upon the heart's capacity for work and its staying qualities. If death does not ensue sooner from some intercurrent disease, the last stage of the disease almost always presents itself under the picture of cardiac insufficiency with predominant symptoms of dyspnœa and general dropsy.

As has been said, we usually can not determine with any accuracy the duration of the disease. It may, at any rate, last many years, probably even ten years or more, although there may be many variations in its course. It is not impossible that, during the earlier period of the disease, there may be a cessation in the process of renal atrophy, but it is hard to decide with certainty. At all events, the disease must generally be termed absolutely incurable, although life may not only be preserved for a long time, but the patient may even exist without much discomfort. We need not refer especially here to the different intercurrent attacks, the possibility of which must always be kept in mind in regard to prognosis.

Diagnosis.—The diagnosis of contracted kidney can be made with certainty only by examining the urine. We must, therefore, dwell again on the necessity of making this examination in all suspicious cases, because only in this way can we avoid overlooking the condition. The suspicion of a developing renal contraction should demand an examination of the urine, especially in all cases where the patient complains of frequent headache, of congestive conditions, of palpitation and dyspnœa, asthmatic attacks, disturbances of vision, general dullness, and dyspeptic symptoms, without finding any other reason for these symptoms. The polyuria, the clear urine of low specific gravity, containing a slight amount of albumen, in connection with the signs in the circulatory apparatus, the tense pulse, and the hypertrophy of the left ventricle, permit us to recognize the disease correctly in most cases. If retinal changes are present, they may sometimes be of much aid in confirming the diagnosis. The ætiological conditions—lead, gout, alcoholism, etc.—of course also merit attention.

The diagnosis presents great difficulty in the quite rare cases where albuminuria is absent. In these cases we are sometimes able to reach the correct interpretation of the morbid condition only by repeated examinations of the urine. Otherwise we can scarcely avoid mistaking it for chronic affections of the heart, such as myocarditis or idiopathic hypertrophy.

The diagnosis is also very difficult if the patient does not come under observation until the stage of fully developed disturbance of compensation. The characteristic features of the urine of contracted kidney are then absent, the urine is scantier, darker, richer in albumen, and it is often scarcely possible to decide whether we have a primary renal affection with secondary cardiac hypertrophy or a primary heart disease with a secondary congested kidney. If general arterio-sclerosis or marked pulmonary emphysema is present at the same time, the judgment as to the condition is still more difficult. In such cases a correct diagnosis is possible only by very carefully balancing all the different symptoms, and considering the whole course of the disease.

Finally, the diagnosis of contracted kidney is very difficult in cases where the first examination of the patient is made during a sudden attack of uræmia or after an apoplectic seizure. Here the albuminuria is the symptom which points most to the existence of a renal disease, although, in spite of this symptom, the judgment as to the condition, and its differentiation from other acute cerebral affections, often presents great difficulties.

We must devote a few words to those not very infrequent cases in which a small amount of albumen is found, often quite by chance, in the urine of youthful individuals who are apparently in perfect health; thereupon the question is propounded, which is often of extreme practical importance, whether we are dealing with a so-called physiological or intermittent albuminuria (*vide supra*, page 589), or with an actual renal disease—viz., an unsuspected contracted kidney. The diagnosis is never easy. In the first place, it is necessary to make careful observations for a considerable period, at the same time varying the conditions as to nourishment (nitrogenous diet, milk diet, use of beer), and activity (complete physical rest, persistent exercise); then we must carefully investigate all the possible ætiological circumstances, such as previous disease and mode of life; and finally, take into consideration the associated phenomena—for instance, of the circulatory apparatus. From a practical point of view it is advisable to be most cautious in all these cases, and, at any rate, to give such advice as to regimen with the aim of preventing damage to the kidneys, as if there were an actual renal disease.

Treatment.—As soon as the diagnosis of renal contraction is established, the whole hygienic condition of the patient must be regulated so as to prevent the advance of the affection in every possible way. In this respect two indications are to be fulfilled, to guard against any irritation which may have an injurious action on the kidneys, and to relieve the work of the heart as much as possible, in order to keep off cardiac insufficiency as long as we can. The diet must be carefully regulated, and must be of scant measure or abundant and strengthening, according to the patient's physical constitution. In these cases, too, milk is a food of great value. The use of meat is, in general, to be limited, while easily digestible dishes made from cereals and eggs, and vegetables and fruits, are to be recommended. Alcoholic beverages should be allowed in only limited amounts, their place being supplied by mineral waters (Fachinger, Wildunger, Wernarzer), and water with lemon-juice or the juice of other fruits. Great care of the skin is an important matter. We should recommend the regular employment of warm baths or salt baths, and daily sponging of the whole body with brandy or warm vinegar. All physical over-exertion is to be avoided, although moderate methodical exercise is to be recommended for corpulent patients. We should always provide for regular evacuation of the bowels by appropriate remedies, dietetic prescriptions, fruits, bitter mineral waters, etc. The general condition is often materially improved by proper air and recreation, and in this way resort to a bath may be of service, chosen according to the individual conditions, such as Brückenau, Marienbad, Carlsbad, Kissingen, Nauheim, Ems, or Baden-Baden. In appropriate cases, a winter residence in the south is advisable.

If disturbances of compensation appear, we must be still more strict in our dietary, and must enjoin the utmost physical rest, at the same time employing other treatment according to the symptoms which may be present. Above all, we must endeavor to impart new vigor to the heart by means of digitalis, though unfortunately such efforts often prove unavailing. The minutiæ of treatment in this connection are almost precisely the same as in chronic heart disease (*q. v.*), and in other renal diseases. The treatment of the dyspnœa and chronic uræmia, which develop in the last stages of the disease, is apt to be most unsatisfactory; but we can, sometimes at least, remove the symptoms by various external applications upon the chest and the head (ice-bags or hot compresses), by various drugs to combat the nervous symptoms, bromide of potassium, antipyrine, nitroglycerine, and by the combination of cardiac stimulants, such as digitalis and strophanthus, with diuretics, such as caffeine and diuretine. In the worst cases narcotics are indispensable, particularly subcutaneous injections of morphine. As yet we are unable to exert a direct beneficial influence by drugs upon the interstitial process in the kidneys. The only ones recommended for this purpose and worth a trial are the preparations of iodine—viz., iodide of potassium, or iodide of sodium, in daily amounts of three to eight grains (gramme 0.2–0.5), and, in anæmic patients, iodide of iron. We do not need to go minutely into the treatment of the numerous complications which occur.

The prophylaxis of renal contraction is evident; we should avoid so far as we can the known ætiological conditions.

CHAPTER V

AMYLOID KIDNEY

Ætiology.—The amyloid kidney is invariably associated with the more or less extensive amyloid degeneration of other organs in the body. In its clinical relations, however, it claims the most interest of all amyloid diseases, since it has by far the greatest significance for the whole clinical picture of amyloid degeneration.

As is well known, we understand by amyloid degeneration a peculiar change which, under certain pathological conditions, is observed in the connective tissue, and especially in the smaller vessels. The walls of the vessels are thickened, they have a lustrous, homogeneous appearance, and they show peculiar reactions on treatment with certain coloring agents. These reactions are due to the presence of an albuminoid substance—amyloid—which either is deposited in the tissue from the blood, or, as is much more probable, is developed in that spot from the albuminoid substances there present. In marked amyloid degeneration the diseased organs often show macroscopically an altered, "bacon-like" appearance, and assume a characteristic red-brown color on treating the affected parts with Lugol's solution of iodine, changing to violet on the addition of sulphuric acid. The microscopic examination alone affords more accurate conclusions as to the presence and distribution of the degeneration. For this purpose we usually stain the tissues with methyl-violet or gentian-violet. The amyloid portions thus take on a very characteristic and clearly defined red color. In this way we can discover that the amyloid degeneration begins everywhere in the walls of the small vessels, that the interparenchymatous connective tissue may also be affected later, but that the parenchymatous cells proper, liver cells, renal epithelium, etc., almost always remain perfectly free. The latter often show atrophic and fatty degenerative changes (*vide infra*), but little if any amyloid degeneration.

Nothing is known as to the special causes which effect this peculiar metamorphosis of the albumen of the connective substance into amyloid. We know only that there are a number of primary diseases in which experience has shown that amyloid degeneration quite frequently develops as a secondary condition in the different organs. These conditions have, for the most part, the common characteristic of being associated with general cachexia and debility, and of being connected with some localized chronic morbid process in some part of the body, from which abnormal chemical matters may be constantly carried into the blood. We suppose that the normal albuminous material is transformed into amyloid by means of such influences. With regard to the relations which may exist between the so-called hyaline degeneration of Recklinghausen, and amyloid disease, the investigations are not yet complete.

The conditions in which amyloid degeneration in general, and consequently amyloid disease of the kidney also, are chiefly observed, are the following, arranged in about the order of their frequency:

1. Chronic pulmonary tuberculosis, particularly the ordinary ulcerative phthisis. Tubercular ulcers of the intestines, with or without co-existing marked pulmonary tuberculosis, may also lead to amyloid disease.

2. Long-continued chronic suppuration in the bones or soft parts, especially chronic fungous processes with fistulæ into the bones or joints, empyema with fistulæ, vertebral caries, etc.

3. Constitutional syphilis, chiefly the cases with ulcerative and usually tertiary processes in the bones and mucous membranes.

4. Other ulcerative processes, or processes associated with chronic suppuration: saccular bronchiectases, chronic intestinal ulcers (for example, of dysenteric origin), purulent pyelo-cystitis, vesico-vaginal fistulæ, ulcerated new growths, such as cancer, etc.

5. In rare cases amyloid has also been observed in other chronic diseases, as in malaria, gout, and other chronic articular affections. In the medical clinique at Leipsic we once saw a case of marked amyloid kidney in a girl of twenty-one with aortic insufficiency.

6. Finally, in a small class of cases, of which we have ourselves seen some examples, no discoverable cause at all may be found at the autopsy for quite extensive amyloid degeneration. In such cases, therefore, there seems to be a primary intoxication with resultant amyloid disease.

Pathological Anatomy.—With regard to the anatomical lesions of amyloid disease in other organs, we shall refer to handbooks on pathological anatomy. As to amyloid liver, see page 580. Here we shall discuss merely the pathological anatomy of amyloid kidney.

In very slight and limited amyloid degeneration of the kidneys, the latter present a perfectly normal appearance to the naked eye. Careful microscopic examination alone shows amyloid degeneration of the walls of different vessels in the cortex, and especially in the medullary substance.

The commonest and most characteristic form of amyloid kidney is the so-called large white amyloid kidney (waxy kidney, lardaceous kidney). The kidney is usually enlarged, and the surface is smooth and of a grayish-white or yellowish color, and usually somewhat mottled. On section, the cortex is wide and also of a yellowish-white color, and the glomeruli may often be recognized with the naked eye as dull, lustrous, translucent points. Hæmorrhages are scarcely ever seen. The medullary substance may be also pale, or darker. In many cases the cortex may itself have a darker reddish or mottled appearance, which is due merely to the greater amount of blood in the organ. The pale-yellow color is due either to the anæmia or to the fatty degeneration, while the amyloid spots show a more translucent character with a bacon-like luster.

If we examine the kidney microscopically, we find first the amyloid degeneration, which, in varying extent and combination, affects most frequently the glomeruli and also the capillaries of the cortex, the vasa recta, and sometimes the membranæ propriæ of the uriniferous tubules. In pure amyloid kidney the rest of the renal tissue is normal, but in many cases we find changes in the epithelium—fatty degeneration, desquamation and disintegration—and also not infrequently interstitial cellular infiltration. We see, therefore, that amyloid degeneration of the kidney is not infrequently associated with degenerative changes of an inflammatory nature. Thus we observe, not infrequently, the combination of chronic diffuse nephritis (large white kidney) with amyloid.

If the process has lasted for a long time, it leads, as in ordinary nephritis, to a complete atrophy of tissue in some parts, with a corresponding increase of connective tissue. Then the renal tissue sinks in at the affected spots, and there is a decided unevenness to the surface of the kidney. There is even a completely developed red or white contracted kidney, in which we find abundant amyloid, and which is, therefore, termed amyloid contracted kidney. In this form the parenchymatous and interstitial changes correspond precisely to those in ordinary contracted kidney, only the amyloid degeneration is added to them.

At present differences of opinion prevail as to the precise connection between amyloid and the inflammatory degenerative processes in the kidney. In our opinion there is, in most cases, an actual combination of both conditions, they being co-effects of simultaneously acting causes. We have seen that in tuberculosis chronic suppuration and genuine nephritis develop, as well as amyloid disease, so it can not be surprising that sometimes with these diseases both sequelæ, nephritis and amyloid, should develop side by side, and that we should, consequently, find in the kidney not only the changes associated with an inflammatory, large, white kidney, a secondary contraction, or a genuine contracted kidney, but also a more or less extensive amyloid degeneration. On the other hand, it, of course, can not be questioned that the disturbance of circulation, which must arise in consequence of a marked amyloid of the vessels, is of influence on the nutrition of the renal tissue, and that, therefore, many changes in it, especially fatty degeneration of the epithelium, are, under some circumstances, the direct result of the amyloid disease.

Clinical History.—If we consider the great diversity which the distribution of the amyloid in the kidneys shows, and its manifold combinations with inflammatory processes, it is clear from the outset that we can not set up a uniform picture of the symptoms of amyloid disease in general. To this we must add that the symptoms of amyloid disease, which is almost always a secondary condition, are also modified in various ways by the primary disease.

We must first state that many cases, where the amyloid in the kidneys is of comparatively slight extent, can not be recognized by any clinical symptom. The albuminuria in particular may be entirely absent, as has been repeatedly proved (Rosenstein, Litten, and others). That in such cases the vasa recta and not the glomerui are chiefly affected by the amyloid degeneration has been affirmed but not proved.

Except in these instances, however, the urine secreted from the amyloid kidneys shows marked changes, which, of course, present considerable variations according to the form of the individual case. The amount of urine is most frequently about normal, or somewhat diminished—in some cases much diminished—but in others it is decidedly increased, so that the patient may pass eighty to a hundred and twenty ounces (2,500–3,500 cubic centimetres) in the twenty-four hours. We quite frequently see considerable variations in the amount of the urine in the same patient at different times. All these differences are easily explained if we remember how many circumstances may affect the amount of urine—the presence or absence of inflammatory changes in the kidney, the presence or absence of cardiac hypertrophy, co-existing perspiration, diarrhœa, œdema, fever, etc.

The color of the urine is almost always light yellow. Only exceptionally, in amyloid nephritis, does it contain an abundant sediment; usually it is entirely, or almost entirely, clear. The very considerable amount of albumen in the urine, which is often one or two per cent., is also characteristic of amyloid kidney. In many cases, indeed, particularly in the combination of amyloid with contracted kidney, the amount of albumen is smaller, although usually decidedly more abundant than in pure interstitial nephritis. Senator has pointed out that the comparative amount of the globuline which is contained in the urine besides the serum albumen is often particularly large in amyloid kidney. The specific gravity of the urine varies very much according to the amount of water and albumen in it. It may be increased (1015–1020) or diminished (1010–1005).

If we examine the urine under the microscope, we usually find only a few hyaline casts, and also most frequently a small number of white blood-corpuscles. In general, it is very characteristic of amyloid kidney that there is an abundance of albumen, contrasting with the scanty or scarcely appreciable microscopic sediment. In the combination of amyloid with more marked nephritic changes the sediment is more abundant, so that the urine is cloudy. The microscope then shows more numerous hyaline or moderately fatty casts, more abundant white blood-corpuscles, sometimes a little renal epithelium, and in quite rare cases even red blood-corpuscles. Amyloid reaction is said to occur in the casts, but it is very rare, and therefore of no value in diagnosis.

The other morbid symptoms which are observed in amyloid kidney depend either upon the change in the kidneys themselves, or upon co-existing amyloid degeneration in other organs; or, lastly, upon the primary disease. The symptoms of the latter are, of course, extremely varied, but in many cases they may be entirely subordinate.

In regard to the directly resulting symptoms of amyloid kidney, their occurrence is of interest, especially in comparison with the analogous conditions in acute nephritis. Dropsy of a moderate, or even a severe degree, is often present in amyloid kidney, but it may also be entirely absent. We must remember that an œdema independent of a renal affection may be produced by marantic venous thrombosis. Uræmic symptoms are distinctly rare in amyloid kidney, but they are sometimes observed, especially in their milder forms, such as vomiting. It is a very important point that a hypertrophy of the left ventricle is absent in most cases of amyloid kidney. This is explained partly by the fact that the disease usually affects feeble, cachectic individuals who have no superfluous material for the manufacture of cardiac hypertrophy, and partly by the fact that in pure amyloid kidney there is no appreciable retention of urinary constituents in the blood. This latter fact explains, also, the infrequency of uræmic symptoms (*vide supra*). If, however, the amyloid degeneration is combined with genuine nephritic changes, the condition of things is altered. In amyloid contracted kidney we have repeatedly observed hypertrophy of the left ventricle.

Albuminuric retinitis hardly ever appears in pure amyloid kidney. In the amyloid contracted kidney it has sometimes been observed, however, in cases where there has probably been originally a pure contracted kidney, with amyloid coming on later. The secondary inflammations in the internal organs, such as renal pneumonia, and the hæmorrhages, such as cerebral hæmorrhage, are also rare.

The patient's general condition is dependent in part upon the renal disease, and in part upon amyloid degeneration in other organs, but mainly upon the primary disease. The patient with amyloid kidney is usually, therefore, cachectic, and shows in high degree a pallid, anæmic color of the skin. If, also, general dropsy develops, we have an external appearance very characteristic of amyloid disease; still, in some few cases of syphilis, bronchiectasis, and unilateral pulmonary contracture, the general nutrition remains tolerably good for a considerable time.

The symptoms which point to a co-existing amyloid degeneration in other organs beside the kidneys are of great diagnostic significance. The symptoms in the liver (enlargement, abnormal firmness, and a hard, sharp lower edge to the organ), the spleen (enlargement and hardness), and intestines (obstinate diarrhœa not yielding to any remedy) are clinically important in this respect. The interpretation of the diarrhœa is, of course, usually difficult, since it may often depend upon tubercular intestinal ulcers as well as upon amyloid of the intestines.

We can scarcely make general statements in regard to the whole course and the duration of amyloid kidney, since the form of the primary disease is to be especially considered in these cases. In regard to the time that it takes for an amyloid degeneration to develop in an existing primary disease, the degeneration is certainly present sometimes after a few months. Of course, it is hardly ever possible to determine its onset accurately, since the first beginnings of amyloid degeneration in the kidneys do not usually permit themselves to be recognized at once by the appearance of albuminuria (*vide supra*). The duration of amyloid kidney varies very much according to the severity of the case; it may last only a few weeks or months before death, while other cases have certainly lasted for a year, especially in amyloid contracted kidney.

The **prognosis** of amyloid kidney is in most cases utterly unfavorable, which is due mainly to the incurability of the primary disease; but trustworthy observers have repeatedly proved that, when the primary disease is curable, as with syphilis and many chronic suppurations, an already developed amyloid kidney may exceptionally be completely restored.

The **diagnosis** of amyloid kidney can be made with considerable certainty when the evident signs of a renal affection are added to those of a disease which we know by experience to promote the development of amyloid degeneration. Whether in such cases we have a pure amyloid or a pure nephritis, or a combination of the two, can be decided with some certainty only from the condition of the urine: a clear urine, containing but few morphological elements, but rich in albumen, points to pure amyloid, while a large number of casts and red and white blood-corpuscles in the urine points to the presence of inflammatory changes in the kidney. The diagnosis of amyloid contracted kidney is justified when the symptoms of contracted kidney (abundance of pale urine, secondary cardiac hypertrophy) are seen in association with such morbid conditions as are likely to occasion amyloid disease. Of great importance, as we have already pointed out, is the comparatively large amount of albumen and the consequently somewhat higher specific gravity of the urine. Of many cases of amyloid kidney it is very characteristic, and so of diagnostic value, that there is a rapid and frequent variation in the amount of urine and the percentage of albumen (Wagner).

A very material support for the diagnosis of amyloid kidney, and therefore one which should always be looked for, is the discovery of amyloid in other organs. We have briefly mentioned above the most important symptoms in the liver, the spleen, and the intestines referable to this point.

Treatment.—Only the treatment of the primary disease can, of course, be considered, both as a prophylactic and also as a causal indication. In many surgical cases, and also in the cases of amyloid in syphilis, there is a possibility of this (as by iodide of potassium); but otherwise we try to improve the primary disease as far as we can.

In other respects the treatment is purely hygienic and symptomatic. We must try to strengthen the patient as much as possible by good food and the exhibition of preparations of iron and quinine. The use of iodide of iron or small doses of iodide of sodium is to be recommended. In a symptomatic point of view the same remedies are used as in other renal diseases.

We insert here a synopsis of the condition of the urine and of the left ventricle in the most important varieties of renal disease.

	Amount of urine.	Specific gravity.	Amount of albumen.	Morphological constituents.	Condition of left ventricle.
1. Acute nephritis.....	Diminished.	High.	Considerable.	Numerous casts; blood-corpuscles; epithelium.	Not hypertrophied.
2. Chronic diffuse nephritis............	Normal or somewhat diminished.	High.	"	Numerous casts; blood-corpuscles; epithelium; compound granular cells.	Hypertrophied.
3. Contracted kidney.	Increased.	Low.	Slight.	Few casts; small number of blood-corpuscles.	Hypertrophied.
4. Amyloid kidney	Variable, normal, or increased.	Variable.	Considerable.	Few casts.	Not hypertrophied.
5. Amyloid contracted kidney	Increased.	Rather low.	Moderate.	"	Hypertrophied.
6. Passive congestion .	Diminished.	High.	Absent or scanty.	Few casts; blood-corpuscles.	

CHAPTER VI

PURULENT NEPHRITIS AND PERINEPHRITIS

1. Purulent Nephritis

Ætiology.—Although in the forms of nephritis so far described the occurrence of large numbers of nuclei in the interstitial tissue has been repeatedly mentioned, none of them ever come to genuine suppuration—that is, to a purulent liquefaction of tissue, a true abscess-formation. The origin of a purulent nephritis is, rather, always associated with the entrance of perfectly definite morbid irritants into the kidneys. These are invariably organized and their special peculiarity is to excite a purulent inflammation.

There are two chief ways through which the morbid irritants may reach the kidneys—the arterial blood-current and the urinary passages. The first-mentioned means of entrance is to be considered in all the cases of purulent nephritis which come on as one symptom of pyæmic processes and certain forms of ulcerative endocarditis (see pages 116 and 307 on the point). Far more rarely purulent nephritis develops in this way as a complication in other diseases, such as dysentery. Purulent nephritis also occurs in actinomycosis (Israel).

The excitants of inflammation take the second path in those cases where a purulent nephritis follows an inflammation of the lower urinary passages, the pelvis of the kidney, the bladder, etc. Here the bacteria, which almost always enter directly into the urinary passages (the urethra and bladder) from without, pass gradually upward from the bladder through the ureter to the pelvis of the kidney; from this they enter the apertures of the collecting tubes and the uriniferous tubules of the kidney, everywhere exciting a purulent inflammation. We therefore term these forms of purulent nephritis—with regard to their origin—purulent pyelo-nephritis.

We must remark in conclusion that a purulent nephritis and perinephritis (*vide infra*) may arise in direct wounds of the kidney from infection of the wound.

Pathology.—Purulent nephritis shows quite characteristic peculiarities and differences according to its mode of origin. (We omit traumatic abscesses here.)

The renal abscesses in pyæmia and analogous diseases are usually focal suppurations, which only exceptionally attain a great extent, but which are usually to be recognized with the naked eye as numerous little yellowish dots or lines, scattered over the whole kidney, about half a millimetre or a millimetre in diameter. On microscopic examination, these nodules prove to be genuine little abscesses, in whose territory the renal tissue proper is completely destroyed. In the middle of them we often find the originating colony of micrococci, the "micrococci embolus," seated in a central vessel. The conditions are still plainer if we examine an earlier stage of the process. We find vessels (the loops of the glomeruli, or the encircling capillaries), which are completely plugged with micrococci, and in whose vicinity the renal tissue is still perfectly normal. We further see analogous spots where the renal tissue is already necrosed in the vicinity of the colony of micrococci, and is infiltrated with emigrated cells. These nodules show, finally, a continuous transition to the completed abscess, which is often surrounded by a hyperæmic or even a hæmorrhagic areola.

In purulent pyelo-nephritis the renal abscesses appear somewhat different. The abscesses also have a characteristic striated appearance, corresponding to the distribution of the inflammation along the straight tubules. They often extend from the point of the renal papilla through the cortex to the surface of the organ, so that from the outside we see the abscesses, showing through as yellowish points. The broader abscesses arise from the confluence of neighboring striæ. The microscope shows that the purulent inflammation arises from the vessels of the interstitial tissue, in whose territory the uriniferous tubules are of course destroyed. The clusters of micrococci form the most interesting feature. These settle originally in the uriniferous tubules and are the special cause of the necrosis of epithelium and the inflammation. Pyelo-nephritis, indeed, was one of the first diseases for which a bacterial origin was discovered (Klebs).

Clinical Symptoms.—We can speak very briefly here in regard to the clinical symptoms of purulent nephritis, since they can never be sharply separated from the symptoms of the primary disease. The pyæmic renal abscesses, and the abscesses in ulcerative endocarditis, hardly ever cause special clinical symptoms, so that their presence is first recognized on the autopsy-table. Since the abscesses do not as a rule communicate with the uriniferous tubules, there is usually no great amount of pus in the urine.

The clinical symptoms of pyelo-nephritis also depend less upon the nephritic abscesses than upon the previous and accompanying pyelitis and cystitis. We will therefore return to renal abscesses in the description of these diseases.

2. PERINEPHRITIC (PARANEPHRITIC) ABSCESS

Perinephritic abscess is the name given to suppurations in the vicinity of the kidney, especially in its fatty capsule or in the peri-renal connective tissue. Apart from any traumatic origin for such abscesses, they develop most frequently as a result of purulent nephritis or purulent pyelitis. The escape of pus, which involves the surrounding tissue in the inflammation, may come from the ureter or pelvis of the kidney, or from the kidney. The special form of primary disease differs very much; it may be either simple purulent pyelitis, or pyelitis from renal calculi, or sometimes tubercular processes and new growths that finally suppurate, such as cancer, or echinococci. The peri-renal suppuration may also take its start from the other organs in the neighborhood. Thus cases have been seen in which the perinephritis followed a perityphlitic abscess, a hepatic abscess, or a psoas abscess after vertebral disease. Perinephritic suppuration may also be due to actinomycosis. It is a very important fact, however, that paranephritic abscesses may develop as a primary disease in persons in apparently perfect health, particularly in middle-aged men, and this without any discoverable cause. It is usually absolutely impossible to determine how and by what path the inflammatory germs reach the peri-renal connective tissue, unless possibly they migrate from the intestine. These cases of abscess are very important from a clinical standpoint, because the symptoms are at first difficult of interpretation. There is fever, usually of a decidedly intermitting pyæmic type, and there are also dull, indefinite pains in the loins or abdomen, constipation, and constitutional symptoms. The cause of all these symptoms is gradually made apparent by the development of distinct local changes.

In almost all cases of perinephritic abscess the accumulation of pus finally becomes so great that a swelling appears, usually in the lumbar region, and this grows more and more prominent; at first it is scarcely noticeable; but later the skin becomes œdematous there, it constantly protrudes more and more, it assumes an inflammatory hyperæmic redness, until finally a definite fluctuation shows the advance of the abscess up to the skin. In other cases the inflammatory swelling extends forward into the iliac fossa; then there is also abnormal resistance and dullness above Poupart's ligament. The swelling may also extend upward toward the diaphragm, so that the diaphragm is crowded upward, giving rise to marked dyspnœa. The relations of the swelling to the descending colon are sometimes the same as in new growths of the kidney (compare Chapter VIII).

Besides the swelling, there is almost invariably a very great pain in the affected region, either spontaneous or on pressure. If the swelling presses on the large nerve-trunks in the vicinity, it produces severe shooting pains in the leg of the same side, and sometimes a numb feeling and paresis. The leg is then often kept in a position similar to that in coxitis.

The fever is persistent, and usually of an intermittent character and associated with rigors. It and the pain make the patient thin and feeble, even to an extreme degree. The urine does not contain pus unless the abscess has some connection with the urinary passages.

Recovery may occur if the pus finds some way out of the body. Apart from operative interference, the most favorable termination is the spontaneous discharge of the pus into the intestine (colon), or into the urinary tract (pelvis of the kidney, bladder). The course of the disease is much more tedious if the pus finds its way through the skin. If renal abscesses discharge outwardly, they most frequently point in the loins, less often like a psoas abscess, below Poupart's ligament. They may also discharge into the pleural or peritoneal cavities, with a consequent empyema or quickly fatal peritonitis. In other cases, if prompt surgical aid is impossible, death occurs from exhaustion.

The **diagnosis** is made chiefly from the swelling, pain, and fever, and a consideration of the ætiological factors. We can settle any doubts in most cases by exploratory puncture. The condition may be confounded with hydronephrosis, psoas abscess, and solid tumors of the kidney.

The only **treatment**, apart from the fulfillment of any symptomatic indications, is surgical, and consists in opening and draining the abscess. The result depends chiefly upon the patient's general condition, and the character of the primary disease. The details are to be found in the text-books of surgery.

CHAPTER VII

DISTURBANCES OF CIRCULATION IN THE KIDNEYS

1. **The Congested Kidney.**—Although local impediments to the flow of venous blood from the kidneys, such as thrombosis of the renal veins, hardly ever attain a clinical significance, the participation of the kidneys in a general venous stasis, as is seen chiefly in heart disease (compare page 331), pulmonary emphysema, etc., is of great diagnostic importance, since we possess in the condition of the urine quite an accurate measure of the intensity as well as of the increase and decrease of the stasis.

The congested kidney is easily recognized anatomically. The organ is often somewhat enlarged, it feels firmer than normal, and shows, both on its surface and on section, a dark, bluish-red color—"cyanotic induration." The medullary substance is usually darker than the cortex. Under the microscope we see considerable dilatation and a tense fullness of the veins and capillaries. The parenchyma is normal, but in more advanced cases it may show a beginning fatty degeneration of the epithelium, which is a result of the defective arterial blood-supply. At first the interstitial tissue is little changed, but if the congestion persists for a long while there may be a gradual destruction of the renal parenchyma, to a certain degree, with the formation of an abundance of contractile interstitial tissue (congested and contracted kidney).

The clinical symptoms of congested kidney concern only the changes in the urine. The amount of urine diminishes, corresponding to the diminution of the arterial pressure and the diminished rapidity of the blood-current. Only twenty-five to twenty ounces (800–500 cubic centimetres), or less, ten to six ounces (300–200 cubic centimetres), are secreted daily. The urine is also more concentrated and darker than normal, and often has an abundant sediment of urates. If nutritive disturbances have begun in the epithelium of the glomeruli as a result of stasis, the urine is also albuminous, but the amount of albumen in pure congested kidney is always slight—about one tenth to one sixth of the volume. The urine often contains, besides, a few hyaline casts, and a few white and red blood-corpuscles, the latter pointing to little congestive hæmorrhages.

If the changes mentioned come on as one symptom of a general venous stasis, and are, accordingly, associated with cyanosis and dropsy, the diagnosis of congested kidney can be made with certainty. If we succeed in restoring the circulation by appropriate remedies, such as digitalis, the urine at once becomes more abundant and clearer and its albumen disappears. Otherwise the characteristics of the urine of passive congestion last until the patient's death.

2. **Embolic Infarction in the Kidneys.**—Since the renal infarction, although it has great pathological interest, is hardly ever of clinical significance, we will limit ourselves here to a brief description of the most essential points.

If one of the smaller renal arteries is plugged by an embolus in heart disease, the affected portion of the organ cut off from the circulation must perish, since all the renal arteries are terminal arteries. The epithelium undergoes the well-known changes of coagulation necrosis, disappearance of the nuclei of the cells, and disintegration, and the tissue becomes entirely or in part a hæmorrhagic infarction (compare page 331). In this way arise the characteristic wedge-shaped, red, hæmorrhagic infarctions in the kidney, or far more frequently the yellowish-gray, anæmic infarctions (often surrounded by a hæmorrhagic areola), the base of which is at the surface of the kidney; the base may reach a width of half a centimetre to a centimetre or more, while the apex extends a varying distance into the cortex, or even into the medullary substance. Later on the gradually disintegrated tissue of the infarction is absorbed, round cells emigrate from without into the region destroyed, and a shrunken connective-tissue cicatrix gradually develops in place of the former infarction. Many kidneys may have such a granular surface from numerous infarction cicatrices that they may be appropriately termed "embolic contracted kidneys."

The anatomical processes just briefly described cause in most cases no special clinical symptoms at all. Sometimes, however, a slight amount of blood in the urine seems to depend on the development of a hæmorrhagic infarction in the kidneys, so that when a cause for embolic processes, such as heart disease, is present, we may entertain the suspicion of the development of a renal infarction during life. In rare cases the development of a rather large infarction of the kidneys is associated with a sudden and violent pain in that region, with marked subsequent hæmaturia or, as we observed in one case, hæmoglobinuria.

The embolic processes in the kidney never demand special treatment.

CHAPTER VIII

NEW GROWTHS IN THE KIDNEYS

Of the primary forms of tumor occurring in the kidney, two especially claim our interest: cancer of the kidney and congenital sarcoma. The latter is of great importance in regard to the general theory of tumors, since it points definitely to the development of the new growth from scattered portions of embryonic tissue. Striped muscular fibers have been repeatedly found in tumors consisting otherwise of round or spindle cells, from which the name of *rhabdomyoma* or *myosarcoma striocellulare* has been chosen for these tumors. Since there are no muscular fibers in the kidney itself, their occurrence in the tumors points undeniably to disturbances of embryonic development. This theory obtains a further interesting confirmation from our own observation of the development of left-sided, and, probably, congenital renal sarcoma in two brothers. Both children died when between two and three years of age, and the autopsy gave almost precisely the same lesions in both: numerous metastases in the liver and lungs, beside a new growth almost as large as a child's head in place of the left kidney.

Renal cancer is also remarkably frequent, comparatively speaking, in children under four years of age, and about equally common in the two sexes. Of course, we find renal cancer in persons of more advanced years; in some cases of this sort renal calculi seem to occasion the development of carcinoma. This is like the relation between gall-stones and cancer of the biliary passages. Usually only one kidney is affected, chiefly the left, as it seems, but the new growth has sometimes been found in both kidneys. In its character, renal cancer belongs either to the denser or to the softer, medullary form. It may permeate the whole kidney and change it to a large tumor, weighing fifteen or twenty pounds (five or ten kilogrammes). Softening and hæmorrhage very often take place within the tumor. The proliferation has been repeatedly observed to extend to the neighboring parts, especially the pelvis of the kidney, and metastases also form in other organs, as in the lymph-glands, liver, or lungs. It should also be mentioned that several times renal cancer and cancer of the testicle have been combined.

The clinical symptoms of renal tumor are entirely absent, or of a very indefinite nature, in the first period of the disease. Dull pain in the renal region is repeatedly given as the first symptom, but, of course, this is hardly ever of definite significance. The diagnosis almost always first takes a definite direction by the appearance of a palpable tumor. This develops in the lumbar and lower lateral abdominal region, constantly extending from this point upward and inward. As stated above, both carcinoma and sarcoma of the kidney may cause enormous tumors, especially in children, which may make the whole abdomen protrude to a marked degree. The tumor feels firm and sometimes smooth, sometimes uneven; it does not usually move with respiration, but this rule has exceptions, particularly in the case of tumors of the right kidney. In tumors of the left kidney some diagnostic importance attaches to the relation of the new growth to the descending colon, inasmuch as the latter is pushed forward by the growth of the tumor and comes to lie between it and the anterior abdominal wall. It is quite often possible to demonstrate that portion of the intestine which traverses the tumor by means of percussion, and sometimes even by means of palpation. For this purpose the colon may be examined alternately when empty and when artificially distended with water, or, still better, with air. In the case of tumors of the right kidney, there may be corresponding relations to the ascending colon, but this is less frequent. Almost all larger tumors of the kidney occasion displacement of the diaphragm upward, and of neighboring organs laterally. Another factor in diagnosis is the *ballottement rénal* described by Guyon: when a gentle thrust is made in the renal region the tumor is felt to strike softly upon the anterior abdominal wall.

There may be scarcely any pain for a long while, but in some cases there is violent and persistent pain. Often the pressure of the tumor upon neighboring nerve-trunks—for instance, the sciatic—occasions obstinate neuralgia, sometimes associated with paresis.

In many cases of renal tumor the urine shows no abnormal conditions at all, since its secretion is performed vicariously by the other healthy kidney. In carcinoma of the kidney it sometimes presents, however, one sign valuable for diagnosis—namely, an admixture of blood. This hæmaturia often comes on very early, even before there is any tumor to be felt. It is repeated either frequently or only rarely in different cases, and sometimes it is entirely absent. The hæmorrhage is associated with colicky pains only when large clots have to pass through the urinary passages. Sometimes, but very rarely, small particles and shreds of tissue from the disintegrated new growth may be found in the urine.

The general symptoms are often very late at first, especially in children; but finally a general condition of marasmus almost always develops. A constant and great frequency of the pulse is often striking. We must also mention the peculiar symptom several times observed in girls with congenital renal tumors—viz., an abnormally early development of the pubic and axillary hair, and sometimes a peculiar pigmentation of the skin (Kühn).

The **diagnosis** of renal tumors is, in many cases, quite evident, but in others very difficult. The position of the new growth, its limited mobility, its relations to the colon, and, above all, our knowledge as to the occurrence of renal tumors in children, often suggest the correct interpretation of the case. In older persons renal hæmorrhages which can not be otherwise explained must direct our suspicions to the possibility of a cancer of the kidney. Often, however, the disease has been confounded with tumors of the retro-peritoneal glands and of the ovaries, with large psoas abscess, tumor of the liver, and splenic tumor.

The **prognosis** is, of course, unfavorable. The disease sometimes lasts only a few months, sometimes a year or two, rarely longer.

The **treatment** must in most cases be purely symptomatic. The only expectation of success lies in the operative removal of the new growth, the details of which are to be found in recent monographs on renal surgery.

CHAPTER IX

PARASITES OF THE KIDNEYS AND OF THE URINARY PASSAGES—CHYLURIA

1. **Echinococcus of the Kidney.***—Echinococcus cysts have been repeatedly found in the kidney, although much more rarely than in the liver. Usually only one kidney is affected, and the parasite is generally situated in the renal substance itself, only exceptionally between it and the capsule of the kidney. The size of the echinococcus cysts may be very considerable, the diameter reaching to twenty centimetres or more.

Clinical symptoms usually first appear when the tumor can be felt through the abdominal walls. Subjective symptoms may even then be entirely wanting. Pain on pressure develops gradually later. The tumor usually has an approximately globular shape. Its relations to the neighboring organs, especially to the colon, are the same as we have learned to recognize in the preceding chapter, in the description of cancer of the kidney. The so-called hydatid thrill which is said to be characteristic, and which is obtained by giving the tumor a little push with the flat of the hand, can be distinctly felt in only the rarest instances.

It is a comparatively frequent occurrence for the echinococcus cyst to burst into the pelvis of the kidney. Then single echinococcus cysts, or at least bits of membrane, hooks, etc., are passed with the urine, usually with severe colicky pains, which are exactly like the renal colic from the passage of a calculus. Such attacks may be often repeated, and may form a very severe type of disease by obstructing the urinary passages—the bladder and urethra. In such cases the symptoms of a secondary pyelitis and cystitis are often added.

Perforations in other directions are much rarer. The rupture of a renal echinococcus into the lungs has sometimes been observed, the patient coughing up echinococcus cysts.

Sometimes, especially after injuries, the sac of the echinococcus becomes inflamed, suppurates, and leads to a general pyæmic condition.

The diagnosis of renal echinococcus is possible only when a tumor can be made out belonging to the kidney, and when portions of echinococcus are passed with the urine, or through an exploratory puncture. It has already been stated that one should be cautious about employing this procedure (see page 577). The cysts are most frequently confounded with hydronephrosis (*vide infra*), and, in women, with ovarian tumors.

The prognosis is not wholly unfavorable. Permanent recovery has been repeatedly observed, especially after the rupture, or single or repeated evacuations, of the sac of the echinococcus; but, of course, echinococcus of the kidney may also be attended with numerous dangers, such as suppuration of the sac. The course of the disease is always very tedious.

A radical treatment is possible only by surgical means. Symptomatically, ice and local blood-letting are used when there are symptoms of local inflammation; and morphine, warm baths, and sometimes mechanical aids, e. g., the catheter, when there are symptoms of colic.

2. **Distoma hæmatobium** (**bilharzia hæmatobia**—*blood-fluke*, see Fig. 71) is a parasite which belongs to the flukes or trematodes, and which occurs especially in Egypt, Abyssinia, and East Africa. Infection is said to take place partly by means of the drinking-water and food, and partly, perhaps, by the parasite creeping into the urinary passages and the rectum during bathing. Natives are much oftener at-

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* In regard to the general natural history of the echinococcus, compare page 575.

tacked than Europeans. The distoma has its chief abode in the branches of the portal vein, and particularly in the venous plexuses of the bladder and the rectum. Its eggs are often deposited in great numbers in the mucous membrane of the pelvis of the kidney, the ureter, and the bladder, and they excite in these places a severe inflammation with ulceration and consequent strictures, or the formation of concretions. Similar violent inflammation occurs, also, in the rectum and in the sexual organs. The course of the disease is very chronic. The main symptom is persistent hæmaturia. Many cases of so-called tropical hæmaturia are due to the distoma, and later we have the symptoms of local inflammation and of severe cystitis. The **diagnosis** may be established by the discovery of the eggs in the urine or the fæces. **Treatment** is merely symptomatic.

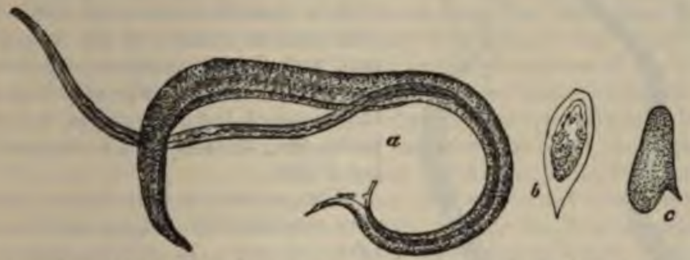

FIG. 71.—Distoma hæmatobium (from LEUCKART). *a*. Male and female, the latter in the canalis gynæcophorus of the former. Ten diameters. *b*. Egg with a terminal spine. *c*. Egg with a lateral spine. 150 diameters.

3. **Strongylus or Eustrongylus Gigas** (*palisade worm*) is a parasite occurring in the pelvis of the kidney in many animals—the dog, the wolf, the marten—and very rarely in man. In size and color it is not unlike an ordinary earth-worm. It may produce symptoms of severe pyelitis, with hæmorrhages, and colicky pains. No well-established cases have been reported of late.

4. **Filaria Sanguinis. Chyluria.**—The blood filaria of man, belonging to the round-worms, has obtained a special clinical interest, since it is recognized, from the investigations of Wucherer in Bahia in 1868, and of Lewis in the East Indies in 1870, as the cause of the tropical chyluria and some allied diseases, such as lymph scrotum, elephantiasis Arabum, and chylous ascites.

The full-grown filaria, "*filaria Bancrofti*," a very thin worm, about three or four inches long, has been found only a few times in man. Its seat is in the larger lymphatics, where it gives rise to chronic stasis of the lymph with its consequences—chronic hyperplasia of the connective tissue, etc. In chyluria, the parasites are probably situated in the main branches of the thoracic duct—at any rate, in such a place that a stasis of the lymph ensues in the lymphatics of the bladder, or perhaps, in some cases, of the pelvis of the kidney and the other urinary passages. If the distended lymph-sac ruptures, the lymph or chyle is poured out into the urinary passages and is evacuated with the urine. Since this process may be often repeated, the intermittent course of chyluria is thus explained. The individual attacks of the disease may come on during years at intervals of weeks or months. They are often associated with pain and febrile symptoms.

The condition of the urine, which in many cases may look almost exactly like milk, is most characteristic. A creamy layer of fat forms upon the surface. If we shake the urine with ether, the greater part of the fat can be removed, and the urine rendered clear. The fat in the urine may amount to two or three per cent. The chyluria is often associated with a hæmaturia coming from the ruptured veins. The urine then looks bloody red, and shows under the microscope many red blood-corpuscles beside the fat-drops. Large clots often form in the urine.

The embryos of filaria, found in the urine in very many cases, although not in all, form the most important diagnostic feature in the urine. These (see Fig. 72) are objects two to three tenths of a millimetre long, with a diameter about equal to that of a red blood-corpuscle. They are usually imbedded in a very delicate sheath, which often projects at the end of the animal, and show a constant, vigorous vibrating motion. They have also been found in the blood of the patient, as well as in the urine, and, strange to say, especially during the night.

The course of the filaria disease may vary considerably. Many patients reach an advanced age; in others, severe general symptoms, like anæmia and emaciation, finally come on. The different forms in which the disease occurs—chyluria, elephantiasis, etc.—are combined in manifold ways.

The region of the geographical distribution of the disease lies almost wholly in hot countries. It has so far been most frequently observed in Brazil, the Antilles, the East Indies, China, Japan, Egypt, Cape Colony, and Australia. Nothing definite is yet known of the precise mode of invasion of the parasites. According to Manson's investigations, mosquitoes play an important part here. [This view is now well established.—V.]

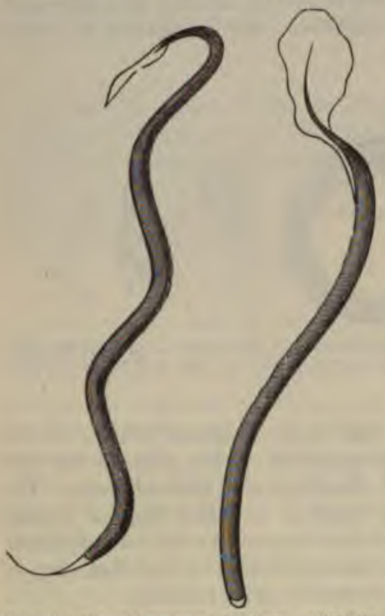

FIG. 72.—(From SCHEUBE.) Embryos of filaria.

In regard to treatment, apart from any surgical interference, we may try picro-nitrate of potassium, three to ten grains (gramme 0.2–0.5), in pills or capsules, several times a day (Scheube).

CHAPTER X

MOVABLE KIDNEY (FLOATING KIDNEY, REN MOBILIS)

Ætiology.—With some practice the physician can frequently palpate the kidneys, particularly the right kidney, even under absolutely normal conditions ("palpable kidney"). This is much oftener the case in women with lax and yielding abdominal walls than in men. With every inspiration the kidney is pushed somewhat downward. In many cases, therefore, the kidney, or at least its lower half, is felt only upon deep inspiration. If the kidney is not only palpable, but also capable in greater or less degree of being moved about by the hand, and if, consequently, we do not always find the kidney in the same place, then we describe it as an abnormally movable kidney; or, if the case is extreme, a floating kidney. What may be the causes of this extreme mobility of the kidney is not settled. Often it may be due to the congenital, anatomical relations of the peritoneum, and other tissues around the kidney. This is indicated, particularly, by the occasional occurrence of movable kidney in children. Certain external mechanical factors, however, undoubtedly play the most important rôle. First among these is the distention and relaxation of the abdominal walls as a result of pregnancy; and, secondly, the influence of clothing, waist-bands and corsets. This explains the fact that movable kidney is found mainly in women, and particularly in married women. At the same time, the abnormal mobility and downward displacement of the kidney are often merely a part of a general "enteroptosis" (see page 465), and are therefore associated with gastroptosis, coloptosis, and corset liver. That the movable kidney is far more often the right than the left, is probably due to the fact that the right kidney is originally less firmly fastened, and is placed lower, and also that it is affected by the great mass of the liver which lies above it. Numerous other causative influences have been mentioned, but none of them is as important as the preceding: they are severe physical labor with frequent tension of the abdominal muscles; injuries involving the region of the kidneys; diseases of the neighboring organs, particularly displacement of the uterus, and finally emaciation, causing absorption of the fat which supports the kidneys and makes tense the abdominal walls. After death a movable kidney can not be recognized, unless it is found in an abnormal position—e. g., in front of the vertebral column, or close to the anterior abdominal walls. In such cases its external edge may be directed downward and its inner edge upward.

Symptoms.—The clinical importance of movable kidney is estimated very differently by different physicians. Many conceive that it is a very frequent cause of manifold abdominal disturbances; others are almost inclined to deny it any influence on the health. We ourselves believe that a movable kidney as such may, in fact, excite disagreeable symptoms, but that this is the exceptional occurrence. As we have already mentioned, palpability and slight movability of the right kidney are so frequent in women that as soon as one's attention is directed to the matter, one finds this condition in a large proportion of all cases in which there is occasion to make a careful abdominal examination. The pains and abnormal sensations in the abdomen, of which women so frequently complain, are difficult of interpretation, and it is, of course, very seductive and convenient to refer them to a movable kidney; but if we consider how often we find an exactly similar movable kidney in women who are entirely free from abdominal symptoms, we can not avoid suspicion as to the importance of the condition.

It is difficult to form a characteristic group of symptoms which may be unhesitatingly referred to movable kidney. First, pain should be named. This is to a certain extent local, but it often radiates into the epigastrium and the sacral and lumbar regions, and sometimes is colicky. Not infrequently the pain is associated with nausea. The discomfort is aggravated by any decided motion of the patient, or by riding, driving, and the like; but during repose it becomes slight, or entirely disappears. In rare cases we have symptoms of strangulation. These occur periodically, and are said to be especially frequent at the time of the menses. They consist of the sudden onset of violent pain; chilliness which seldom amounts to a rigor; great tenderness and tenseness of the abdomen; slight fever; vomiting; and collapse. During this time the urine is usually scanty, and the amount does not increase again till the attack is over, about three to five days. The precise cause of these symptoms is supposed to be a sudden damming up of the urine by a kink or twist in the ureter. This occasions acute hydronephrosis, and resultant phenomena, which last until the urinary channels are free again. In certain cases floating kidney seems actually to be the cause of persistent hydronephrosis, with secondary pyelitis.

The discovery of floating kidney by palpation is usually easy. The physician examines the patient lying on her back, applying his left hand on the right lumbar region and pressing forward, while his right hand presses from in front in the opposite direction. It is then often possible, especially upon deep inspiration (*vide supra*), to catch the kidney between both hands, and so form an opinion as to its position and mobility.

The next point is to determine whether, in any particular case, the symptoms which may be present are actually due to the movable kidney which has been discovered, or whether the latter is an unimportant matter. This question demands a careful and complete physical examination and a consideration of all the symptoms. In the first place, we should determine accurately the position of the stomach (gastroptosis, etc.), and we should also think of the possibility of gall-stones, which may produce a very similar group of symptoms. Gall-stones are especially apt to occur in women with corset liver and enteroptosis, and this explains why a movable kidney is not infrequently found in cases of biliary colic, although it has nothing to do with the attack.

In a great majority of cases of "floating kidney" we have to do with those familiar and frequent conditions of a "nervous" character which are termed hysteria or neurasthenia. Women of this kind suffer very frequently from all sorts of painful sensations in the abdomen, and from dyspeptic disturbances and the like, and of course we very often find in them a movable kidney. In such cases the abdominal symptoms are also of a nervous character, as can usually be easily determined from the general condition of the patient, from her psychical and cerebral symptoms, and the indications of a cardiac neurosis, and also from the results of suggestive treatment. Still, a certain number of cases may be very difficult of diagnosis. If we find a movable kidney in a nervous, hysterical woman, it is not always advisable to apprise the patient of the fact, for with a person of this sort the mere idea of possessing a "floating kidney" is enough to stir up a host of subjective symptoms. On the other hand, it may be said that the floating kidney may serve as a very handy instrument for suggestive therapeutics. If we say to such a patient that her annoyance would immediately vanish after the application of a suitable bandage, we may sometimes attain great therapeutic success, although this is apt to be but temporary.

Treatment.—If we deem that the movable kidney requires treatment, the first thing to recommend is the wearing of a suitable support. The bandages, with special pads and the like, we regard as usually of no value, while a good elastic abdominal bandage, with thigh-straps, or a well-fitted "abdominal corset," is sometimes decidedly efficient, particularly in all cases of general enteroptosis. We should also see that the patient is properly nourished. If she is emaciated, we should prescribe rest with the largest possible amount of food, so-called over-feeding. As more and more fat is deposited in the abdomen, the abdominal walls regain their tension and the kidneys receive better support. It is also advisable to sponge the abdomen with cold water or brandy.

If there are any symptoms of strangulation, we should of course put the patient to bed and order hot poultices and opium. We may also make a cautious attempt to replace the organ. If severe attacks of this sort recur, the possibility of surgical treatment should be considered (nephrorrhaphy or nephropexy). In a fairly large number of cases the patients are said to have been entirely freed of their discomfort by this method.

APPENDIX

THE DISEASES OF THE SUPRA-RENAL CAPSULES AND ADDISON'S DISEASE (BRONZED SKIN)

Ætiology and Pathological Anatomy.—In the year 1855 the English physician Addison published for the first time a list of cases in which, besides the symptoms of a general bodily weakness and anæmia, a peculiar dark pigmentation of the skin had gradually developed. Since disease of the supra-renal capsule was found at the autopsy in all cases, Addison concluded that this was the immediate cause of the bronze coloring of the skin. Observations similar to Addison's were soon made in greater numbers, so that the fact itself can not be doubted; but even at present nothing definite is known as to the special cause of the disease or the true nature of this remarkable connection between disease of the supra-renal capsules and pigmentation of the skin.

Attempts have been made in different quarters to obtain an explanation from experiments on animals. From these it has appeared that the complete extirpation of both adrenals in animals usually results, after a short time, in death, but if vestiges of the adrenals, or if so-called accessory adrenals are left behind, there is no unfavorable result; or, if any, it is much delayed. The symptoms of the extirpation of the adrenals consist in emaciation, paretic weakness of the muscles, indigestion, and nervous disturbance. It has not yet been possible to obtain by experimental methods such an abnormal pigmentation of the skin as is seen in Addison's disease (Nothnagel and others). Very many experiments have been made with regard to the effect of extract of the adrenal gland; it affects chiefly the vaso-motor system, as shown by the increase of blood-pressure and slowing of the heart's action. The results thus obtained do not as yet, however, justify absolute conclusions. Chemical examination of the gland shows an abundance of lecithine, also brenzcatechine, and a substance, sphygmogenine, which raises the blood-pressure. Most investigators are at present inclined to the opinion that the adrenals have the task of destroying, or rendering innocuous, certain poisons which are formed in the body, particularly in connection with muscular activity. It may also be that the adrenal gland secretes some material of physiological importance. We are as yet far from a clear understanding of the matter.

The anatomical lesions found in man up to the present time are not calculated to add clearness to the case, since they seem to contradict one another in various points. In the first place, some observations must be mentioned in which the supra-renal capsules were found perfectly normal in spite of an existing pigmentation of the skin. Such cases, however, prove little, since it of course can not be put in question that a staining of the skin may sometimes develop from some other reason besides disease of the supra-renal capsules. On the other hand, it has been asserted that extensive changes are sometimes found in the supra-renal capsules on autopsy without the existence of the symptom of bronzed skin during the patient's life; but these cases are also open to the objection that the disease has perhaps not been extensive and intense enough to cause the bronze coloring of the skin. The contradictions just mentioned, however, have led to many other attempts at an explanation of Addison's disease, of which one especially deserves consideration. According to this, the symptoms met with are produced not by the disease of the supra-renal capsules themselves but by the invasion of the solar plexus and the semilunar ganglia of the sympathetic by the morbid process (Risel, Burger, and others). According to this theory, the symptoms of Addison's disease may arise when the aforesaid nervous parts are diseased independently, or by the extension of a pathological process from some other neighboring organ; but pathological anatomy has not furnished any corroboratory evidence of this view. On the contrary, the more recent investigations of the sympathetic nervous system in Addison's disease have had ambiguous results.

Addison himself has pointed with emphasis to the fact that the precise form of disease in the supra-renal capsules is by no means always the same. At any rate, the disease named from him is not to be regarded as a definite anatomical affection, but rather as a particular group of symptoms. By far the most frequently it is tuberculosis of the supra-renal capsules which lies at the bottom of Addison's disease. The capsules then are either enlarged and studded with caseous tubercular new growths, or they are in part cicatricially contracted. Other tubercular affections are almost always present in the body at the same time, especially caseation of the mesenteric lymph-glands and pulmonary tuberculosis. Other morbid processes besides tuberculosis may also be found in the supra-renal capsules: tumors (adenoma or struma, carcinoma, sarcoma), hæmorrhage, atrophy, etc. It is self-evident, from what has been said above, that in every case we must consider some implication of the neighboring sympathetic ganglia, by compression, cicatricial contraction, or chronic inflammation. Both supra-renal capsules are almost always diseased at the same time, rarely only one is affected.

Of the lesions in other organs we must also mention that Peyer's patches and the solitary follicles of the intestine are as a rule swollen. The spleen is somewhat enlarged in some cases, but not in others. There is no striking pigmentation of the internal organs. The changes in the skin and in certain mucous membranes will be mentioned below.

Considering the variety of the anatomical causes there can be no question of a uniform ætiology of the disease. Among the causal factors, those most frequently reported are defective nutrition, care and anxiety, and finally injury of the abdomen. The majority of cases are met with in the male sex and in middle life. Addison's disease, however, must be regarded as a rare affection, of which but few cases are observed even in the larger cliniques.

Symptomatology.—The purest type of Addison's disease appears in those cases where the symptoms are apparently primary in their development, and do not come on in the course of some other disease, such as phthisis or cancer.

The first symptoms of the disease are usually of a general nature, and are referable to a gradually increasing anæmia and to general weakness and physical lassitude. The anæmia shows itself objectively through the pallor of the skin and the diminution in the number of red blood-corpuscles, but without other definite anomalies of the blood that can be made out. Moreover, there are a number of symptoms which depend in part upon the cerebral anæmia, and in part upon nervous disturbance. In this number are the mental torpor and lack of energy, the headache and tendency to syncope, and the tinnitus aurium. Sometimes there are marked psychical disturbances, such as impairment of memory, feebleness of mind, or exaltation. The patient's general nutrition often suffers very considerably; but it must be added that in Addison's disease, as in other anæmias, the fatty layer, especially over the abdomen, often remains remarkably well developed.

The great muscular weakness, asthenia, of the patient is still more characteristic than the anæmia. This is shown less in inability to make a few vigorous contractions than in extremely rapid exhaustion of the separate muscles. As a consequence, all long-continued muscular exertion, such as walking for any length of time, standing, continuous labor, becomes absolutely impossible.

Beside the symptoms which have been enumerated there is often gastric disturbance. The appetite is poor, and there is very often vomiting. The latter may sometimes be almost uncontrollable, and then it is one of the most distressing symptoms of the disease. It is usually due not to an anatomical change in the stomach, but probably to the anæmia of the brain, or to other nervous influences. Cardialgic symptoms are also frequent. The bowels are sluggish as a rule, but there is sometimes diarrhœa. We sometimes hear functional murmurs in the heart, but as a rule its sounds are pure though feeble. The pulse is usually moderately accelerated. The liver and spleen do not show any special changes. Albuminuria is exceptional and depends upon complications, such as amyloid kidney; the bodily temperature is not elevated, and not infrequently, indeed, it is decidedly subnormal.

The special characteristic symptom, which alone renders the diagnosis possible, is the gradual onset of a peculiar pigmentation of the skin. This usually shows itself first in the face and on the backs of the hands, and also in those parts which normally present a greater pigmentation (the areola of the nipples, the axillæ, and the genitals), or which are exposed to greater pressure by the clothing, as the hips and shoulders. It is especially noteworthy that dark pigmented spots and stripes usually develop on the mucous membrane of the lips, particularly at the corners of the mouth, and inside the cheeks. The intensity of the coloring differs in different cases. It usually increases as the general condition grows worse. In the most intense cases almost the whole skin may become dark brown or black, like that of a mulatto or negro. Sometimes, however, the pigmentation remains limited to separate large or small spots, and in other parts of the skin there may then be even a marked loss of pigment. The nails and the sclera always remain white, and frequently the palms of the hands and the soles of the feet also. The hair is usually not changed. The pigmentation of the skin usually increases during the whole disease; only exceptionally does the skin become light again in the later stages.

The real cause of the accumulation of pigment in the skin is wholly unknown. We find on microscopic examination of the skin that the pigment lies not only in the cells of the rete Malpighii, but also in the corium, especially along its blood-vessels. It is probably formed from the blood-pigment, and is carried by wandering cells from the cutis into the epithelial layers of the skin (Demiéville, Nothnagel).

The course of Addison's disease is almost always chronic, and may last for years, but cases have been described with a rather acute course. The disease sometimes begins with violent initial febrile symptoms, vomiting, and diarrhœa. The disease then has a comparatively rapid termination after a few months, or a second chronic stage may follow the first acute one.

The final termination of Addison's disease is always unfavorable. Temporary remissions are often observed, but the disease always becomes worse again after them. Death usually ensues gradually amid the signs of increasing general uræmia and weakness. In some cases severe nervous symptoms also come on toward the end of the disease—coma, delirium, or epileptiform attacks. Conditions of this sort may develop comparatively suddenly and unexpectedly; indeed, they seem to support the hypothesis of toxic influences (*vide supra*).

Treatment.—Organo-therapy has been repeatedly tried in Addison's disease, on the same lines as the treatment of diseases of the thyroid gland. The adrenals of the sheep have been administered, either as fresh glands, finely minced and eaten with bread, or as an extract, or in the form of tablets of the dried glands. In some cases excellent results seem to have been obtained in this manner, even to diminution in pigmentation, but in many other cases this prescription has no beneficial effect. We ourselves administered adrenal tablets in one case for a long time without any advantage. Still, it would be proper to continue these trials. Whether treatment with Koch's tuberculine might cause permanent benefit in those cases which are due to tuberculosis, is as yet doubtful. A cautious trial might be made under proper circumstances. As to other remedies, tonics are generally employed—nourishing food, iron, quinine, and arsenic; iodide of potassium, bromide of potassium, and electricity have also been tried, but without success. Symptomatic treatment is needed for the vomiting, diarrhœa, and nervous attacks. Experience has shown that great caution should be exercised in prescribing laxatives, because in repeated instances such remedies have had decidedly unfavorable results.

SECTION II

DISEASES OF THE PELVIS OF THE KIDNEY AND OF THE BLADDER

CHAPTER I

INFLAMMATION OF THE PELVIS OF THE KIDNEY. PYELITIS

Ætiology.—Isolated primary pyelitis hardly ever occurs as an independent disease. Pyelitis is rather in most cases either a complication or a result of other diseases, and in such cases often attracts but little clinical attention.

We sometimes find a rather moderate pyelitis in the bodies of persons who have died of severe general infectious diseases, typhoid fever, small-pox, diphtheria, or pyæmia. The affection depends, in all probability, upon the elimination by the kidneys of substances that excite inflammation, and is thus to be regarded as analogous to the renal changes which often coexist. Toxic substances, such as cantharides and copaiba, which pass through the kidneys, may also cause pyelitis as well as other disturbances.

Pyelitis very often arises from a direct extension of inflammation from the neighboring organs. In many cases of acute and chronic nephritis the pelvis of the kidney takes part in the inflammation to a greater or less degree; but an ascending extension of the inflammation from primary diseases of the urethra or bladder is still more common. Any case of urethritis or cystitis may, if it lasts long, advance upward to the ureters and the pelvis of the kidney, so that in severe cases we often find an inflammation of the whole urinary tract, a pyelo-cystitis, and even a "ureteritis." It has been already mentioned (page 640) that the inflammation may extend still farther to the kidneys themselves (pyelo-nephritis), and we shall refer to this repeatedly. Of all these varieties of ascending inflammation in the urinary passages, none is so frequent or of so great practical importance as that which results from persistent narrowing of the urethra (stricture, hypertrophy of the prostate), and the consequent obstruction to the flow of urine. We shall revert to this important variety when discussing hydronephrosis. Very frequently the ascending pyelitis is a sequel to cystitis in disease of the spinal cord, with paralysis of the bladder.

Another form of pyelitis is that caused by the presence of foreign bodies in the pelvis of the kidneys—e. g., calculous pyelitis due to the mechanical irritation of renal calculi. This also will have a special consideration below. Much less frequent causes are retained blood-clots, parasites (*vide supra*), and genuine foreign bodies.

Primary idiopathic pyelitis, as a result of catching cold or similar influences, is, as we have already said, of extremely infrequent occurrence. We have ourselves seen a few cases which did not seem to be capable of any other interpretation, and yet we must strongly emphasize the necessity of regarding all such cases of pyelitis with great suspicion, from a diagnostic point of view. It is only too often that the "simple" pyelitis will prove to be renal tuberculosis, or an inflammation due to calculi or other causes. The pyelitis coming on in women in childbed, or following different sorts of diseases of the sexual organs, may in all cases be referred to an infection of the pelvic mucous membrane from the bladder or from the kidneys.

Pathological Anatomy.—In simple catarrhal inflammation the mucous membrane of the pelvis of the kidney is reddened, swollen, and covered with an abundant secretion, which contains varying amounts of pus-corpuscles and epithelium.

In severer inflammations we often find quite numerous little hæmorrhages in the mucous membrane, and sometimes little gray nodules, which correspond to the swollen lymph-follicles.

In severe cases, which are seen almost solely as a complication of a more extensive affection of the urinary passages, such as pyelo-cystitis, we have a purulent, ulcerative inflammation, which may even assume a diphtheritic character. In these cases the kidneys are almost always coincidently involved—pyelo-nephritis. If the nephritic abscesses break into the pelvis of the kidney, there arises an ulcerative destruction of the renal tissue, so that the pelvis of the kidney is filled with pus and bounded by extensive ulcers, which often penetrate deeply into the substance of the kidney—pyonephrosis. The pyelo-nephritic abscesses reaching to the outer surface of the kidney, and usually giving a striated appearance to a section of the organ, have already been described (see page 641), and their bacterial origin mentioned.

The condition differs when the kidney is involved, as in many cases of chronic pyelitis. This appears most frequently as a result of retention of urine, and hence it is usually associated with a dilatation of the pelvis of the kidney. In these cases we sometimes find pronounced processes of contraction in the kidneys—that is, a partial atrophy of the renal tissue, increase of the interstitial connective tissue, and evident cicatricial depressions on the surface—in a word, a secondary contracted kidney, arising as a result of pyelitis, which differs from genuine contraction of the kidney only in its ætiology.

Clinical Symptoms.—Since in most cases pyelitis develops only as one symptom of a more extensive morbid process, its clinical symptoms are usually but slightly prominent in the general course of the illness. In what follows, therefore, we can not give any complete description of the clinical course of pyelitis, but we must mention only those symptoms from which, when there is an affection of the urinary passages, we may conclude that the pelvis of the kidney takes part in the morbid process.

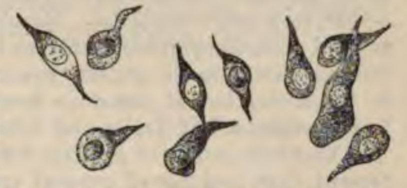

FIG. 73.—Epithelium from the pelvis of the kidney.

The most essential sign which the urine presents in all inflammatory affections of the urinary passages, the presence of mucus and pus, will be described more fully in the chapter on cystitis (*vide infra*). In pyelitis, also, the muco-purulent secretion of the pelvic mucous membrane must mix with the urine, and in every severe purulent inflammation the amount of pus in the urine must be considerable. We can never decide with certainty, from the mere presence of pus in the urine, as to the place where the pus mixes with the urine, whether in the pelvis of the kidney or in the bladder, or even in the urethra. If only we were able to demonstrate other morphological elements besides the pus-corpuscles, so characteristic that their origin might be affirmed with certainty to be the pelvis of the kidney, we might, by means of them, make our diagnosis of pyelitis absolute. Unfortunately, however, the microscopic sediment of the urine leaves much in this regard to be desired. The greatest weight was formerly ascribed to the discovery of pelvic epithelium; especially the triangular, long-tailed epithelial cells, sometimes arranged one upon the other, like the tiles of a roof (see Fig. 73), were regarded as an indication that the pelvis of the kidney was involved in the inflammation; but the diagnostic significance of these cells is by no means indubitable, for, on the one hand, they may be absent in a severe case of pyelitis; and, on the other hand, precisely similar epithelium comes from the mucous membrane of the bladder. Greater value is given of late to the discovery of certain casts from the mouths of the urinary canaliculi (*ductus papillares*)—structures which are almost invariably involved in all but the milder cases of pyelitis. Tubuliform epithelial casts, cylindrical formations composed of pus-corpuscles, and, above all, casts composed of micrococci, have been repeatedly found in the urinary sediment in cases of pyelitis, and possess some diagnostic importance (Fürbringer and others).

With regard to the other characteristics of the urine, it should be mentioned that it is often remarkably abundant in pyelitis, and that it then of course is pale and has a comparatively low specific gravity. The reaction of the urine is usually acid, despite the admixture of pus. That this acid reaction is an effectual distinction from the urine of cystitis can by no means be maintained (*vide infra*). We may, however, say that the tendency of the urine to ammoniacal fermentation is decidedly greater in cases of cystitis than in cases of pyelitis. The amount of albumen in the urine corresponds to the amount of pus. If there is a very large amount of albumen, it arouses suspicion of a coincident nephritis. The only decisive proof of such a complication lies in the finding of genuine urinary casts. In cases of simple pyelitis it is exceptional for the urine to be bloody, while in calculous pyelitis (*q. v.*) blood is often present.

Besides the quality of the urine, another symptom is pain in the region of the kidneys. This is present in many cases of pyelitis. The pain is often very severe, and radiates along the ureters to the bladder. In some cases, however, there may be no pain at all, so that while its presence is one symptom of pyelitis, its absence does not weigh against the disease.

All the other symptoms may be directly dependent upon the pyelitis, but they may usually be referred in great degree to the other co-existing affections. First among these is fever, which either shows an irregularly remitting course, or appears in single high elevations of temperature, usually associated with rigors. The fever, however, seldom shows this latter pyæmic character except in the severe purulent forms, where we usually also have the formation of renal abscesses —that is, a pyelo-nephritis. Besides the fever there are often, in severe cases, general nervous symptoms, such as headache, delirium, and sopor, which are to be referred partly to the general pyæmic infection of the body and partly, perhaps, to the absorption of ammonia from the decomposing urine into the blood—the "ammoniæmia" of Treitz and Jaksch.

The whole course of pyelitis differs so much according to the primary disease present that nothing of general application can be said about it. The milder forms, which often pass off rapidly, are found most commonly in childbed, and sometimes in acute infectious diseases, poisonings, and as a result of mild cystitis. The severe forms are chiefly, as we have said, pyelo-cystitis and pyelo-nephritis, as a result of strictures of the urinary tract (*vide infra*), of severe cystitis in diseases of the spinal cord, and in other severe diseases of the kidney and of the pelvis of the kidney, such as new growths, and parasites. They usually constitute a very tedious and incurable affection, which lasts until the patient's death.

The important factors in the diagnosis of pyelitis have been already pointed out. The main thing in every case is a careful consideration of the cause and the course of the disease, and, with regard to the special involvement of the pelvis of the kidney, a study of the changes in the urine, and the local symptoms if there be any. If the bladder itself is unaffected there will be no special bladder symptoms, such as tenesmus and frequent micturition. If there is severe disease of the urinary passages, and especially if the patient has considerable fever, we may often be justified in diagnosticating pyelitis, or pyelo-nephritis, even when there are no direct indications, because experience has shown that such an extension of the disease is the rule in all severe and long-continued cases.

The implication of the kidneys is shown by the presence of casts in the urine in addition to the pus-corpuscles. In the cases above mentioned, in which a chronic pyelo-cystitis is complicated with a contracted kidney, the condition of the urine is the same in many respects as in genuine contracted kidney. It is abundant, usually has a low specific gravity, and contains, besides the pus-corpuscles, a few short hyaline casts. In such cases there may be developed a secondary hypertrophy of the left ventricle, unless the general nutrition of the patient is too much impaired.

Treatment.—The treatment of pyelitis coincides mainly with the treatment of the primary disease—for example, renal calculi—and therefore it needs no detailed description here. Ordinarily only the accompanying cystitis (*vide infra*) is accessible to a direct local treatment, and here an important prophylactic factor is discovered, since by a timely treatment of the cystitis we can certainly hinder the advance of the inflammation to the pelvis of the kidney.

Among the internal remedies to which we ascribe a favorable influence on the mucous membrane of the urinary tract, which are therefore used in like manner both in pyelitis and in cystitis, we may mention the astringents, tannin, alum, and acetate of lead. Their therapeutic effects, however, are usually very slight, and often they are ill-borne, so that certain antiseptic remedies are preferable, particularly salol in capsules of eight grains (gramme 0.5) three to five times a day; [urotropine, ten grains three times a day]; also, camphoric acid and chlorate of potash. Balsamic remedies are sometimes useful, including turpentine, sandalwood, and copaiba. The details in regard to all these remedies will be found in the treatment of cystitis. The copious ingestion of fluids usually acts favorably, especially the use of certain mineral waters, among which the waters of Carlsbad, Vichy, Ems, Neuenahr, and Wildungen have obtained the most reputation. A methodical milk cure is also greatly to be recommended, especially when there are symptoms of irritation, such as pain on micturition.

Local applications to the region of the kidneys, warm poultices, or exceptionally local blood-letting, are indicated only when there is severe pain, and then, of course, narcotics must also be used under some circumstances. In this respect warm baths also do good service at times.

CHAPTER II

NEPHROLITHIASIS

(*Renal Calculus. Renal Gravel. Pyelitis Calculosa*)

Occurrence, Chemical Composition, and Ætiology of Renal Concretions.—The precipitated concretions of the urinary constituents which form in the pelvis of the kidney, and which, under some circumstances, may be passed from it with the urine, are designated, according to their size and nature, as renal sand, a fine, pulverized precipitate; renal gravel, gravel-like, granular concretions about the size of the ordinary coarse grains of sand, which can usually pass through the ureters without special difficulty; or renal calculi, the larger concretions. The last are about the size of a millet seed or a pea, but larger stones are occasionally seen which may even resemble actual casts of the pelvis of the kidney. We usually find a calculus in only one kidney, although both kidneys may be affected.

In regard to the chemical nature of renal concretions, they consist most frequently of uric acid. They then are hard, have a brown-red or blackish color, and a crystalline fracture, which in large stones is usually plainly laminated; and, on the whole, a smooth although irregularly-shaped surface. More rarely the renal concretions consist of calcic oxalate. The oxalate calculi are extremely hard, have a dark-brown color and a rough surface, often furnished with many prickles, from which reason they are often called "mulberry calculi." Their fracture sometimes has a radiated but never a laminated arrangement. Stones are also frequently seen, which consist of alternating layers of uric acid and calcic oxalate, or which have a nucleus of uric acid and a coating of calcic oxalate. The phosphatic calculi are another variety of renal concretions. We only rarely have to do, however, with stones which consist exclusively of basic calcic phosphate or ammonio-magnesic phosphate, but we oftener have secondary deposits of layers of phosphate which are precipitated on uric acid or mulberry calculi in urine which has become alkaline. The pure phosphatic calculi are grayish-white and rather soft, so that they can be crushed with the finger. The largest specimens of this kind are not found usually in the pelvis of the kidney, but in the bladder; still, as we have ourselves observed, large and pure phosphatic calculi do occur in the pelvis of the kidney, without any uric-acid nucleus. All the other calculous formations are so rare that they scarcely possess clinical interest. We may mention the light-yellow cystine calculi with a surface of waxy luster, the xanthine calculi, and the indigo calculi.

As to the precise causes of all these concretions we have as yet no certain knowledge. The uric acid probably originates from the nucleines. An increase in the destruction of leucocytes seems to be associated with an increased excretion of uric acid. The formation of a solid deposit of uric acid is usually associated with marked acidity of the urine. Yet, we do not know at all in what way these circumstances bring about the development of renal calculi. There is much probability in the assumption that some solid substance or other usually forms the nucleus, and gives the impulse to the formation of at least the larger renal calculi. Such nuclei may be coagulated mucus, bits of epithelium, and perhaps bacteria. It is an interesting fact, although we can not completely explain it, that, as Meckel, Ebstein, and others have shown, the microscopic examination of minute scales of calculi disclose the crystalline uric acid deposited in a supporting structure of an albuminous character. With regard to the formation of oxalate calculi, likewise, we possess no exact knowledge. Oxalic acid in the urine probably is due in part to the oxalic acid which is introduced into the system in vegetables, and in part is probably formed from the decomposition of albuminous substances. The deposits of crystals of calcic oxalate from acid urine is well known to be a very frequent occurrence, while the actual formation of calculi is, as has been said, comparatively rare. The cause of the deposit of phosphatic concretions must lie in the urine becoming alkaline. In such cases, therefore, the development of concretions is probably preceded by disease of the pelvis of the kidney, and the ingress of bacteria capable of exciting an alkaline fermentation of the urine.

In regard to the predisposing causes of calculus formation we must mention, first of all, that stones are often found in children, and next in frequency in advanced life. Men show a greater disposition to renal calculi than women. Heredity also seems to play a certain part, since the disease has been repeatedly observed in different members of the same family. The many relations which have been imagined between the formation of calculi and certain conditions in the manner of life and in the food taken, all lack definite proof. As to mode of life, the chief blame is laid upon an excessive meat-diet, drinking copiously of new sour wines, and drinking water containing lime. It is remarkable that calculi are much more frequent in some countries (England) and regions than in others. In regard to the occurrence of uric-acid concretions in gouty patients, compare the chapter on gout.

The Anatomical Changes caused by Renal Calculi.—The usual change which the presence of concretions in the pelvis of the kidney excites is pyelitis. This may exhibit all degrees, from a simple catarrhal inflammation to a diphtheritic or severe purulent inflammation of the pelvic mucous membrane. As a result of the mechanical irritation, there are quite frequently large or small hæmorrhages.

If a severe purulent pyelitis has developed, this may bring with it all the sequelæ with which we have previously become acquainted. In severe cases the process may involve the kidneys, when there arises a pyelo-nephritis, with a purulent breaking down of the renal tissue, and, under some circumstances, even a perinephritis, with extensive suppuration in the vicinity of the kidney, and with occasional perforation into the neighboring organs. If the renal calculi have previously passed outward, they are not found at the autopsy, although they form the special starting point of the disease. Sometimes, however, the pus-cavity is entirely filled with calculi.

A second important sequel of a renal calculus, which sometimes develops, is hydronephrosis (*vide infra*). It arises when a large stone blocks the passage from the pelvis of the kidney into the ureter, or when a smaller stone remains fast in the ureter and completely shuts off the passage of the urine. In the latter case there may also arise a pressure necrosis and perforation of the ureter. It goes without saying that inflammation and hydronephrosis or pyonephrosis may be combined.

Clinical Symptoms.—If there is merely the formation of renal sand or renal gravel in the urinary tract, this condition is sometimes associated with no symptoms at all. The little granules are washed away by the urine and evacuated, and at most they may give rise to slight pain in the region of the kidney. Larger stones, however, may sometimes be wholly, or almost wholly, without symptoms, if their position and their smooth surface are such that they are comparatively harmless.

The characteristic clinical symptoms of nephrolithiasis do not appear until the results of mechanical irritation of the pelvis of the kidney arise, or until there is an incarceration of a calculus in the ureter. It is the latter circumstance which, after the analogy of gall-stones, causes the most important symptom in the diagnosis of renal calculi—the pain, the so-called renal colic. Such an attack of colic sometimes comes on quite suddenly and unexpectedly; in other cases it is produced by some exciting cause—jumping, running, walking, or riding. The pain often becomes frightful; it radiates from the lateral portions of the abdomen along the course of the ureters upward and downward, spreading particularly toward the bladder, testicles, and thighs, and also up the back. In severe attacks there may be a general state of collapse with a small rapid pulse, cold sweat, and attacks of fainting. The temperature may be somewhat raised. We often see nausea and repeated vomiting. The urine is often scanty but sometimes entirely normal, inasmuch as it comes exclusively from the other free kidney; but oliguria, or even complete anuria, with its consequences, invariably sets in if both ureters be stopped. Still, even when one kidney is normal the amount of urine secreted by it may be scanty, as a result of a reflex inhibitory influence reaching it in some unknown way. If there is at the same time an inflammatory process, the urine may contain blood and pus. The duration of renal colic depends upon the duration of the incarceration; it may last for a few hours or several days. The attack often ends with the passage of the stone outward into the bladder.

The other symptoms occurring in nephrolithiasis refer mainly to the results of the mechanical irritation of the pelvis of the kidney. In most of the severer cases there is pain in the region of the kidneys, which is persistent, although of varying severity. The urine then shows an admixture of pus, and contains pelvic epithelium and often blood. The frequent appearance of blood in the urine, which usually has its cause in purely mechanical lesions of the mucous membrane, is a characteristic symptom of pyelitis calculosa. If we find, as sometimes happens, the urine at many times perfectly clear and normal, but at other times purulent, we may suspect an occasional blocking of the ureter coming from the diseased kidney by a renal calculus.

The symptoms are much more severe if the trouble goes on to a severe purulent pyelitis and pyelo-nephritis. We need not describe the details again here—the pain, fever, swelling, and perforation internally or externally—since they agree completely with what has been said before (see the previous chapter and Chapter VI in the previous section). A special chapter will be devoted to the symptomatology of hydronephrosis.

The course of nephrolithiasis is, as a rule, very tedious. Since the disposition to the formation of calculi usually persists, and since also the sequelæ which have once developed may last for a long time, a very chronic state often develops, which, in varying ways and with manifold exacerbations and remissions, is composed of attacks of colic, hæmorrhages, and symptoms of pyelo-cystitis.

In many cases, of course, complete recovery may finally ensue. The calculi present are passed, new ones are not formed, the pyelitis that has arisen disappears, and all the morbid symptoms cease; but, on the other hand, nephrolithiasis has also a number of dangers in itself, which threaten life very seriously. These are, besides the rare occurrence of uræmia, first of all the development of pyelonephritis and of still more extensive suppurations, with a general decline in strength, pyæmic states, etc. There is also a possible danger in such chronic suppurations that a general amyloid degeneration of the internal organs may ensue. With regard to complications on the part of other organs, there is only one point of special interest—viz., that sometimes gall-stones and renal calculi are found in one and the same patient.

Diagnosis.—Diagnosis is rendered absolutely certain by the discovery of renal calculi in the urine. For this purpose the urine must be examined as soon as possible after its evacuation, and the best method is by pouring it through a fine sieve. In many cases, however, we can make a tolerably certain diagnosis of nephrolithiasis, without the direct demonstration of concretions, by means of the characteristic clinical symptoms, particularly the periodical renal hæmorrhages, the attacks of colic, and in most cases the early appearance of pyelitis. If these symptoms are not well marked, it must be confessed that it is not always easy to distinguish renal colic from intestinal colic, biliary colic, neuralgia, or other painful abdominal diseases. It is possible to confound the condition with renal carcinoma and renal echinococcus. It is of great importance, particularly with regard to any operative treatment, to determine which kidney is the seat of the trouble, and whether one kidney, at any rate, is perfectly healthy or not. In considering these questions we must be guided by the chief seat of the pain; the appearance of the urine—for instance, observing whether there is an increase in the amount of pus if pressure is made upon the diseased kidney, and whether from time to time normal urine is evacuated as a result of occlusion of the ureter upon the diseased side; and by the results of external examination with regard to tenderness, or the discovery of a tumor due to hydronephrosis. In difficult cases the special catheterization of each ureter is sometimes practiced. Numerous methods of performing this have been devised, some of them involving an incision into the bladder, and cystoscopy has of late become so perfected that important information may be obtained by its means. With regard to the minutiæ as to these methods and their employment, we must refer the reader to special monographs.

In a few cases admirable diagnostic results have been obtained in case of renal calculi by examination with the Röntgen rays. It appears, however, that only the phosphatic calculi can be recognized in this manner, while the concretions of urates, which are of much more frequent occurrence, do not throw any distinct shadow.

Treatment.—Since the uric-acid concretions are by far the commonest, the methods of treatment most in use for nephrolithiasis refer especially to these.

If the tendency to the formation of urinary gravel be confirmed in a patient, or if the severer symptoms of nephrolithiasis have already appeared, we must first give a number of general dietetic directions, in order to check the formation of uric acid in general, and promote the solution of the uric acid already formed as far as possible. Without entering too much on theoretical reasoning, we will give in what follows the measures which have won general approval. In the first place, the patient must avoid immoderate indulgence in any sort of food, and particularly in meat. Articles of diet, such as liver, sweetbread, and calf's kidneys, which contain an abundance of nucleine, should be forbidden. The patient should be advised to choose a chiefly vegetable diet, with milk and a moderate amount of meat; spirituous beverages are to be used little, if at all, and acid viands and liquids are inadmissible. It is advantageous to weigh the patient regularly in order to keep watch on the nutrition, and to avoid any further increase of weight in such individuals as are well nourished; and in the obese, to bring about a diminution of weight. Moreover, so far as the general condition of the patient allows, he should have regular exercise in the gymnasium, or at sawing wood, or gardening. He should also promote the assimilation of his food by the frequent use of warm baths or mud baths; and an abundance of liquid must be ingested, so as to dilute the urine, and thus increase its solvent power.

This last indication is usually met, in conjunction with that of diminishing the acid reaction of the urine, by the ingestion of alkalies, so as to prevent, as far as possible, the deposit of uric acid; consequently, alkalies and alkaline mineral waters are very extensively employed in nephrolithiasis. Pfeiffer and others have been able to demonstrate by direct experiment that the urine secreted after the use of such waters and drugs does possess an increased power to dissolve uric acid. The simplest way is to have the patient use phosphate of soda in a daily dose of one to four drachms (grammes 5–15); or, better, carbonate of soda, one drachm to two drachms and a half (grammes 5–10); or, finally, the specially recommended carbonate of lithia, two to eight grains (gramme 0.1–0.5), several times a day, dissolved in a large amount of simple water or carbonated water, or lemonade. Also, mixtures of these remedies are often used with good results; thus Cantani recommends bicarbonate of soda, eight grains (grammes 0.5); effervescing carbonate of lithia, four grains (gramme 0.25); citrate of potash, fifteen grains (gramme 1); also the so-called uricedine, which is composed of the citrates of sodium and lithium, sulphate of sodium, and chloride of sodium. Of this, fifteen grains are to be given several times a day. Another remedy is borocitrate of magnesium, of which a teaspoonful may be taken in soda-water with syrup, three times a day. Of the natural mineral waters, the results of experiment point out the springs of Fachingen, Vichy, and Vals as specially efficient, but good results are also obtained in Carlsbad, Salzbrunn, Neuenahr, and Wildungen. The natural lithia waters of Assmanshausen and Salzschlirf contain such minute amounts of lithia salts that, in general, the artificial waters are to be preferred to them.

The attempt has been often made to promote the solution of uric acid by administering certain chemical substances, but unfortunately the results have not been brilliant. The conditions which obtain in the human organism are essentially different from those in a test-tube. Piperazine, fifteen to forty-five grains (grammes 1–3) daily, has been much recommended, but has not proved at all efficient, and the same may be said of lysidine, fifteen to sixty grains (grammes 1–4) a day, and urotropine, fifteen to twenty-five grains (gramme 1–1.5) daily, in water. Still, further trial of these remedies would not be unjustifiable.

The symptomatic treatment is very important. In so far as this relates to the accompanying pelvic and vesical catarrh, we may refer to the appropriate chapters in this book. For renal hæmorrhages some internal remedies, such as ergotine or tannin, have been recommended, but their action is quite doubtful. The treatment of the attacks of colic is of great practical significance. The chief remedies are the narcotics, opium, and morphine, internally, or, with very severe pains, better subcutaneously. In severe cases chloral and inhalations of chloroform may be employed. Warm baths, warm poultices, or narcotic embrocations, such as chloroform liniment, also frequently give relief. Local blood-letting is only rarely indicated. An abundant supply of fluid is always advisable, in order to aid the washing out of the incarcerated stone by an increased secretion of urine.

Since the initiative of Simon in 1871 the operative treatment of renal disease has made continuous progress, and it has been eminently successful in many cases of renal calculi. It is, therefore, most important in all instances of nephrolithiasis with severe symptoms, and particularly with secondary purulent inflammation, to consider the possibility of surgical interference by nephrotomy or nephrectomy. Particulars in these matters must be sought in special treatises on renal surgery.

What has been said thus far refers, as we have stated, chiefly to the treatment of uric-acid calculi. If there are calculi made up of oxalate, the amount of vegetables eaten must be limited, but the use of alkaline waters is advisable. We do not know any special directions to give in case there were cystine calculi. On the other hand, when phosphatic stones are present, inasmuch as these can not be deposited unless the urine is alkaline, we should recommend the employment of acids, particularly lactic acid, eight to fifteen grains (gramme 0.5–1), and salicylic acid; also, hydrochloric acid and phosphoric acid. Of course, in most cases there is some disease of the urinary passages which has occasioned the formation of the calculi, and if such disease is discovered its treatment is of paramount importance.

CHAPTER III

TUBERCULOSIS OF THE GENITO-URINARY APPARATUS

Ætiology and Pathological Anatomy.—It does not seem remarkable that, with the presence of many tubercular processes in the body, tubercle bacilli should quite easily reach the kidneys by way of the blood-current, and there give rise to an eruption of tubercle. Accordingly, we quite frequently find a few or many miliary tubercles in the kidneys in acute miliary tuberculosis, in pulmonary tuberculosis, etc., which are distributed over the whole kidney, or sometimes only in the territory of one arterial branch.

While miliary tuberculosis of the kidney, however, is without any clinical significance, there is also an extensive local tuberculosis of the kidney, as well as of the urinary tract and the sexual organs. Such affections sometimes occur as a result of pronounced pre-existing tuberculosis of other organs, especially the lungs, or they arise as an apparently independent disease, which is termed genito-urinary tuberculosis. In such cases the infection with the tubercle bacilli often seems to take place by means of the blood from some previously existing—perhaps concealed—tubercular focus in the body, such as a gland, tubercular bone or joint disease, etc. In other cases, the tubercle bacilli perhaps enter the urinary tract from without, but the point of the first anatomical lesion need not, apparently, always be the same. The kidneys often seem to be first diseased, in other cases the bladder, and quite frequently, as it seems, the prostate, and sometimes perhaps the vesiculæ seminales or the testicles. From the organ first affected the process then extends continuously or by leaps to the neighboring parts. If the cases come to autopsy, the tuberculosis is often so extensive that we can no longer make out with certainty the place where it first began. In women the urinary apparatus is only very rarely affected by tuberculosis, while uterine and ovarian tuberculosis represents a localization of the tubercular poison of clinical importance.

In the kidneys the tubercular infiltration develops either chiefly from the pelvis of the kidney or in the renal substance itself. Yellow cheesy nodules arise, which finally break down and thus lead to an actual "nephro-phthisis." If the disease arise from the pelvis, the infiltrated renal papillæ are usually first affected. Ulcerative recesses are formed in the pelvis of the kidney, and finally the entire lining of the pelvis is transformed into a raw surface covered with necrotic tissue and cheesy detritus. In very advanced cases almost the whole kidney is destroyed. The process is usually bilateral, but it is often more advanced on one side than on the other.

If the process invade the ureters, their walls also are infiltrated with tubercular deposits, and hence they are thickened, while the mucous membrane is often changed in great part to a necrotic ulcerating surface. Precisely analogous conditions are found in the bladder, and in some cases even in the urethra; while in the prostate, the vesiculæ seminales, and the testicles there is more frequently the formation of cheesy tubercular nodules, and rarely disintegration and perforation.

Clinical Symptoms.—The picture of genito-urinary tuberculosis corresponds in most of its details completely to that of a severe chronic pyelo-cystitis. The occasional local symptom is pain in the region of the kidneys and bladder. This may sometimes assume great severity, like colic, if the ureter become plugged by a broken-down, crumbling mass; yet in other cases the pain is but slight during the whole disease.

The urine shows the most important changes. It almost invariably contains an abundant sediment, consisting of pus-corpuscles and detritus. Its amount usually remains normal for a long time; its reaction is faintly acid, but in severe cases it may become alkaline through complication with an alkaline fermentation of the urine. The discovery of shreds of tissue in the urine, elastic fibers and connective tissue, is sometimes possible, and is of diagnostic value because it is direct evidence of an ulcerative process. The discovery of tubercle bacilli in the purulent urinary sediment (Rosenstein and others) is, however, far more important. This is possible in almost all cases, and is a reliable and absolutely decisive factor in diagnosis. There is, however, one unfortunate circumstance about the demonstration of tubercle bacilli in the urine, for not infrequently other bacilli (smegma bacilli) are stained by the ordinary method of staining, which is that employed for sputum, and are thus mistaken for tubercle bacilli. As yet, no easy and certain method of distinguishing these two varieties of bacilli has been discovered, and hence in doubtful cases it is necessary to resort to pure cultures and to inoculation, in order to reach a decision. [Stain with carbol-fuchsine; decolorize with twenty per cent. nitric acid; wash in water; and still further decolorize in seventy per cent. alcohol for at least ten minutes. This will bleach the smegma bacilli.—V.]

Admixtures of blood in the urine are also seen in genito-urinary tuberculosis, but they may often be entirely absent. In several of our cases a slight hæmaturia was the first symptom which called the patient's attention to the trouble with the bladder.

The local objective examination of the kidneys usually gives a negative result. Only in a few cases have we been able to feel the diseased kidney as a tumor through the abdominal walls. This is usually due less to the tubercular infiltration of the kidney itself than to the dilatation of the pelvis of the kidney from hydronephrosis. We can sometimes feel the thickened walls of the bladder. The local examination of the prostate and the testicles is far more important in diagnosis. Especially in the latter we often feel the hardening corresponding to the tubercular infiltration, and manifesting itself chiefly in the epididymis, while the hardening and enlargement of the prostate and seminal vesicles can usually be easily detected by rectal palpation.

Among the general symptoms we must mention, first of all, fever, which is only exceptionally absent, and usually, in the severe cases, shows a pronounced remitting, hectic character. The other general symptoms are the same as in most of the other tubercular diseases—anæmia, emaciation, loss of appetite, increasing bodily weakness, etc. We have a special sign in the occasional co-existence of other tubercular diseases in the body, the lungs, the intestines, the bones, etc., but these may also be wholly absent, so that we may have to do with a purely local genito-urinary tuberculosis.

The course of the disease is steadily progressive. Recovery does not occur, at least not in any cases where the disease has attained any extent. The disease lasts from a few months to a year or two, but sometimes much longer. The fatal termination usually ensues from the increasing general weakness, more rarely under the symptoms of ammoniæmia, or sometimes from a miliary tuberculosis or some other tubercular disease, such as pulmonary tuberculosis, tubercular meningitis, etc.

Diagnosis.—The diagnosis of genito-urinary tuberculosis is now seldom difficult in fully developed cases, since it can be made with complete certainty by the discovery of the tubercle bacilli joined to the presence of pus in the urine. Of course this gives no information as to the exact distribution of the process. In order to judge of this, we must add the local symptoms and the physical examination of the different organs. We are aided in the confirmation of our first suspicion of a tubercular disease chiefly by the consideration of the general condition and the habit of the patient; the discovery of a hereditary taint, or at least the probability of tubercular infection; and also the discovery of other tubercular affections, especially in the testicles; the hectic fever; and the tedious course, upon which nothing has a favorable influence. At any rate we must make it a rule, in every case of persistent pyuria which can not be otherwise explained, to examine the purulent sediment for tubercle bacilli (*vide supra*). We may then often be able to recognize with certainty the milder and incipient cases of this not very rare affection. [Another valuable diagnostic method, briefly referred to by the author, is by inoculation of eight to fifteen minims of the urinary sediment into the peritoneal cavity of a guinea-pig. If tubercle bacilli are present, the animal will develop tuberculosis in six to eight weeks.—V.]

Treatment.—Whether specific treatment of genito-urinary tuberculosis with Koch's tuberculine is likely to have permanent success can not yet be determined, as so few trials have been made. Nevertheless, it would be justifiable to make a cautious trial of the remedy, although we can hardly cherish the hope that it would prove very beneficial. Beyond this we must resort, in the treatment of tuberculosis of the urinary passages, to the same remedies as in ordinary chronic pyelitis and cystitis. Of internal remedies we have most frequently used chlorate of potassium and turpentine, and have sometimes seen good results, especially from the latter. The internal exhibition of creasote sometimes seems to us to act well. [Urotropine has also been recommended.—V.] In vesical tuberculosis it is well to wash out the bladder. As an operative procedure we may remove tubercular testicles and epididymes and one kidney, but we must remember that in such cases there is usually co-existing disease of other parts of the genito-urinary apparatus, such as the prostate, the other kidney, etc.

CHAPTER IV

HYDRONEPHROSIS

(Dilatation of the Pelvis of the Kidney)

Ætiology.—If a contraction arises in any part of the urinary tract and checks the flow of urine, there is a stasis of the urine in the portion behind the stenosis, which gradually leads to a constantly increasing dilatation of the tract as a result of the pressure of the retained fluid.

In general, it is evident that gradual constrictions of the urinary tract and periodic obstructions, as from calculi, interrupted by free intervals, lead to more marked degrees of hydronephrosis than rapid and complete obstructions. Under the first-named circumstances the renal secretion persists much longer and is more abundant than in the latter case, when it usually soon ceases. Nevertheless, there may be even then a very slow distention of the pelvis of the kidney, particularly if its mucous membrane continues to secrete. If the obstruction is located in the ureter, the proximal portion of that canal dilates, and still more the pelvis of the kidney, giving rise to what is called hydronephrosis; but if the obstruction is located in the urethra there is a gradual dilatation of the bladder, both ureters, and finaly there develops a bilateral hydronephrosis.

A closure of the ureter arises most frequently in adults from impacted renal calculi, and also from new growths in the vicinity, in the uterus or ovaries, which compress the ureter from without. So great a pressure may also be exerted on the ureters by the gravid uterus as to be followed by a hydronephrosis, which is usually bilateral. Cicatricial strictures, valve-formations and bends, also are found in the ureter, and form an obstacle to the flow of urine. Finally, in cancer of the bladder the lower opening of the ureter may be contracted or entirely closed. Constrictions of the urethra, which finally lead to a bilateral hydronephrosis, arise most frequently from strictures as a result of gonorrhœa, and also from enlargements of the prostate. In rare cases phimosis may form the obstacle.

It is worthy of note that hydronephrosis may also be congenital, and then it is usually due to congenital defects of development in the ureters or other urinary passages. In later life hydronephrosis is in general more frequently observed in women than in men.

Pathological Anatomy.—The pathological anatomy of hydronephrosis is on the whole very simple. We have a dilatation of the pelvis of the kidney, which is associated with a pressure atrophy of the renal tissue. The papillæ are flattened, the uriniferous tubules and the glomeruli are gradually more and more obliterated, and finally the whole kidney may be changed to a connective-tissue sac filled with fluid, and displaying in its walls a few vestiges of the renal parenchyma. The size of such a hydronephrotic sac may sometimes be sufficient to contain ten or fifteen quarts (litres) of fluid. The latter consists, of course, at first of urine, but the farther the atrophy of the kidney advances, the more it merely represents the secretion of the mucous membrane. Inflammatory conditions are found in hydronephrosis only when they have existed previously, as in pyelitis calculosa, or when excitants of inflammation in addition have reached the pelvis of the kidney. We then have a pyonephrosis.

Clinical Symptoms.—Since the whole type of the disease is, of course, dependent in many respects upon the nature of the primary disease, we have here to describe only those symptoms which point to the development of hydronephrosis. Such a condition often causes no special clinical symptoms at all, so that we can at most suspect its existence from the presence of an ætiological factor.

The appearance of a visible and palpable tumor is the first definite point in the diagnosis of hydronephrosis. This first shows itself in the region of the affected kidney, but then it gradually enlarges toward the hypochondrium and the median line of the body, and it may finally reach very considerable dimensions. Hydronephrosis on the left side does not usually move with respiration. On the right side, however, there may be a distinct downward motion upon deep inspiration. The resistance of the hydronephrotic tumor is usually quite considerable, but there may sometimes be a distinct sense of fluctuation. On percussion, the tumor gives a dull note, from which the tympanitic note of the colon in front of the tumor is sometimes distinct (see page 645). It is an important diagnostic sign if the tumor show variations in its size at times—decreasing in size with a simultaneous increase in diuresis, and increasing again when the amount of urine becomes smaller ("intermittent hydronephrosis"). In such cases there is usually a very peculiar alternation in the other clinical symptoms. At the time of the scanty secretion of urine the patient suffers from violent pain, vomiting, chills, and similar symptoms, while upon the appearance of an abundant urinary secretion, all these symptoms quickly vanish. In doubtful cases an exploratory puncture of the tumor may also be of significance in diagnosis. It of course favors the assumption of hydronephrosis if urinary constituents, especially urea, can be found in the fluid evacuated; but if the hydronephrosis be of long standing, its contents, as we have said, will be simply sero-mucous, and then chemical examination gives no definite data for distinguishing hydronephrosis from ovarian tumors, or other cystic tumors of the kidney.

The secretion of urine in unilateral hydronephrosis may be completely normal if the other healthy kidney acts vicariously. In stricture of the urethra, and also in bilateral constrictions of the ureters, however, there is, of course, an obstacle to the passage of urine, so that the amount of urine may be abnormally small. There may be at times complete anuria, and even uræmic symptoms. The composition of the urine depends entirely upon the form of the primary disease. If the healthy kidney alone secretes, the urine passed is normal. If there be at the same times pyelitis or cystitis, the urine may contain pus or blood. If the urine can also come from the diseased kidney at one time and not at another, the urine also exhibits a varying composition, as we have said before (page 660).

In many cases of hydronephrosis quite severe local symptoms are constantly present; there are frequently severe pains in the tumor, which shoot chiefly toward the thigh. Still, these local symptoms are sometimes surprisingly slight. In regard to the symptoms on the part of other organs, gastric disturbances appear to be of the most frequent occurrence; among them are nausea, loss of appetite, vomiting, and eructations. In some cases the bowels are constipated, in others there is obstinate diarrhœa. It is a very interesting fact that, particularly in case of bilateral hydronephrosis, there may be developed a distinct hypertrophy of the left ventricle, with all its sequelæ. The explanation of its occurrence, in our opinion, is precisely the same in this case as in chronic nephritis (*vide supra*, page 602)—viz., the toxic effect of the urinary constituents retained in the blood.

The whole course of the disease is always chronic. There are often variations in its course, but no general statements can be given, because the conditions vary in the different cases according to the form of the primary disease. Most cases of hydronephrosis end fatally, either in consequence of the primary disease or in consequence of secondary pyelonephritic or perinephritic inflammations, of uræmia, etc. Recovery takes place in rare cases, especially if one kidney be perfectly normal, and there be no incurable primary disease. Recovery may ensue spontaneously from perforation or obliteration, or it may be brought about artificially from operative procedures.

In the **diagnosis** of hydronephrosis, the points especially to be considered have already been mentioned. The diagnosis is usually not easy, especially if the ætiological factors be unknown; and the disease is often confused with other renal tumors and echinococci of the kidneys, with ovarian tumors, and even with splenic and hepatic tumors.

Treatment.—Except for the symptomatic treatment of the pain and any accompanying pyelo-cystitis, an efficient treatment of hydronephrosis can be attempted only by surgical means, the details of which are to be found in the special surgical treatises.

CHAPTER V

CYSTITIS

(*Vesical Catarrh*)

Ætiology.—In most cases of vesical catarrh the agents of inflammation reach the bladder from without through the urethra. The most unequivocal demonstration in this regard is, unfortunately, often made by the physician himself, when he excites a cystitis by the use of an insufficiently purified and disinfected catheter or bougie. The development of the vesical catarrh is generally aided in such cases by the fact that there is usually a defective evacuation of urine, from stricture of the urethra or paralysis of the detrusor, with a consequent retention of urine, in which the bacteria can develop undisturbed. The agents of inflammation may also enter from the urethra into the bladder in incontinence of urine. On account of the imperfect closure of the sphincter, a stagnating column of urine, directly connected with the contents of the bladder, forms in the urethra, and to this column the air and the bacteria that excite decomposition of the urine have direct access. In this way many cases of cystitis arise in patients with nervous disease who have paralysis of the bladder, and also many cases of cystitis arise in this way in persons who are severely ill and stupid from some other disease, such as typhoid fever.

Cystitis often follows diseases of the neighboring portions of the urinary tract. Among these, gonorrhœal urethritis is the most common, and this invades the bladder directly and leads to a gonorrhœal cystitis. In this case, it has not yet been absolutely determined whether the gonococci themselves invade the bladder and occasion cystitis, or whether the inflammation is due to the secondary introduction and dissemination of other pyogenic cocci, such as staphylococci (*vide infra*). It is, moreover, a fact of great importance that the development of cystitis is decidedly promoted by the unskilful employment of urethral injections. In women, the agents of inflammation may quite easily enter the bladder from the vagina through the short female urethra. Thus arise especially the frequent cases of cystitis in childbed. In some cases communication may develop between the bladder and certain neighboring organs, as in vesico-rectal or vesico-vaginal fistulæ, by which again access to the bladder is opened to the agents of inflammation.

Another group of cases is due to the presence of foreign bodies, which irritate the vesical mucous membrane mechanically. Among these is, first of all, the cystitis which so often accompanies stone in the bladder. It must be stated, however, that probably these cases of vesical catarrh are not directly dependent upon the calculi, but are first excited by examination with catheters and sounds, and in brief are due to secondary infection.

In distinction from the methods of origin of cystitis so far described, the production of inflammation by way of the blood-supply is much rarer. Certain chemical substances, already mentioned (page 654), which are eliminated by the kidneys and provoke an inflammation of the urinary tract, are the most important in this respect. Cantharides shows the most intense action of this sort, and it may cause an actual croupous cystitis. Slight irritative states of the bladder also frequently appear after taking certain foods and drinks, as after drinking new beer. Infectious substances only rarely come under consideration in this regard. Most of the cases of cystitis in severe acute infectious diseases are secondary complications due to germs reaching the bladder through the urethra (*vide supra*). It can not be doubted that in some cases an apparently idiopathic primary cystitis appears after exposure to cold, but it is very rare. In such cases we usually have to do with acute exacerbations of an old chronic cystitis—for example, of gonorrhœal origin.

As to the special variety of inflammatory germs in cystitis, our knowledge is as yet incomplete. In many cases, particularly in the cystitis of puerperal women, the germs are perhaps the ordinary pyogenic cocci (streptococcus and staphylococcus pyogenes). In gonorrhœal cystitis the invasion of the gonococcus into the bladder has been absolutely demonstrated in some cases. Moreover, the bacterium coli commune seems to play a great rôle in cystitis. The development of ammoniacal fermentation of the urine (*vide infra*) is, nevertheless, not due to the bacterium coli, but in most instances to special micro-organisms (formerly termed by Cohn "micrococcus ureæ"), among which the proteus deserves prominence. The staphylococcus pyogenes also has the power of exciting ammoniacal fermentation.

It has been stated in the previous chapters how frequently cystitis is only one symptom of a more extensive disease of the urinary tract. As cystitis may invade the pelvis of the kidney through the ureters, so, on the other hand, any pyelitis of primary origin may extend downward and involve the bladder.

Pathological Anatomy.—The pathological anatomy of cystitis presents the same conditions as the inflammation of any other mucous membrane. In simple catarrhal cystitis the mucous membrane is swollen and covered with pus, and is often studded with hæmorrhages. In chronic cystitis the mucous membrane sometimes takes on a granulated appearance, because of the swelling of numerous follicles, and in other cases assumes a slaty, grayish-black color, because of the pigmentation resulting from numerous small hæmorrhages. The severer forms of cystitis, such as are often observed in diseases of the spinal cord, are termed vesical diphtheria. These cases come to a necrotic destruction of the superficial layers of the mucous membrane, ulcerations, etc. In such severe cases submucous and pericystitic abscesses sometimes develop, which may perforate into the surrounding parts in various ways. The incrustation of the mucous membrane with urinary salts, especially with ammonio-magnesic phosphate, is also frequently found in chronic cystitis, and is worthy of mention. If the disease of the bladder is associated with a stricture of the urethra, the bladder is usually much dilated, and the muscular layer is hypertrophied and stands out in ridges on the inner surface of the bladder.

Clinical Symptoms.—The local symptoms are sometimes quite severe in cystitis, but in other cases they are only slight. In general, they show a greater intensity in acute cases than in chronic cystitis. The pain in the region of the bladder is rarely continuous; it usually occurs only on micturition, but it is often very distressing then, and shoots to the opening of the urethra. Since the inflamed vesical mucous membrane shows an increased irritability, and since the morbidly altered urine (*vide infra*) also exerts an abnormal irritation on the mucous membrane, there is very often an increased desire to micturate. The patient has to empty the bladder much oftener than normal, and in severe cases there is an almost constant, painful "vesical tenesmus," and at every attempt to micturate, only a very small amount of urine is passed, with severe burning.

As a result of the increased irritability of the vesical mucous membrane, there sometimes comes on a very troublesome reflex spasm of the sphincter, by which the symptoms are increased.

The character of the urine is alone decisive in the diagnosis. This is secreted in a perfectly normal amount and quality, in case there is no complication on the part of the kidneys; but in the bladder it is mixed with the products of the diseased mucous membrane, and it is here exposed to the action of the bacteria in a way that will presently be described. The abnormal constituents of the urine are chiefly pus-corpuscles, bladder epithelium, and mucus secreted by the mucous membrane. As a rule, the urine of cystitis contains innumerable bacteria, under the influence of which develops that important chemical transformation of urea into carbonate of ammonia, which is characteristic of all severe cases of cystitis, and which has been termed the alkaline (ammoniacal) fermentation. This formation of carbonate of ammonia decidedly aggravates the inflammation. As Lépine and Roux have shown, we can produce a severe cystitis, and even nephritis, by injecting very small amounts of a pure culture of micrococcus ureæ into the bladder of a guinea-pig. The stagnation is a factor which greatly aids the whole process, since the activity of the bacteria, as we have said, can develop much better than if the bladder were to a certain degree constantly purified and washed out by the urine; but cystitis can not be produced by mere retention of urine.

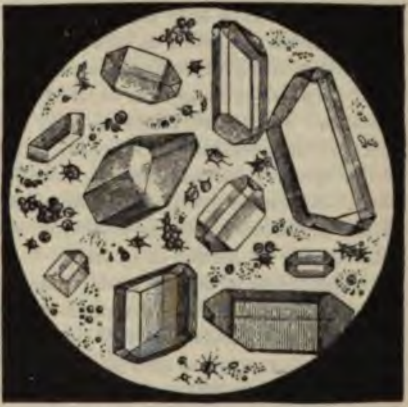

FIG. 74.—Crystals of triple phosphate and ammonic urate. (From FUNKE.)

As soon as a part of the urea is changed to carbonate of ammonia, the urine must be less acid in reaction. The urine has a faintly acid or neutral reaction, and sometimes it is decidedly alkaline when passed. The latter, however, is only rarely the case, but it is often simulated by the fact that the urine is not examined until it has stood for some time. Since during this time the alkaline fermentation makes rapid progress, the urine that has stood is very often alkaline. Many crystals of ammonio-magnesic phosphate and urate of ammonium then form in it; the former are easily recognized by their "coffin-lid shape," and the latter by their "thorn-apple shape" (see Fig. 74). There is also a deposit of amorphous phosphates.

If we then briefly sum up what has been said, the urine is passed in about the normal amount in cystitis. It usually looks pale, and has an abundant sediment, which can often be recognized as purulent with the naked eye, and in which, microscopically, we can find pus-corpuscles, often bladder epithelium, and constantly innumerable bacteria—usually short rods in vigorous motion. The alkaline fermentation may usually be recognized by the strong ammoniacal odor, and also, as we have said, by the reaction of the urine. In the severe diphtheritic forms of cystitis we find shreds of necrotic tissue in the urine. If there are hæmorrhages in the bladder, the urine often contains red blood-corpuscles and sometimes even large blood-clots. The mucus in the urine appears in milder cases as a cloudy opacity—"*nubecula.*" The viscid masses which can be drawn out into threads, and which are usually abundant in the urine in severe cystitis, and sometimes form gelatinous casts corresponding in shape to the bottom of the vessel containing the urine, are not mucine, but they arise from the pus-corpuscles and the epithelium, changed and dissolved in the alkaline urine, and hence give the reactions for albumen. It goes without saying (compare page 590) that cystitic urine is always albuminous from its mixture with pus-serum. The presence of slimy threads in the urine—the so-called "clap-threads" (*Tripperfäden*)—is characteristic of gonorrhœal cystitis.

There can be no doubt that, as already stated, the decomposing alkaline urine acts as a chemical irritant on the vesical mucous membrane. Hence cystitis often arises perhaps in this way, that the bacteria which have entered the bladder first excite only an alkaline fermentation, and that then the mucous membrane is affected by the irritation of the ammonia salts that are formed. Sometimes, however, the mucous membrane endures this irritation for a long time, and this explains the fact that alkaline fermentation may be observed in cases which, upon autopsy, present an almost normal condition of the vesical mucous membrane. In addition to the alkaline fermentation of the urine, it seems probable that many other processes of disorganization may develop in the urine, as is indicated by the various sorts of bad odors which the urine of severe cystitis may have. In a few cases there is even a formation of gas (carbonic dioxide, nitrogen, hydrogen), giving rise to pneumaturia. This indicates the development of special bacteria which generate gas. If the patient is also suffering from diabetes mellitus, the pneumaturia may be due to the fermentation of the sugar in the bladder.

The other morbid symptoms associated with cystitis usually depend only in part upon the disease itself and in part upon some existing primary disease. The most important symptom is the fever, which is often to be referred directly to the cystitis. In severe cases it may be very intense, and often assumes a pyæmic intermittent character, especially if there have arisen pericystitic suppurations or if the cystitis has extended to the pelvis and parenchyma of the kidney (see page 654). An acute cystitis also may begin with a chill and high fever. If the escape of the purulent urine, however, is absolutely free, fever may be entirely absent in spite of the existence of cystitis.

Sometimes in severe cystitis with a marked alkaline fermentation certain nervous symptoms appear, such as headache, vertigo, stupor, and nausea. The idea has been advanced that in these cases we have to do with an auto-intoxication of the body, since ammonia and perhaps other products of decomposition, such as sulphuretted hydrogen (?), are absorbed from the bladder into the blood (ammoniæmia), and in this way excite the symptoms of poisoning mentioned.

According to the course of the disease we distinguish an acute and a chronic cystitis. The former, which may come on, for example, after catheterization, in gonorrhœa, etc., often terminates favorably after a few days or weeks. The amount of mucus and pus in the urine is never great. Chronic cystitis is observed especially as a complication in other diseases of the urinary tract, such as stricture, chronic diseases of the spinal cord with paralysis of the bladder, etc. It is very often incurable because the primary disease is incapable of improvement and the cause of the disease therefore persists. The longer a cystitis lasts, the greater is the possibility of the development of severe and dangerous complications, especially the development of a pyelo-nephritis, and the formation of pericystitic suppurations. In this way cystitis, especially in chronic nervous diseases, may become the immediate cause of death.

Treatment.—The dangers last mentioned must urgently impress upon us the prophylaxis of cystitis. Fortunately, a good deal can be done in this respect, in the first place, by the avoidance of all unnecessary use of bougies and catheters, by the greatest care for cleanliness in the use of all instruments of this sort, and by the timely treatment of all those conditions which may lead to cystitis.

The treatment of cystitis is, in the milder and acute cases, hygienic and medicinal, but the severer cases demand careful local treatment.

In any severe, and especially in any acute cystitis, the greatest bodily rest (if possible rest in bed) is urgently desirable, since otherwise an increase of the symptoms and a prolongation of the course of the disease is the almost inevitable result. The diet must be mild and unirritating. Spiced food and alcoholic drinks are to be avoided, but we should recommend an abundant supply of fluid, by which the urine is diluted and the bladder washed out. We have the patient drink plenty of ordinary water, tea (made from uva ursi, etc.), or a suitable mineral-water, like Wildunger or Fachinger. A diet mainly of milk is very good; under it, improvement is often surprisingly rapid.

Among internal remedies those are to be considered which are eliminated with the urine, and are thus able to act on the diseased mucous membrane, or directly upon the agents of inflammation and the ammoniacal fermentation. One of the most efficient drugs, which never does harm if proper caution is exercised, is chlorate of potassium, of whose favorable influence on vesical catarrh we have often convinced ourselves. It is prescribed in an aqueous solution, forty to seventy-five grains a day (grammes 3–5), and it should never be taken on an empty stomach. Far less efficient than chlorate of potash are the true astringents, such as tannin and uva ursi, which were formerly much employed. The latter drug is said by Lewin to contain a glucoside, arbutine, which is itself recommended in doses of forty-five to sixty grains a day (grammes 3–4), but its efficacy is doubtful. Of metallic astringents, acetate of lead in powders of half a grain to a grain and a half (gramme 0.03–0.1), repeated several times a day, is at present seldom employed. In cases with a decided purulent secretion, if the initial symptoms of irritation have abated, we may often prescribe with good results resinous drugs, of which the best are oil of turpentine, in capsules or in milk, or balsam of copaiba. If, on the other hand, the ammoniacal fermentation is more prominent, salicylic acid in daily doses of thirty to sixty grains or more (grammes 2–4) in capsules, and salol, in powders of eight grains (gramme 0.5), repeated several times a day, are specially to be recommended; naphthaline, a grain and a half to five grains (gramme 0.1–0.3), repeated several times a day, has also been recommended, but it may produce decidedly disagreeable symptoms. [In the same class with the remedies just named, but far superior to them, is urotropine, which may be given in doses of ten to fifteen grains in water, after meals.—V.]

If there are severe local symptoms, we prescribe warm compresses and poultices to the region of the bladder. In robust persons with acute cystitis, local blood-letting (three to six leeches to the perineum) sometimes has a decidedly favorable symptomatic action in such a case. In other respects narcotics, especially subcutaneous injections of morphine, are the best remedy when there is severe pain and tenesmus. Camphor, extract of belladonna, etc., are much more uncertain in their action. The frequent use of protracted warm baths may, however, be greatly recommended.

In chronic cystitis all the remedies previously mentioned are also to be considered; but they are usually not sufficient alone, and at any rate they are far less effective than a methodical local treatment. This consists in a regular daily washing of the bladder by means of an elastic catheter. A rather long rubber tube is attached to the catheter at one end, and to a funnel at the other, and by raising or lowering this funnel the bladder can be filled or emptied. We allow a moderate amount, say three to seven ounces (100 to 200 cubic centimetres), of the warm fluid to run into the bladder and escape, repeating the process until the wash-water is clean. For this, perhaps we employ either a simple one-half to one-per-cent. solution of common salt, or preferably a warm and weak solution of acetate of lead, 1 to 1,000; permanganate of potash, 1 to 1,000; or a combination of boracic and salicylic acids. By treatment of this sort many cases of chronic vesical catarrh may be cured, while the most obstinate may, at any rate, be kept within bounds. In rebellious cases of purulent cystitis it is advantageous to employ lunar caustic. The bladder is rinsed out, and then about five ounces (grammes 150) of a weak solution of nitrate of silver, at first 1 to 3,000, later as strong as 1 to 1,000 or 1 to 500, are injected through a catheter. The solution is allowed to remain two or three minutes in the bladder and then withdrawn. What is called the Janet method of irrigating the bladder is also very useful. The fluid is placed in a fountain syringe, and made to enter the bladder from the urethra, by raising the reservoir high enough to give a pressure which overcomes the sphincter of the bladder. The advantage of this method lies in the coincident dilatation and lavage of the posterior portion of the urethra.

It is very important, in treating chronic catarrh of the bladder, to consider the possible cause of the condition—for instance, stricture, or vesical calculi, or paralysis of the bladder.

In pericystitic suppuration surgical treatment is only rarely possible. We must, therefore, confine ourselves to purely symptomatic procedures.

CHAPTER VI

NEW GROWTHS IN THE BLADDER

1. **Cancer of the Bladder.**—Primary carcinoma of the bladder is rare. It forms localized tumors, sometimes with a pedicle; or it extends diffusely over the wall of the bladder, and in that case leads to such thickening that the bladder can sometimes be felt through the abdominal walls as a firm tumor. Secondary carcinoma of the bladder is not infrequently due to direct involvement by extension from neighboring parts, such as the uterus, prostate, or rectum.

The early symptoms of primary cancer of the bladder consist of disturbances of micturition, which are often difficult to explain. Usually the first indication of the special local disease consists of a change in the character of the urine, which is probably coincident with the beginning of ulceration in the tumor. Then the complete picture of severe chronic cystitis is rapidly developed, a particularly characteristic symptom being the frequent, though irregular, appearance of blood in the urine. The condition becomes most distressing if the vesical termination of the urethra is occluded by the new growth. In the cases which we have seen, cancerous cachexia did not develop until rather late. One case was in the person of a quite young man. In general, the entire course of the disease seldom occupies more than one or two years.

The **diagnosis** of vesical cancer is not always easy, particularly at first. The main point is, that when there is a chronic disease of the bladder and no other sufficient reason for it is discovered, we should think of the possibility of tuberculosis or cancer, and make a careful examination with these severe conditions in mind. It is sometimes possible to confirm the diagnosis by finding bits of the tumor in the urine, but this is not invariably possible; consequently a number of methods have been elaborated to facilitate early recognition of vesical tumors by means of a direct and careful examination of the bladder—thus we have: cystoscopy (Nitze); exploratory cystotomy; and in women dilatation of the urethra and digital examination of the bladder. Particulars with regard to these procedures and their value are to be found in monographs, and in them the reader can also inform himself as to the results of the attempts which have thus far been made to remove vesical tumors by operation.

2. **Papilloma** of the bladder is usually a very soft fibroma, which is located in the trigonum or fundus and has delicate waving papillæ, which are covered with

several layers of cylindrical epithelium. The local discomfort which this not very rare form of tumor occasions is often slight, but the hæmorrhage it causes may be very important, being chronic and obstinate; for months and years the hæmaturia may be more or less continuous. The clotted blood as it passes through the urethra often assumes the shape of a worm. There is not much disturbance of micturition unless portions of the tumor obstruct the inner end of the urethra. Any one who has had the experience which we have had of seeing an otherwise healthy and vigorous man die because of persistent hæmorrhage from a papilloma hardly as big as a walnut, can not insist enough upon the value of an early and certain diagnosis (reached by examining portions of the tumor, or, still better, by cystoscopy) and of surgical treatment.

CHAPTER VII

ENURESIS NOCTURNA

(*Nocturnal Incontinence of Urine*)

Enuresis nocturna is a nervous affection of the bladder by no means rare in children of both sexes, and therefore quite important in its practical relations. Of course, in small children there is no sharp boundary to be drawn between normal and pathological conditions; but it is decidedly pathological if larger children, from four to ten years of age and even older, pass their urine in bed more or less frequently during sleep, in spite of well-developed reasoning powers and professedly the best intentions. This may extend to the years of puberty and even beyond, and then it frequently produces a very depressing mental influence upon the patient. Special causes for it are not to be discovered in most cases. We are compelled to assume either an abnormal weakness of the sphincter, which is probably sometimes congenital, or an abnormal irritability of the detrusor. Sometimes, but not invariably, the child has a neurotic constitution. At any rate, in wetting the bed at night the process of micturition comes on in a purely reflex way, but it is often accompanied by certain ideas in dreams referable to micturition. It does not hold in all cases that the sleep is especially deep. Many patients, of course, do not notice the mishap till morning, but others almost always wake directly after. The involuntary micturition usually occurs in the first hours after going to sleep, but sometimes it is later, and even toward morning. By day micturition is often perfectly normal; but in many cases there is even then a noticeable weakness of the bladder, so that the child has to make water oftener than usual, and sometimes even wets its clothes.

Although, as we have said, we can usually find no special cause for the trouble, still, in some cases, certain morbid changes in the urinary organs may give rise to the incontinence. We should, therefore, in every case at least think of the possibility of stone in the bladder, of congenital phimosis and adhesions of the prepuce to the glans penis, of ascarides, of inflammatory conditions, and foreign bodies in the vagina, and make a special examination into these points. We must also bear in mind polyuria caused by diabetes or renal disease, and finally, of course, in the diagnosis of a purely nervous nocturnal incontinence of urine, we must exclude the existence of any actual anatomical spinal affection.

In all the cases just mentioned, the treatment must be directed first to the primary disease; but in the ordinary nocturnal incontinence the treatment must be aimed at the symptom of nocturnal micturition as far as possible. The child must take only a very little fluid in the evening, and he should be made to empty his bladder immediately before going to sleep, and perhaps once again later. He should not be covered up too warmly, and, if possible, he should not lie on his back during sleep. Tying a brush to the back is therefore a well-known domestic remedy. It is strongly recommended that the child should be made to lie in bed, with the hips elevated and the head low. A somewhat strict mental treatment is often effective, since thus the attention to the process is increased, although unconsciously, and the child often learns to wake up at the right time. We have seen many cases of enuresis, which did not recover when treated at home, get well quite rapidly when the children were isolated in the hospital. The employment of the rod is rarely allowable; on the other hand, we often must shield the child from injudicious parents.

Internal remedies, such as belladonna and tincture of nux vomica, were formerly recommended, but they seldom do good. Lately the tincture of rhus aromatica has been employed again; of this, fifteen drops are given in the afternoon and in the evening before going to bed. In anæmic children preparations of iron are indicated. It is often useful to employ electricity, although it may be that the only influence it has is psychical. We put the broad anode over the lumbar cord, and the smaller kathode over the region of the bladder or on the perineum, and let quite a strong constant current pass through for two or three minutes. The current may also be interrupted and closed a few times. Then we pass the wire end of one conducting cord, which we make the kathode, into the mouth of the urethra for one or two centimetres, while we place the other broad electrode on the perineum or above the symphysis, and let quite a strong and somewhat painful faradic current act for one or two minues (Seeligmüller). The sittings must at first be repeated daily. It is also a very good plan to let the whole body be well rubbed with cold water before going to sleep. It is said that in obstinate cases the introduction of large bougies and the consequent distention of the posterior portion of the urethra are often attended with rapid improvement.

Even in cases in which, despite careful treatment, no permanent success is attained, we have this consolation with regard to the prognosis, that in simple enuresis the abnormal condition usually undergoes gradual and spontaneous improvement as the patient grows older.

[Belladonna, strychnia, or nux vomica, or a combination of the two, are often of unquestionable service. If the enuresis is only nocturnal, belladonna alone may be used, either in a single dose at bedtime or three times a day. If the enuresis is diurnal also, the two drugs should be combined and given three or four times a day.]

DISEASES OF THE ORGANS OF LOCOMOTION

CHAPTER I

ACUTE ARTICULAR RHEUMATISM

Ætiology.—Acute articular rheumatism is an infectious disease. This is shown by all its clinical and anatomical characteristics, and although we are not yet definitely acquainted with the specific organized poison which gives rise to it (*vide infra*), still, it is impossible to come to a correct understanding of the symptoms and the course of the disease, unless we assume that it is caused by an infectious germ.

Like many other infectious diseases, acute articular rheumatism is often indisputably endemic and epidemic. According to Hirsch, the disease is most prevalent in the temperate zones, being much rarer in cold and tropical latitudes; but even in Europe it is by no means uniform in its frequency, and certain districts of England, Belgium, and Russia are said to be almost exempt from it. It is also possible to observe epidemic influences with regard to the frequency of its appearance, as already intimated. In Leipsic, where articular rheumatism is one of the most frequent of acute diseases, we observed for years that at certain times there were only a few cases, while at others there was a striking increase in their number. Usually attacks are most prevalent in the winter and spring months, but again it is sometimes in summer that the disease is especially common.

Among the exciting causes of the disease, taking cold is always mentioned as of first importance; and in fact it can not be denied that the influence of cold does often seem to contribute to the occurrence of the disease. This result, however, seldom follows a single severe exposure, but it much more frequently follows long-continued "rheumatogenous influences," as we may call them, and in particular the long-continued influence of wet and cold, certain occupations—for example, washing and scrubbing—inhabiting unhealthy damp dwellings, and the like. This explains why those who follow certain callings are especially subject to articular rheumatism; thus servant-girls and coachmen are frequently victims to the disease. And yet it is possible to regard all these injurious influences as being merely indirect causes, assuming that they favor the development or action of the specific micro-organisms; and, furthermore, it is by no means exceptional to see a case of articular rheumatism in which no history of exposure to cold can be obtained.

Sex exerts no special influence upon the frequency of the disease. As to **age**, acute articular rheumatism is most frequent in young adults between fifteen and thirty-five years old. In later life, and particularly in old age, it is much rarer. In children six years old or more the disease is not especially infrequent, but in younger children it occurs only exceptionally. We may be permitted to mention a single interesting case which we met with in Leipsic, where a child who died when only a few days old, and whose mother at the time of its birth was suffering from a severe attack of acute articular rheumatism, was found to have multiple purulent arthritis.

Of precisely what variety the special germ of acute rheumatism may be we do not yet know. Still, many recent investigations (Sahli and others) seem to prove that, at least in many cases, the chief rôle is played by the staphylococcus pyogenes. If this claim is confirmed it will deprive acute articular rheumatism of the most part of its specific character, from an ætiological point of view. In that case, we would have to regard it merely as a special clinical form of staphylococcus infection, and place it among the mild varieties of septic infection. Indeed, upon more careful consideration, this conception of articular rheumatism seems very probable. It is well known that in all varieties of constitutional sepsis the joints are particularly apt to be involved. It has also been shown by numerous observations that staphylococci are most frequently found to be the infectious germs in cases of acute benign endocarditis, consequently we should expect that a constitutional infection with the staphylococcus would, as a rule, display itself by fever and multiple serous arthritis. Very often there are at the same time other local disturbances. These are the so-called "complications" of articular rheumatism, which, however, would be more correctly termed special localizations of the infectious germs (*vide supra*, endocarditis, pericarditis, pleurisy, etc.). Not infrequently these localizations in the other serous membranes may appear previous to the swelling of the joints, or even without any articular swelling, and yet they may be, in an ætiological point of view, precisely analogous to articular rheumatism. If, however, we are not yet inclined to accept the designation of acute articular rheumatism as a mild septic infection, then it would seem advisable to speak of all the forms of disease which belong in this class and which are closely related to one another, as due to a "rheumatic infection," employing the word rheumatic with a special idea of ætiology.

If we conceive of articular rheumatism in the way just indicated as a clinically well-characterized constitutional infection—perhaps with staphylococci—the next question is the place of infection, and its gate of entrance. It seems probable that the place is not the same in every case. Very often it is impossible to determine the path by which infection entered; yet we may sometimes ascertain by careful questioning that the arthritis was preceded by a mild sore throat, or a laryngitis, or an intestinal disturbance. Indeed, we often find even small abrasions of the skin and similar lesions. It is probable that such statements of the patient and discoveries of the physician indicate the starting-point of the infection in the individual case.

Now that we have become clear as to the mode of development of articular rheumatism, we have gained also a satisfactory conception of the so-called secondary rheumatism which occurs subsequently to scarlet fever, meningitis, and other acute diseases. It is probable that in most instances these are due to secondary infection with the staphylococcus, although, of course, it can not be denied *a priori* that other varieties of germs may produce inflammation of the joints—e. g., multiple purulent arthritis is probably due in most cases to streptococci. Very frequently, however, the primary disease itself is occasioned by staphylococci. This is the case in many instances of chronic valvular disease. If a "secondary" acute articular rheumatism occurs in such diseases, as it often does, the process is to be regarded merely as a fresh acute infection, starting from the old focus.

A very noteworthy fact is that acute articular rheumatism can not be numbered among those infectious diseases which usually occur but once in the same individual. On the contrary, the disease has the same peculiarity as pneumonia and erysipelas, of appearing with noticeable frequency several times in the same individual, so that when a person has once had articular rheumatism, even though it has left no sequelæ behind it, he seems to be more liable than before to the disease. It is not exceptional to meet persons who have had five or more attacks of acute articular rheumatism.

Symptomatology.—The chief symptom of acute articular rheumatism is an acute febrile synovitis, which almost always affects several joints. The synovitis is associated with the usual local phenomena of swelling and tenderness in the parts affected. Often this articular affection is the first symptom, and, indeed, it may be the only symptom of the disease. It is, however, by no means exceptional for the arthritic trouble to be preceded by certain prodromal or initiatory symptoms, as is true of other infectious diseaſes. These prodromata consist either of a slight general malaise, or of certain local symptoms. It is not rare, as already mentioned, to have sore throat, or, as we have repeatedly had opportunity to observe, laryngitis. Still, the precursors are usually mild; and they may, as we have said, fail to appear at all, or at any rate they may easily escape notice. In some few cases the only symptoms at first are febrile; there may be no other marked constitutional symptoms, or there may be a typhoidal condition with enlarged spleen, and diarrhœa. Then, some days later, the characteristic swelling of the joints appears (Wagner). Taken in connection with the view of the nature of articular rheumatism which we have elaborated above, this behavior has nothing inexplicable about it—merely, the constitutional symptoms develop some little time before the local manifestations of the disease.

The articular disturbance is almost always very rapid in its development. Some of the larger joints are usually first affected, and perhaps those of the lower extremities somewhat oftener than those of the upper. It is extremely exceptional for all the joints that are affected to be attacked at one and the same time. It is somewhat characteristic of acute articular rheumatism that it "jumps from one oint to another." To-day this joint will be affected and to-morrow that, while the oint first attacked may still remain diseased or undergo rapid recovery. Thus, there may be either a few joints affected or many, in varying sequence, and they may sometimes be affected rapidly and at other times more slowly. In many of the milder cases the disease is an extremely temporary one, while in others it may attach itself most persistently to some one or more joints.

There is usually fever in addition to the arthritis; but it is not usually very high, seldom exceeding 103° (39.5° C.). The fever, on the whole, corresponds with the arthritic phenomena, and does not present an especially typical curve, but one which is irregularly remittent. We have scarcely ever seen the disease begin with an initial rigor; and the so-called "general febrile symptoms" of headache, stupor, and subjective feeling of heat are as a rule ill marked in acute rheumatism. This indicates that the constitutional infection does not, owing to the peculiar nature of rheumatic infection, attain great severity. The skin is noticeably inclined to perspiration, but the perspiration is not at all a result of any udden fall in temperature such as is seen in other diseases.

The course of the disease is marked by alternate ameliorations and aggravaions of the local symptoms and of the fever, and lasts, particularly if it is not reated, one or more weeks, or a still longer period. Often the entire course of he disease under such circumstances is made up of separate "attacks" (*vide nfra*), there being repeated improvement followed by articular swelling and fever. At last, however, the symptoms entirely disappear, and the patient enters upon a edious but complete convalescence.

In contrast with this simple course the clinical picture may be, in other cases, much more complex, for the peculiar infectious character of articular rheumaism renders possible many complications and peculiarities. The protean character of the disease will be evident upon a perusal of the following description of the symptoms referable to the different organs of the body.

Symptoms referable to the Different Organs, and Peculiarities in the Course of the Disease.—1. JOINTS AND SHEATHS OF THE TENDONS.—The favorable termination of most cases of acute articular rheumatism prevents us from often examining the anatomical changes in the affected joints; but there can be no doubt that in most instances the trouble is merely a simple serous synovitis—that is, an inflammation of the synovial membrane, with an exudation into the cavity of the joint, composed mainly of serum with but little admixture of fibrine and pus. The synovial membrane itself, in the cases which do come to autopsy, is usually very little affected. It is somewhat injected, opaque, and thickened. Necrosis of the cartilages is seen only in severe cases, or in those which have lasted a rather long time. From a clinical standpoint, the articular disturbance is noticeable chiefly for the pain which it causes the patient upon every movement of the joint and any pressure upon it. The tenderness is often in striking contrast with the slight objective signs of inflammation, for a joint which is extremely sensitive may appear to be scarcely at all diseased. Usually, however, the joints exhibit the signs of synovitis. The effusion into the joint produces an evident swelling, which can be seen particularly well in the knees, but also in the joints of the ankle, wrist, shoulder, and elbow, and sometimes even in the smaller joints of the fingers and toes, particularly the great toe. It is rather exceptional to detect swelling of the hip-joint. It should be remembered, however, that the swelling in the region of the joint, particularly the ankle or wrist, is often less the result of a synovial effusion than of an inflammatory periarticular œdema. This œdema, for example, may extend over almost the whole posterior surface of the hand. The joints are by no means invariably the only parts attacked. Not infrequently there are analogous inflammatory changes visible in the sheaths of the tendons, the bursæ, and perhaps, in many cases, even the fasciæ and muscles. The skin over the affected joints often has an inflammatory blush, which is usually pale-red and spotted, and can be best seen at the ankle, knee, and wrist. It has been maintained that the cutaneous sensibility is diminished over the joints affected, but we regard this as a mistake.

As might be expected, the number of joints attacked and the sequence in which they are attacked differ greatly in different cases; but almost invariably a number of joints suffer, so that any monarticular arthritis should not be regarded as rheumatic except after careful deliberation (*vide infra*, diagnosis). It should be said that, in mild cases, there may be only two or three joints affected, these being usually some of the larger joints of the extremities; and of these, one may be so much worse than the others that their participation in the trouble can be ascertained only by careful questioning and examination. In severe cases, on the other hand, the number of joints attacked is often very great. Such patients become extremely helpless, because any movement is possible, if at all, only under the penalty of very severe suffering. The patient usually lies with bended knees and feet curved so as to be concave on the plantar surface, and screams with pain at any attempt to change his position. The joints of the trunk sometimes participate in the disease, but hardly ever except in the severe cases; the articulations of the vertebræ, the sterno-clavicular joint, the articulation of the lower jaw, and the symphysis pubis, are then particularly apt to be affected.

The fugitive character of the arthritis has been spoken of as characteristic of acute articular rheumatism, and, indeed, it is not infrequently the case that comparatively large swellings of the joints soon abate and yield to new disturbances in other joints; but, on the other hand, the disease may persist very obstinately in a single joint. In this case one joint or rarely several joints are attacked with marked severity, either from the start or subsequently to milder affections of other joints, and often remain for weeks swollen or painful, long after all other symptoms have vanished.

2. CARDIAC SYMPTOMS.—The condition of the heart in acute articular rheumatism is next in importance to that of the joints. The physician should therefore, in every case, even the mildest, maintain a continuous watch over this organ. In 1836 Bouillaud made careful auscultatory investigations in this disease, and was thus the first to discover that the course of acute articular rheumatism is, with noticeable frequency,.accompanied by endocarditis, and sometimes even by pericarditis. Complications of this sort may occur in any case, whether mild or severe, or may be absent in any case, even the worst. They may develop at the beginning or later on in the course of the disease. Their development is often unattended by any subjective symptoms, so that they can be recognized only by careful physical examination. In many cases, however, the onset of cardiac disease is marked by a fresh exacerbation of the fever, or possibly by palpitation, or by painful sensations in the præcordia, or by dyspnœa.

We will consider first rheumatic endocarditis. This is almost always the benign verrucous variety (see page 306). It is far more prone to attack the mitral than the aortic valve, and is accordingly usually betrayed by a blowing systolic murmur at the heart's apex. Uncertainty may be cast upon the diagnosis by the fact that functional murmurs are not very infrequent at the apex or at the base of the heart in cases of acute articular rheumatism. We once observed a case of "hyperpyretic rheumatism" (*vide infra*) in which there was an evident murmur of this sort during life, and yet at the autopsy we were able to assure ourselves of the normal condition of the cardiac valves. Even an expert may for a time be in doubt as to the significance of many cardiac murmurs, and this explains in part the conflicting statements as to the frequency of cardiac complications in acute rheumatism. In general, one may say that such complications occur in twenty-five to thirty-three per cent. of the cases. The ultimate results of this endocarditis we do not need to describe over again in this connection (see the chapters on acute and chronic endocarditis). Complete recovery is possible. Often, however, the lesion gives rise to a chronic endocarditis—that is, to a cardiac valvular disease, which lasts through life.

The close connection between endocarditis and the arthritis must formerly have seemed very puzzling, despite the many hypotheses made to explain it. Our present conception of articular rheumatism as a septic (staphylococcus) infection renders the frequent development of endocarditis easily explicable. It is due to a settlement of the germs, which are circulating in the blood, upon the cardiac valves (see page 304). As a matter of fact, staphylococci are almost invariably found in the inflammatory deposits of "rheumatic" endocarditis. The endocarditis, therefore, is not strictly a "complication," but a symptom of the disease.

Rheumatic pericarditis is not infrequent, although less common than endocarditis. The only certain way to recognize it is by a characteristic friction-sound; and even when this is heard there may be a doubt as to its significance, inasmuch as functional murmurs are not infrequently heard at the base of the heart. The pericarditis is of a sero-fibrinous nature. Sometimes it is of slight severity; but it may be extremely severe, with a large effusion and the most urgent dyspnœa (see page 359). In rare instances this pericarditis proves fatal. Usually, however, recovery ensues, although in severe cases there may be obliteration of the pericardial sac, with the consequences described on page 364.

As to the way in which pericarditis develops, it is probable that in most cases there is a direct infection of the pericardium from the blood. In some cases, however, we are inclined to assume that the infection of the pericardium comes from the endocardium, and probably in most instances from the aortic valves (see page 359). We have repeatedly found a distinct aortic insufficiency to have been left behind after rheumatic pericarditis, the heart having previously been healthy.

We should also mention that there may be functional cardiac derangement without any grave anatomical lesion. We have already spoken of the functional murmurs. There may also be a rapid and irregular pulse, and, in rare instances, attacks of angina pectoris of apparently purely nervous origin.

3. Serous and Mucous Membranes.—We have not only pericarditis in association with articular rheumatism, but also pleurisy and peritonitis, so that we may say that in acute articular rheumatism the incidence of the disease is mainly upon all serous membranes, inclusive of the joints. It should be said that rheumatic pleurisy, and particularly rheumatic peritonitis, are much less frequent than endocarditis and pericarditis, but we have repeatedly seen in severe cases rheumatic pleurisy, with serous effusion, on one or even on both sides. In such severe attacks it is not exceptional to find several serous cavities simultaneously involved—for instance, the pericardium and pleura; so that in such instances the inflammation may have extended directly from the pericardium to the pleura, or *vice versa*, although direct infection of the pleura by the blood is perfectly possible. Rheumatic peritonitis, indicated by distention of the abdomen with a moderate exudation and by tenderness, has been absolutely diagnosticated in a few cases. Here, also, the inflammation may appear independently, or may be due to extension from the pleura through the diaphragm.

The mucous membranes are seldom greatly affected in acute articular rheumatism. As we have already mentioned, in the beginning of the disease there may be catarrhal affections of the pharynx, larynx, stomach, or intestines; which lesions probably facilitate the "rheumatic" infection. Bronchitis is spoken of by the older authors as frequent, but it is probably in many cases not due directly to the rheumatism, but is a complication, just as in any disease attended by great prostration.

4. Skin.—Cutaneous phenomena are not infrequent in the course of acute articular rheumatism. A prominent symptom of the disease is the tendency to profuse perspiration. The perspiration often has a strongly acid odor and reaction. Many patients exhibit an abundant crop of sudamina, the back in particular being sometimes entirely covered. Sometimes there are other cutaneous eruptions. In a whole series of cases we observed erythema nodosum. This affected the lower extremities more than the upper. Urticaria is not very infrequent, while herpes labialis has been very rare in our experience. It is well known that arthritic affections and the so-called "hæmorrhagic diseases" are in many ways related to each other; and it is therefore an interesting fact that extensive hæmorrhagic disturbance of the skin also occurs in connection with acute articular rheumatism, as we have repeatedly had opportunity to observe. We have seen several cases of hæmorrhagic urticaria: wheals appear upon the skin, and a hæmorrhage takes place into their centers and spreads gradually. There may also be simple cutaneous ecchymoses. These may in severe cases be merely one symptom of a general hæmorrhagic diathesis, with hæmorrhages from mucous membranes. The occurrence of all these symptoms shows clearly that no sharp dividing line can well be drawn between the various disorders.

5. The Muscles and Nervous System.—The condition of the muscles is very important in many cases of acute polyarthritis. They are often quite painful on pressure, and apparently somewhat swollen about a joint that has been long affected. The muscular atrophies and muscular paralyses that often remain after the arthritis has disappeared are especially important.

According to a general law, there are certain definite trophic relations between a joint and the muscles belonging to it, whereby almost every severe and persistent disease of the joint is necessarily followed by an atrophy of the affected muscles.

The extensors of the joint are often most affected by this atrophy. This atrophy has long been recognized, and it was formerly regarded, particularly by surgeons, as merely the result of inactivity of the muscles—"atrophy from disuse"; but this view is certainly erroneous. We do not know its precise cause, but it is without doubt the result of the disease of the joint, and may therefore be termed "muscular atrophy of arthritic origin." If an attack of acute articular rheumatism affects any one joint for a long period there is a secondary atrophy of the corresponding muscles. This is seen most often, and in its most typical form, when there is obstinate trouble in the shoulder-joint, the deltoid becoming extremely atrophied. This atrophy of the muscles may contribute largely to the sum-total of functional derangement. We have repeatedly seen cases where the patient could hardly lift his arm at all, although the inflammation of the shoulder-joint had passed away, and that without leaving any anchylosis. It is therefore entirely justifiable to speak of a rheumatic paralysis. We have seen similar muscular paralyses after acute articular rheumatism in the rest of the muscles of the upper arm, also in the quadriceps extensor, and once in the serratus magnus. The explanation given by Charcot, that the cause of the atrophy is a "reflex" implication of the trophic centers in the cord due to the disturbance in the joint, seems to us hardly satisfactory. There are probably local disturbances of nutrition, and sometimes apparently a direct extension of the inflammatory process from the joint to the neighboring muscles. It is noteworthy that the atrophied muscles respond promptly to the faradic current, and never exhibit the reaction of degeneration.

Chorea (*vide infra*, page 1152) may be a sequel of acute articular rheumatism, and it is one of the nervous symptoms which may arise in connection with it. This complication is seen most frequently in children. Endocarditis may accompany it, but it does not always do so.

There are certain peculiar cases of acute articular rheumatism which excite the greatest interest. In these, very severe cerebral symptoms are developed, often most acutely. They are therefore called "cerebral rheumatism"; or, as they are almost always characterized by an extraordinarily high temperature, another name is "hyperpyretic articular rheumatism." In these cases the disease may exhibit severe nervous symptoms from the start, particularly delirium; or it may at first run an apparently favorable course, and not change for the worse until after several days, or even at a later period. The change may be quite abrupt. The temperature rises to 104° or 106° (40°–41° C.). There are great uneasiness, delirium, and sometimes also signs of motor irritation, such as general convulsions, or tonic spasm of the extremities, or trismus. The face grows pale and cyanotic, the pulse small and extremely rapid. With slight interruptions, the temperature continues to rise, and attains 107.5° to 109.5° (42°–43° C.). This great rise is most apt to occur just before death, and there may be a still further increase of temperature after death occurs. As has been implied, the termination is usually unfavorable. It is only in exceptional cases that recovery takes place.

It has been stated that cerebral rheumatism attacks mainly hard drinkers and other individuals whose nervous system has been previously impaired; but our own experience does not confirm this view. No case is absolutely secure from the occurrence of hyperpyrexia; but it is a very rare phenomenon, occurring perhaps once in several hundred cases. On post-mortem examination, the brain seldom shows any change in these cases of cerebral rheumatism. We are therefore obliged to regard the condition as the result of an unusually severe infection, affecting chiefly the intellectual, motor, and thermal centers.

Cases have also been reported which presented actual anatomical lesions of the brain—in particular, purulent meningitis. Probably, however, most of these cases were falsely diagnosticated, the observer having confounded articular rheumatism with epidemic meningitis, pyæmia, and similar diseases. Of course, if there be endocarditis, cerebral embolism is possible.

Mental derangements deserve a brief mention. They rarely occur during the course of the disease, but are somewhat more frequent after it has terminated. We may have either melancholia attended with marked excitement or anxiety, or a more general insanity. The prognosis is usually favorable.

6. Other Viscera.—Other parts of the body than the heart, the serous membranes, and the brain, are seldom much affected in articular rheumatism. Lobar pneumonia occurs only in especially severe cases, but in such it may attain quite a considerable development and occasion great dyspnœa. It usually requires quite a long while to recover from it. Whether we actually have to do in these cases with a specific pneumonia, or with an ordinary lobar pneumonia as a complication, has not yet been determined. It is also possible for lobular, deglutition pneumonia to develop in severe articular rheumatism. Acute nephritis has been demonstrated in some instances, but this is very rare. The spleen, as already mentioned, may be somewhat swollen; but as a rule we do not find the distinct splenic tumor which is present in other acute infectious diseases.

7. General Symptoms.—In many cases the general condition of the patient is but little affected, but in others the disease seems to exert a peculiar influence upon the constitution. This may show itself in a striking anæmia; and we have observed this repeatedly where there was no cardiac complication. Ordinarily, there is a slight leucocytosis. A dangerous but extremely rare complication has already been briefly referred to, namely, the occurrence of a general hæmorrhagic diathesis. This is almost always associated with high fever and great prostration, and is usually fatal. In all such cases there is probably profound sepsis.

Course, Duration, and Prognosis.—Acute articular rheumatism may be described as generally a benign disease, for it usually terminates in recovery. It is only in very few cases that an unfavorable termination takes place immediately, whether as the result of pericarditis or other severe cardiac complication, or from hyperpyrexia, or the development of a general hæmorrhagic diathesis.

The entire duration of the disease varies greatly according to its severity. There are mild cases, which terminate in a few days; and, on the other hand, the disease may be very tedious, lasting for weeks and months. This latter form is seen, upon careful consideration of the whole course of the disease, and particularly of the temperature-curve, to be made up of separate attacks, which can often be very clearly distinguished. In each attack of this sort (see Fig. 75) the temperature rises for some days and then slowly abates, so that the entire length of an attack is about seven to thirteen days. Then there is an afebrile interval of a few days, followed by a fresh rise of temperature, etc. The recurrence of the fever is usually associated with new trouble in one or more joints, or with some additional rheumatic disorder, such as endocarditis or pleurisy, while during the afebrile period the local symptoms abate, and the patient feels almost well. Friedländer has tried to deduce a series of general laws about the course of the disease, based upon this typical behavior of articular rheumatism, but in our opinion he has gone too far in this direction, although we have ourselves frequently observed this interesting course of acute articular rheumatism, with a series of separate attacks, and regard it as very characteristic of individual cases; still, there are many other forms which the disease may assume.

Quite often the violent symptoms experienced at first disappear promptly, but only to be replaced by such milder ones as pain and stiffness of the joints—these latter persisting for a long time. It is a general rule, that the severity and persistency of the case correspond with the number of joints affected; but to this that we can not be dealing with a simple acute articular rheumatism. Again, after childbirth there may be swelling of the joints, of septic origin, and but distantly related to genuine rheumatism.

If a single joint be attacked, the diagnosis of articular rheumatism must be made with extreme caution. These monarticular inflammations often prove to be something entirely different, namely, fungous disease of the joint, or an osteomyelitis. The arthritis which follows gonorrhœa is usually [though not invariably] monarticular (affecting especially the knee-joint or wrist). In conclusion, it should be stated that it is not very rare to observe pain and swelling in various muscles and joints at the commencement of the secondary stage of syphilis, simulating an acute articular rheumatism.

Sometimes the diagnosis is doubtful in those cases which present cutaneous ecchymoses (purpura and peliosis) and erythema nodosum, because we may be unable to determine which should be regarded as the primary symptoms and which the secondary. Until we become acquainted with the specific germs which cause all these diseases, it will be merely a dispute about words, whether we shall call a particular case by one name or another. In our opinion, all these conditions have a close ætiological relation to one another.

Genuine gout (*q. v.*) can usually be readily diagnosticated from articular rheumatism by its localization in the toe, and by the gastric and other symptoms which attend it.

Finally, we must mention that acute multiple neuritis (*q. v.*), beginning with fever and severe pain in the extremities, may sometimes, if we are not sufficiently careful, be mistaken for polyarthritis.

Treatment.—Acute articular rheumatism is one of the few diseases for which we possess an undoubtedly specific and universally accepted remedy. Kolbe suggested its use, and since 1876 it has been largely employed upon the recommendation of Stricker, Buss, and others, for articular rheumatism. This remedy is salicylic acid. Although this medicine does not in all cases produce its surprisingly favorable results with equal rapidity and completeness, yet it almost invariably does produce a decided and beneficial effect upon the disease. We might even say that this influence is so constant that when salicylic acid proves entirely inefficient in a fresh case, such failure throws doubt upon the correctness of the diagnosis. Thus, where there is monarticular arthritis dependent upon some local cause, the remedy has hardly any beneficial influence. The same is true with regard to affections of the joints connected with gonorrhœa, pyæmia, and similar troubles. In genuine acute articular rheumatism, on the other hand, the salicylic-acid treatment is so superior to any other that it is the first duty of the physician in every case to give this remedy a fair trial.

There are but two preparations of salicylic acid used in rheumatism—the pure acid and its sodium salt, salicylate of sodium. Each of these two remedies has its peculiar advantages, but the specific influence of each is about the same. Salicylic acid should never be prescribed in solution, but always in wafers or capsules, usually containing ten grains (gramme 0.50). In this way the salicylic acid can be taken by almost any patient quite easily, especially if a little water or milk be drunk after each dose. Adults should receive ten grains every hour until about one or two drachms (grammes 5–8) have been administered. Usually there will by this time be a very decided abatement of the articular pain and swelling, while on the other hand there will also usually be such toxic "salicylic symptoms" (*vide infra*) as to forbid its further use. The salicylate of sodium is best exhibited in single large doses of a drachm to a drachm and a half (grammes 4–6), each dose being given with about an ounce (grammes 20–30) of peppermint water, or still better, in wafers. The quite disagreeable taste of the medicine is only exaggerated by the addition of such substances as syrup or fluid extract of licorice, added for the sake of elegance; but the simple solution in peppermint water is quite well taken, at least by most patients. It is a very good plan to give salicylate of sodium in a glass of Hungarian wine, or in strong black coffee without sugar. The advantage of the salicylate over the pure acid consists in its being possible to give a larger dose at one time, so that it need not be taken more than two or three times a day. In general, the amount given in twenty-four hours should not exceed two and a half drachms (grammes 10); one and a half to two drachms (grammes 6–8) may suffice. For children the dose is, of course, smaller, about five grains of salicylic acid (gramme 0.30), or in twenty-four hours, half a drachm to a drachm (grammes 2–5) of the sodium salt.

Which of these two preparations shall be employed is, as has been stated, of little consequence. We ourselves usually prescribe, at first, capsules of salicylic acid to be taken hourly, as being most agreeable to the patient; but if our first visit be made in the evening, we prescribe a single large dose of a drachm to a drachm and a half (grammes 4–6) of salicylate of sodium, so that the patient may not be disturbed every hour through the night in order to take medicine. It is often possible to give the two remedies in alternation. This is a good way later on in the disease, when the patient has already acquired a distaste for the medicine. In such cases also it may be desirable to give the salicylate of sodium as an enema. About two and a half drachms (grammes 10) should be given, in two ounces of water (grammes 60). There is no doubt that the specific effects can be obtained in this manner.

The benign influence of this remedy upon the disease is apparent in many fresh cases as early as ten to eighteen hours after treatment begins; and it is often astonishing to see how soon a patient, who before lay helpless and complaining, becomes free from pain and able to move his limbs. It must be confessed, however, that, apart from its taste, salicylic acid may produce disagreeable incidental effects. In the first place, there may be nausea with epigastric distress, and even vomiting. Tinnitus aurium may be exceedingly troublesome, and may be attended with marked vertigo. In somewhat rarer instances the mind is especially affected. Young girls in particular are often peculiarly excited; but the frame of mind is, however, in general a cheerful one. After large doses there may be an actual "salicylic delirium." It should also be said that respiration may be affected, becoming very deep and rapid (salicylic dyspnœa). All these incidental effects, and particularly the nausea and ringing in the ears, render difficult the employment of the remedy in those large doses which alone are of any benefit. This is the more unfortunate, as it is often very desirable to employ salicylic acid persistently.

Although it is not exceptional to have the symptoms almost entirely vanish at the end of one or two days, yet it is only in the minority of cases that the entire process ends with this release from pain. There is very often, sooner or later, a relapse, with fresh pain or even fresh swelling in one or more joints. It has been recommended to continue the salicylic acid in smaller doses for some time, in order to avert such relapses; but of late we have rather abandoned this method, for the reason that these small doses do not prevent the return of the disorder, but are calculated to give the patient a strong dislike to the remedy, and lessen his confidence in it. We therefore recommend to stop the medicine entirely as soon as the pain is wholly gone, and to guard the patient as much as possible from relapses by preventing his catching cold (*vide infra*). If there be fresh pain, we should at once resume the acid or its salt in large doses, and thus we shall very frequently be able to cut short the relapse at once. In case there is persistent discomfort in the joints, small doses may be kept up for some time.

[One other salicylic compound deserves mention—the oil of wintergreen; this has been used largely by Kinnicutt, who finds it efficacious, easy to take, and not likely to produce the unpleasant symptoms which sometimes follow the compounds in more general use. It is given in doses of ten to fifteen minims every two hours, either in milk, on sugar, or in capsules.

The salicylic treatment markedly diminishes the pain and fever, shortens the time spent in bed by four or five days, does not shorten the time spent in hospital, and seems to have little or no influence on the cardiac complications. The full alkaline treatment does not curtail the pain and fever in the same degree, but does seem to afford some protection against the heart affection, and to shorten the stay in hospital several days.

A combination of the salicylic and alkaline treatments seems therefore rational, and it has been adopted by the editor for some years. Citrate of potash is given in sufficient doses to produce and maintain alkalinity of the urine. The salicylic compound is given at first in full doses, which are diminished in size and frequency with the control of the articular symptoms, but are not omitted entirely until recovery is practically complete. The editor is inclined to believe that relapse is less common if the system be kept somewhat under the influence of the drug. Salicine is often perfectly well borne when the sodium salt causes gastric irritability, and the oil of wintergreen is another form which has advantages in some cases.]

Despite the admirable qualities of salicylic acid, it must be confessed that we can not always bring about a rapid and complete cure of the disease by this remedy. There are cases in which, although at first evident improvement follows its use, relapses continually recur, or the disease fastens itself obstinately in single joints. In such, the continued use of salicylic acid proves almost unavailing, and indeed the patient can hardly be persuaded to take it. In these cases, and also when the salicylic preparations are ill borne or cause unpleasant attendant symptoms, antipyrine is the best substitute. Many physicians therefore often prescribe it from the first in place of salicylic acid, although we think the action of the latter upon acute articular rheumatism is greater than that of antipyrine. We should give antipyrine in doses of fifteen to thirty grains (grammes 1–2) several times a day in water or wine. Its attendant symptoms (sweating, nausea, and sometimes an eruption like measles) are usually without special significance, and its influence on the articular pains is often so favorable that the remedy is of much value in the treatment of protracted rheumatism. We have also often used salipyrine (salicylate of antipyrine) with good results, and likewise phenacetine, and salol (phenyl-salicylate), which last was recommended by Sahli, and which is administered in powders up to fifteen grains (gramme 1) several times a day, the total daily dose being seventy-five to ninety grains (grammes 5–6). Antifebrine or acetanilide is decidedly less efficient, but it has the advantage of being inexpensive.

From the other remedies which may be sometimes tried in obstinate cases, we can seldom expect satisfactory results; at least, we have ourselves never seen any decided benefit from iodide of potassium, preparations of colchicum, the alkalies, trimethylamine, or veratrine. Far more important in refractory cases is the local treatment of the diseased joints. This means chiefly massage, the results of which, when scientifically performed, are often admirable. Electricity also may have a beneficial effect, particularly the galvanic current. We would caution against the too early use of warm baths, as these often aggravate the pain instead of mitigating it. Steam baths are sometimes very beneficial, but they may also do harm, and they should therefore be given only when the acute inflammatory symptoms have entirely vanished, leaving behind only stiffness and tenderness in the joints. On the other hand, it is advantageous to give the patient a sweat in bed. This is a favorite remedy of the author's, and may be accomplished by introducing heated air under the bed-clothes, or in other ways.

The application of an ice-bag in genuine articular rheumatism is seldom necessary, but it may sometimes be desirable when there are violent and obstinate symptoms of acute inflammation. Warm, moist applications are seldom beneficial in acute cases. In the advanced stages of subacute cases poultices or a wet-pack may afford some relief. Painting the skin over the joints with tincture of iodine produces no effect in acute cases, and even in the chronic ones it is probably mainly a subjective remedy. Some observers report that injections of carbolic acid beneath the skin of the affected joints greatly relieve the pain. A Pravaz's syringeful (thirteen minims) of a one-per-cent. solution may be injected one to three times a day. We have had no personal experience with this remedy. In all severe cases careful attention should be given from the start to maintaining a correct position of the diseased joints, because of the possibility of anchylosis. Before salicylic acid was introduced, "the treatment of articular rheumatism with splints" was largely and very advantageously employed. The use of salicylic acid has greatly diminished the necessity of such procedure, but even now it is sometimes required. It is often possible to give the patient great relief by applying a suitable pasteboard or wooden splint to an affected extremity.

General hygienic and dietetic treatment should not be undervalued. An equable temperature should be carefully maintained in the sick-room, inasmuch as cold, or draughts, or moisture have very often been found to exert an evil influence upon the disease and excite fresh pain. The patient should therefore be kept warm, and it is sometimes advantageous to wrap up the affected joints in cotton batting. It is of great importance that even in the mildest cases the patient should be strictly confined to bed, and he should by no means get up too soon. If possible, we keep our own patients in bed for a week after the pain has ceased. Getting up too early will very often bring on a relapse. With regard to diet, milk is the best food. We may also allow soup, eggs, and a little meat. In France, great weight is laid upon an exclusive milk diet; but this would seem to us an extreme view.

We do not need to speak at length about the treatment of the complications and sequelæ, since we should merely repeat what has already been said in the appropriate chapters of this work. There has been much said on both sides as to the influence of salicylic acid in preventing complications, particularly cardiac complications. This much is certain, that cardiac complications are not absolutely prevented by the salicylic treatment, and that they too frequently occur while it is being employed; but we do believe that this treatment decidedly shortens the course of the disease as a whole, in many instances, and thus lessens the liability to endocarditis. If, however, a cardiac complication has made its appearance, salicylic acid does not exert any appreciable influence upon it.

Another important question is in regard to the efficiency of salicylic acid in the graver forms of articular rheumatism, particularly in cerebral rheumatism. It may be stated, in the first place, that in Leipsic cerebral rheumatism has apparently become much less frequent since the salicylic treatment was introduced. At any rate, not a single case of hyperpyrexia occurred in the clinique in that city out of many hundred cases treated, where the salicylic acid was properly employed from the first. We had an opportunity to observe a case in which hyperpyretic symptoms had already appeared when we first saw it, and which had not been treated with salicylic acid. Here large doses of that remedy produced no effect. We should nevertheless be inclined to employ it, first of all, in such cases; and the energetic use of cool baths would probably be the most speedy way of modifying the dangerously high temperature. Stimulants, in particular camphor, are also required in these severe cases.

In the severe hæmorrhagic varieties of rheumatism we should also give salicylic acid a trial. The milder hæmorrhagic cases (hæmorrhagic urticaria) do well under ordinary methods of treatment.

If the acute affection merge into a chronic condition of stiffness and swelling of certain joints, such as the wrist or shoulder, we must employ the same remedies as in chronic articular rheumatism. Passive movements and massage furnish the best results. Warm baths may also be ordered in such cases (see the following chapter). The patient might be sent to Teplitz or Wiesbaden. These baths are good resorts for convalescents. The subsequent muscular atrophies and paralyses recover most rapidly under electricity.

Prophylaxis requires, first of all, that one should avoid cold or wet, and other "rheumatogenous influences." Persons who have already had one attack of articular rheumatism must be especially careful, inasmuch as they are more than ever liable to the disease, as has already been said. It is not inconsistent, however, with the exercise of due caution, to endeavor to lessen the sensitiveness of the skin by such procedures as cold sponging, followed by friction.

CHAPTER II

CHRONIC ARTICULAR RHEUMATISM (CHRONIC POLYARTHRITIS) AND ARTHRITIS DEFORMANS

Ætiology.—The two diseases known as "chronic articular rheumatism" and "arthritis deformans" are considered together here, because it is impossible to draw a sharp distinction between them. It is, indeed, not unlikely that the above names are sometimes applied to diseases which differ essentially from each other; but as we do not yet understand the nature or the ætiology of many chronic diseases of the joints, we must provisionally be guided by the external changes they produce. We shall therefore embrace all chronic inflammatory processes affecting the joints under the name of chronic arthritis. It is not intended to include traumatic arthritis, much less those chronic affections of the joints which are evidently of tubercular origin, and which have ordinarily been termed fungous arthritis. These belong to the domain of surgery. We would also exclude chronic syphilitic diseases of the joints, about which, indeed, there is still less known than about the tubercular affections; besides, they are rare. Chronic articular affections from gonorrhœa are more frequent, and of great practical importance.

The ætiology of those cases of chronic arthritis where the disease is a direct sequel of acute articular rheumatism is evident enough. It is hardly possible to doubt that the same specific poison which excited the acute arthritis maintains possession of the joints, and produces the chronic inflammatory changes. Cases of this sort especially deserve the name of chronic articular rheumatism. They are not very infrequent, and may be of slight or great severity. The worst cases produce macroscopic changes which fully justify the other appellation of arthritis deformans.

It is also possible that many cases which are chronic from the start have the same ætiology—that is, are due to the same pathogenic agents. This might be inferred from analogy with numerous other diseases, and it is rendered still more probable by the fact that the same exciting causes which promote acute articular rheumatism often play a conspicuous part in chronic arthritis. Such causes are exposure to cold and wet, and working in cold or draughty places, or dwelling in newly built and damp houses. This explains why those who follow certain callings—for instance, washerwomen—are more apt to suffer from the disease than others, and why arthritis deformans has, with some justice, been called a disease of the poor, in contrast with the gout of the wealthy. Many of these sary, but it may sometimes be desirable when there are violent and obstinate symptoms of acute inflammation. Warm, moist applications are seldom beneficial in acute cases. In the advanced stages of subacute cases poultices or a wet-pack may afford some relief. Painting the skin over the joints with tincture of iodine produces no effect in acute cases, and even in the chronic ones it is probably mainly a subjective remedy. Some observers report that injections of carbolic acid beneath the skin of the affected joints greatly relieve the pain. A Pravaz's syringeful (thirteen minims) of a one-per-cent. solution may be injected one to three times a day. We have had no personal experience with this remedy. In all severe cases careful attention should be given from the start to maintaining a correct position of the diseased joints, because of the possibility of anchylosis. Before salicylic acid was introduced, "the treatment of articular rheumatism with splints" was largely and very advantageously employed. The use of salicylic acid has greatly diminished the necessity of such procedure, but even now it is sometimes required. It is often possible to give the patient great relief by applying a suitable pasteboard or wooden splint to an affected extremity.

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, and even some physicians, believe that gout and arthritis deformans are in way related, but this view is erroneous.

t is very questionable whether all cases of chronic multiple arthritis are refer- to the causes already enumerated. Such other influences, however, as are erned in its production are not at all understood. Various authorities have tained that arthritis deformans is the result of a primary disease of the nerv- enters, and in particular of the spinal cord. We regard this statement as ely unwarranted. It originated at a time when there was a tendency to be all sorts of ills to disease of the "trophic centers," but there is no doubt this tendency was carried very much too far. We may state in this connec- that a careful microscopic examination of the spinal cord in one case of very e arthritis deformans yielded an entirely negative result.

Some striking cases are reported by Blake, which go to show that the arthritic ges may be set up by chronic, though slight, suppuration, in analogy, perhaps, the synovitis of gonorrhœa. Careful examination should consequently be of all the mucous membranes, especially in cases which seem to have no ection with true rheumatism.]

hronic arthritis is mainly a disease of advanced years. Certain monarticular ties, about whose ætiology, it must be confessed, we know little as yet, have termed arthritis senilis—in particular the *malum coxæ senile.* Even the non and, in a certain sense, typical form of arthritis deformans (*vide infra*) t apt to occur in people under thirty-five years of age. This rule, however, xceptions, and we have ourselves seen a few perfectly characteristic cases of itis deformans in children between ten and fifteen years of age. Women are oftener attacked than men. It is often said that trouble, anxiety, and other ional influences favor the outbreak of the disease; but the proof of this is ng. The disease does not often seem to be hereditary.

athology.—The process is described as simple chronic arthritis (or synovitis) ng as it is confined mainly to the synovial membrane of the joint and the rticular connective tissue. These parts often undergo decided inflammatory ening (*synovitis hyperplastica*); the synovial membrane becomes cloudy; the amount of synovial fluid is more or less increased (*synovitis serosa*)— is, we have chronic dropsy of the joint. Sometimes different parts of the vial membrane are connected by adhesions, which considerably interfere with novements of the joint. There may even be complete anchylosis: for exam- n the shoulder or knee.

hronic synovitis may pass imperceptibly into arthritis deformans. In this, nly the capsules of the joint, but the articular cartilages and the articular mities of the bones, are so much affected as to produce the most striking mity. These changes almost always originate in the articular cartilages. iany cases the morbid process consists merely in a fibrillation, destruction vearing away of the articular cartilage, beginning usually on its outer edge; y, the bone is exposed and itself undergoes ulceration. All this while, the ular capsule may escape involvement, and there may be no serous effusion the joint (*arthritis sicca*). In other cases, however, there are soon added ferative changes affecting not only the cartilage itself, but also the synovial brane. The inner surface of the synovial membrane becomes tufted, and of these tufts may undergo secondary calcification, break off, and become bodies in the joint. The articular cartilages are also affected by inflamma- new growth and proliferation; these in part become disintegrated, and lead eper and deeper excavations of the cartilage. From the cartilage the pro- extends to the bones, and thus at last the articular extremities of the bones ne almost completely destroyed. Sometimes, also, the periosteum near the s undergoes ossific periostitis. On microscopic examination, we find fibrous disintegration of the matrix of the cartilage, and proliferation and subdivision of the cartilage-cells, at the same time that there is destruction of the newly formed cells by simple or fatty degeneration.

Of course the normal structure of the joint is at last completely destroyed by these various processes. The articular extremities of the bones waste away more and more, and take new relative positions, as the parts which impinge upon each other are worn away. Externally, the joint usually becomes larger and larger; and this is the more evident because the surrounding muscles undergo great atrophy. There is often no collection of synovial fluid in the joints during the entire illness, but sometimes the joints are swollen—e. g., the knee joint—and even to a considerable degree. There is a tendency for the affected joints to suffer more or less complete anchylosis and contracture (*vide infra*).

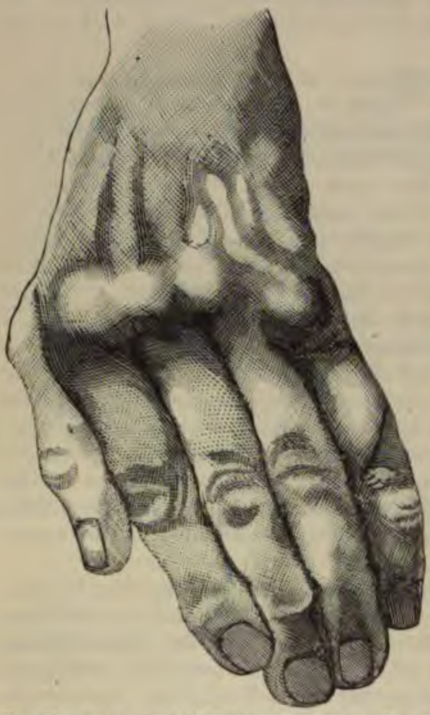

FIG. 76.—Appearance of the hand in a case of protracted arthritis deformans (personal observation).

Symptomatology.—The clinical symptoms of chronic arthritis are very simple and uniform. They relate almost exclusively to the local disturbances, and to these they are directly due. The general course of the disease, however, presents great diversities. Often the arthritis limits itself to a single joint or to a few joints; in other cases almost all the joints become finally involved.

Except in the cases which are preceded by acute articular rheumatism, the disease usually begins quite gradually and insidiously. The first subjective symptoms are stiffness and pain in the joints, the pain being aggravated by pressure or movement. The stiffness is most noticeable when the joint has remained quiet for some time previous, and is therefore ordinarily greatest on waking up in the morning. The pain often shoots from the joints upward and downward, and is of a burning character, or less often neuralgic. Even in advanced cases the pain usually occurs only when the affected joints are moved, although then it may be very severe. When the body is entirely at rest there is little or no pain. Besides actual pain there are apt to be at the onset of the disease peculiar paræsthesiæ and other nervous symptoms, particularly in the fingers. Motion is impaired very early. This is due at first to the pain, and to a certain reflex inhibition and ataxia of the muscles; to which are later added the purely mechanical hindrances and the ever-increasing atrophy of the muscles.

The objective changes in the affected joints begin to appear soon after the symptoms just mentioned, at least in cases of any severity. The joints often seem enlarged and thickened. If we attempt to move them, we not only cause pain and meet with mechanical obstruction, but we may hear and feel the cracking and grating produced by the rubbing of the denuded and uneven surfaces upon each other. This is often noticed by the patient himself. In chronic arthritis sicca there may be no great swelling of the joints; still, there are usually marked pain, crepitation, and impairment of motion.

apparent arrest of the process extending over months, or even longer. Sometimes the progress of the disease is marked by remissions and exacerbations, affecting either the general or the local manifestations. In general, however, the disease continually advances.

The **prognosis** is therefore unfavorable. Recovery, if it ever occurs, is extremely rare, and is possible only in the early stages. For the encouragement of the patient, it may be said that, under proper care and treatment, the disease often runs so gradual a course that the general condition remains at least bearable for a very long while, although there may be considerable local disturbance. The disease is not directly dangerous to life. The eventual fatal termination ensues either from general debility, or because of some intercurrent disease.

The prognosis is somewhat more favorable in the milder cases of "chronic articular rheumatism," where the anatomical changes are less severe, and are completely limited to the synovial membrane. Even here, however, recovery is by no means frequent, and it is always to be feared that grave deformities of the joints will gradually be developed.

Treatment.—With regard to regimen, it is requisite in the first place to avoid all unfavorable external influences. If possible, the dwelling should be dry and warm; and it may often seem advisable to make a change of climate. The patient must dress warmly, without, however, undermining his powers of resistance too much, as he will be in danger of doing. The diet must be abundant and nutritious.

Internal remedies may be tried, with the hope of modifying the disease, but our chief reliance must be upon local treatment of the joints. Among internal remedies, the most important are iodine and arsenic. Iodine may be given in the form of tincture (a few drops in mucilage several times a day), or a better form is in combination with potassium. As yet, we ourselves have not seen any great benefit from iodine, but we have in repeated instances witnessed a quite striking result from the use of arsenic. It is best administered in pills containing one thirtieth to one fifteenth of a grain (gramme 0.002–0.004) of arsenious acid, one pill two or three times a day. If this remedy prove beneficial, it must be continued for at least months, perhaps with occasional brief intermissions. The persistent use of small doses of salicylic acid or salicylate of soda, thirty to forty-five grains a day (grammes 2–3), and similar remedies, is recommended by many physicians. We have ourselves seen no special advantage from their employment, and therefore usually prescribe them only during acute exacerbations of the disease. The preparations of colchicum may be tried, but they will seldom be found efficient. Iron, quinine, and cod-liver oil are sometimes indicated by the general condition.

First among local methods of treatment comes massage, although the good it accomplishes is, of course, apt to be evanescent. It will, however, do much to hasten the absorption of inflammatory exudations, and also to loosen up the joints, invigorate the muscles, and improve the general health. The Swedish movement cure will be found of great benefit in all cases if begun early and methodically persevered in. It preserves the mobility of the joints as long as anything can. Electricity also has a beneficial influence. The galvanic current is applied to the affected joints, and the faradic current to the atrophied muscles.

Baths are universally employed in chronic arthritis. Their value should not be overestimated, but, notwithstanding, their usefulness is undeniable in many cases. Simple warm baths, or salt-baths (five to ten pounds of salt for each bath), are practicable in almost any household. As health-resorts in arthritis deformans, experience shows the following to be most desirable: The simple warm baths, such as Teplitz, Wildbad, Ragatz, and Baden in Switzerland; the warm chloride-of-sodium baths in Wiesbaden; the acidulated baths of Oeynhausen and Nauheim; and the mud-baths of Elster, Marienbad, Franzensbad, and Schmiedeberg. Steam-baths are admissible only in the early stages of the disease, and for pacians have reported precisely similar observations. Marie has proposed for them the name "*spondylose rhizomélique.*"

There is hardly any affection of parts of the body other than the joints i arthritis deformans. The muscles should be excepted, for they always underg that muscular atrophy which we have already described (*vide* page 680) as th result of joint disease. This atrophy is most marked in the interossei, the should

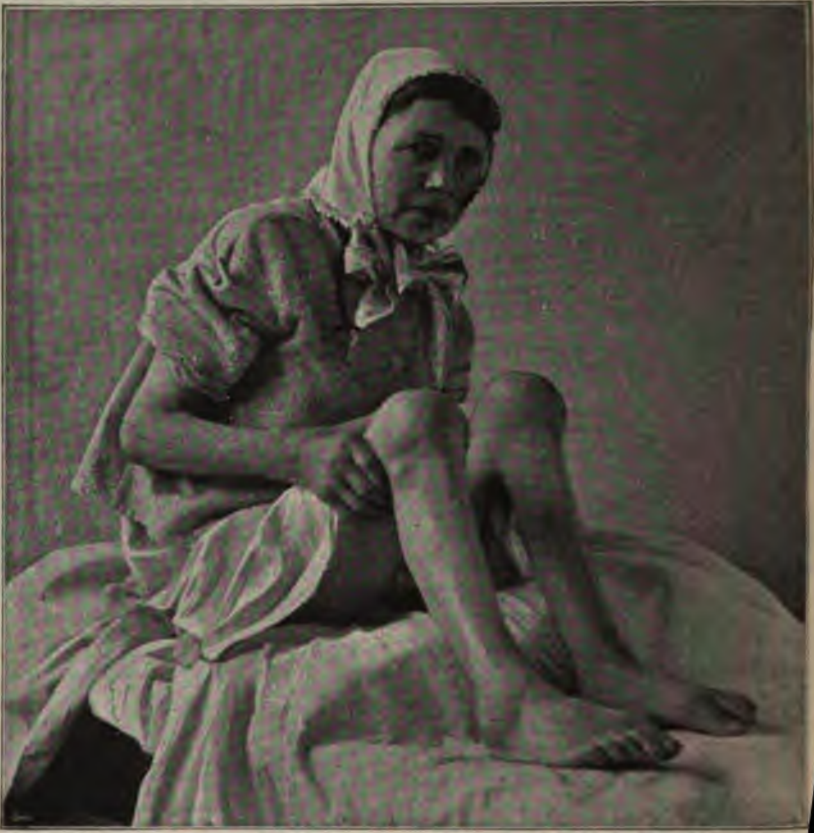

FIG. 77.—Chronic arthritis deformans with contracture of the hip- and knee-joints.

muscles, and the muscles of the calf and thigh. The associated muscular tractures (*vide supra*) are also of great importance. Sometimes the skin ove wrist and other affected joints appears peculiarly wrinkled and flabby. The nal organs almost always perform their functions in a perfectly normal m Appetite and digestion remain good, although there is often some tender constipation. Rarely there is valvular disease of the heart, but usually o such cases as originate in an acute articular rheumatism. Still, we hav valvular disease, exceptionally, in cases which had been chronic from the fact which is not without interest from an ætiological point of view. attendant symptoms which are sometimes observed, such as headache, co states, psychical depression, and bronchitis, probably are not directly co with the disease, but are easily explicable sequelæ.

General Course of the Disease.—Arthritis deformans is an extremely trouble. It may last even ten or twenty years, or more. Sometimes th apparent arrest of the process extending over months, or even longer. Sometimes the progress of the disease is marked by remissions and exacerbations, affecting either the general or the local manifestations. In general, however, the disease continually advances.

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The prognosis is somewhat more favorable in the milder cases of "chronic articular rheumatism," where the anatomical changes are less severe, and are completely limited to the synovial membrane. Even here, however, recovery is by no means frequent, and it is always to be feared that grave deformities of the joints will gradually be developed.

Treatment.—With regard to regimen, it is requisite in the first place to avoid all unfavorable external influences. If possible, the dwelling should be dry and warm; and it may often seem advisable to make a change of climate. The patient must dress warmly, without, however, undermining his powers of resistance too much, as he will be in danger of doing. The diet must be abundant and nutritious.

Internal remedies may be tried, with the hope of modifying the disease, but our chief reliance must be upon local treatment of the joints. Among internal remedies, the most important are iodine and arsenic. Iodine may be given in the form of tincture (a few drops in mucilage several times a day), or a better form is in combination with potassium. As yet, we ourselves have not seen any great benefit from iodine, but we have in repeated instances witnessed a quite striking result from the use of arsenic. It is best administered in pills containing one thirtieth to one fifteenth of a grain (gramme 0.002–0.004) of arsenious acid, one pill two or three times a day. If this remedy prove beneficial, it must be continued for at least months, perhaps with occasional brief intermissions. The persistent use of small doses of salicylic acid or salicylate of soda, thirty to forty-five grains a day (grammes 2–3), and similar remedies, is recommended by many physicians. We have ourselves seen no special advantage from their employment, and therefore usually prescribe them only during acute exacerbations of the disease. The preparations of colchicum may be tried, but they will seldom be found efficient. Iron, quinine, and cod-liver oil are sometimes indicated by the general condition.

First among local methods of treatment comes massage, although the good it accomplishes is, of course, apt to be evanescent. It will, however, do much to hasten the absorption of inflammatory exudations, and also to loosen up the joints, invigorate the muscles, and improve the general health. The Swedish movement cure will be found of great benefit in all cases if begun early and methodically persevered in. It preserves the mobility of the joints as long as anything can. Electricity also has a beneficial influence. The galvanic current is applied to the affected joints, and the faradic current to the atrophied muscles.

Baths are universally employed in chronic arthritis. Their value should not be overestimated, but, notwithstanding, their usefulness is undeniable in many cases. Simple warm baths, or salt-baths (five to ten pounds of salt for each bath), are practicable in almost any household. As health-resorts in arthritis deformans, experience shows the following to be most desirable: The simple warm baths, such as Teplitz, Wildbad, Ragatz, and Baden in Switzerland; the warm chloride-of-sodium baths in Wiesbaden; the acidulated baths of Oeynhausen and Nauheim; and the mud-baths of Elster, Marienbad, Franzensbad, and Schmiedeberg. Steam-baths are admissible only in the early stages of the disease, and for patients whose general condition is still vigorous. Even then they should be employed cautiously.

[The mineral springs within the limits of our own country chiefly to be recommended are Sharon and Richfield, in New York State, the Sulphur Springs and Hot Springs of Virginia, and the Hot Springs of Arkansas. At the two former, particularly, there is every provision for comfort as well as for the use of the waters.]

We have repeatedly seen quite excellent results follow the employment of hot sand-baths. These also can be easily used at home, particularly if applied merely to the hands or feet. They are employed more elaborately in Köstritz and Blasewitz. These hot sand-baths seem to do good, not only from the temperature, but also from the uniform and persistent compression which they exert. Sometimes, also, the long-continued local application of warmth, of hot compresses and packs, is very effective. Recently special apparatus has been constructed (first in England) for the local application of hot air. The diseased parts are thus exposed for about an hour, in closed copper chambers, to a heat of 150°–212° (65°–100° C.) and higher. The reports as to the success obtained in this way are very favorable [but, unfortunately, they are not fully confirmed by experience.—V.].

Stimulating or narcotic remedies may be rubbed into the joints, but they are beneficial only because of the massage which accompanies their employment. In practice it is not always possible to omit their use. The application of tincture of iodine is usually entirely without effect. As to morphine and other narcotics, the disease is so chronic that it is desirable to employ them as little as possible. A considerable number of those who suffer from chronic arthritis become opium-eaters. We should try, therefore, if there is severe pain, to give relief as long as possible with antipyrine, phenacetine, and similar remedies.

We may say, therefore, that the use of the various remedies which have been suggested one after another will enable us to oppose some obstacles to the progress of the disease. Persistent treatment will, in many cases, be rewarded by considerable, at least temporary, improvement.

CHAPTER III

ACUTE AND CHRONIC MUSCULAR RHEUMATISM

(Myositis, or Myalgia, Rheumatica)

Definition and Ætiology.—Certain acute affections may originate primarily in the muscles. These are to all appearance inflammatory in their nature, and not infrequently result from taking cold, or other causes similar to those which produce acute articular rheumatism. These affections are classed as "acute muscular rheumatism" or rheumatic myositis. It is possible that this disease also is an infectious one, but the question remains entirely undecided. The analogy which this trouble bears to acute articular rheumatism is not complete. It is seldom that the two processes are seen in combination; and, furthermore, acute myositis is not "polymuscular," but is usually confined to one muscle, or to a single group of muscles; it is attended with little if any fever; and it is never followed by acute endocarditis. The two diseases, therefore, are alike only in certain symptoms (pain and impairment of motion), and in the fact that they are often, although not always, ascribable to cold or the like.

There are many cases in which pain suddenly occurs in the muscles ("myalgia") without any attendant objective change. These cases can not be called genuine acute myositis. Indeed, it is sometimes difficult to know how to regard them. In practice they are usually termed muscular rheumatism, especially when they are referable to exposure; and it is possible that many such cases are really a very mild form of the genuine inflammatory disease. Yet in our opinion it is perfectly possible that some chemical change in the substance of the muscle occasions the local disturbance. That the influence of cold is often, though not always, important in this connection, seems to us indubitable. There are individuals who are almost certain to have a stiff neck if they are exposed to any decided draught upon the back of the neck; and others who, every time they get chilled, are liable to lumbago. However, other injurious influences may, of course, be important. Thus, traumatic pain in the muscles is the result of some excessive strain, and in many instances is apparently due to laceration of some of the muscular fibers. This is generally occasioned by too violent muscular exertion. Any physician who sees many patients from the laboring classes meets with an abundance of cases of this sort.*

The limitations of acute muscular rheumatism are obscure; but still more so are those of "chronic muscular rheumatism." This disease also is a frequent one, and only imperfectly understood. It does not bear a close analogy to chronic articular rheumatism, except in this unimportant point, that chronic muscular rheumatism seems to be quite often occasioned by meteorological influences. While the anatomical changes in chronic articular rheumatism are almost always striking, analogous lesions are very exceptional in chronic muscular rheumatism. On the contrary, the name is usually applied to cases where there is pain in various muscles all over the body, but where there is no discoverable objective disturbance. Older authorities used to speak of "rheumatic induration" of the muscles, but this or any other actual anatomical change is very exceptional.

These facts justify a doubt as to whether all cases of chronic muscular rheumatism actually deserve their name. It is certainly quite appropriate in those not infrequent cases which seem to be due to "rheumatogenous influences," and are so evidently aggravated upon every exposure to cold, or every period of bad weather, that the patient often asserts that he carries in his bones the best of barometers. Such is the "old rheumatism" of those who have passed a large part of their lives in the open air, regardless of wind or weather. There are other cases, the character of which is different. In them the muscular pain is associated with a general neurasthenic condition, or with corpulence (when it is, perhaps, the result of circulatory disturbance), or possibly with chronic poisoning. An important instance is the "rheumatic pain" sometimes complained of by topers, which we are inclined to ascribe not to changes in the muscles but to nutritive disturbances of the nerves. For these and similar disorders there are no special names, and the practicing physician often terms them all "muscular rheumatism," a diagnosis with which the patient is usually quite contented.

Clinical History.—Genuine acute muscular rheumatism is ordinarily, as has been said, limited to some one definite group of muscles. The affected muscles often seem somewhat swollen and infiltrated, are very sensitive to pressure, and, if not quite useless, are nearly so, greatly impairing the motion of the corresponding member of the body. All these symptoms are best illustrated in acute myositis of the deltoid (omalgia). The whole shoulder is swollen, the muscle is very painful, and the upper arm is almost incapable of voluntary motion, although, if caution be exercised, passive movement can be made without causing any pain.

The various forms of acute muscular rheumatism have received names descriptive of the locality of the affection. We have, besides the omalgia already mentioned—

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* Some time ago the author saw an organ player who had to work the pedals for many hours a day; he had an extremely painful affection of the lower extremities associated with swelling, which could be regarded only as an acute myositis.

1. Rheumatism of the cervical muscles (splenius, trapezius, sterno-cleido-mastoid, etc.), *Myalgia cervicalis, or rheumatic torticollis.* The pain is located in the back and side of the neck, and is usually unilateral. The head is held stiffly to one side, the occiput inclines toward the painful side, and the chin is turned toward the healthy side. All active and passive motions which tend to stretch the painful parts are much impaired, and cause suffering.

2. *Myalgia lumbalis, or lumbago.* This, the most acute form of muscular rheumatism, known among the common people in Germany because of its sudden onset as "witch's shot," or "dragon shot," is a well-known disease. The region of the sacrum and loins in a greater or less extent (erector spinæ, quadratus lumborum, etc.) is very sensitive. All motions of the trunk, such as bending and twisting, are very painful and difficult. The disease is more frequent in men than in women. Certain persons seem to be especially predisposed to it. It should also be stated that lumbago is not always of a rheumatic character, but of traumatic origin, as from lifting a heavy weight, or from sudden stooping.[*] [It is very common in the traumatic neuroses, where it was once regarded as indicative of spinal injury.—K.]

3. *Rheumatism of the thoracic muscles,* and particularly of the intercostals. This may cause great discomfort, as it renders breathing, coughing, and sneezing very painful. It is comparatively rare; and caution should be exercised in diagnosticating it, to avoid confusion with pleurisy and periostitis of the ribs. Very often, also, thoracic disturbance is regarded as rheumatic when it is really traumatic, being the result of stretching or laceration of the fibers of the pectoral or other muscles.

4. *Rheumatism of the head* also, probably, belongs in this category, although the affection is seldom confined to the muscles of the scalp, but involves also the fasciæ, and may even be almost confined to them. It is not infrequently excited by exposure to cold. The pain is quite violent, and greatly increased by any movement of the scalp. Of course, the diagnosis requires the previous exclusion of the various forms of headache described on pages 824 and 905.

The duration of acute muscular rheumatism is brief. Usually the pain abates in a few days; but a tendency to relapse persists for some time. Constitutional symptoms in addition to the local disorder are, as a rule, absent or slight; still, we sometimes meet cases in which acute muscular rheumatism is associated with fever and marked general disturbance.

In chronic muscular rheumatism there are, as a rule, no objective lesions discoverable. The statements of masseurs as to palpable nodes and indurations are, in our opinion, to be received with very great skepticism. The pain is seldom located permanently in any one place, but it is felt first here and then there. It is usually increased during bad weather, and is less severe when the weather is warm. The pains are often described as "wandering." Motion is seldom much impaired. Sometimes, however, there may be a certain stiffness of the muscles, most marked after a period of repose.

The diagnosis of chronic muscular rheumatism rests, therefore, mainly upon the rational signs. Hence it is often impossible to avoid the suspicion of malingering, particularly where certain applicants for hospital care are concerned. We should not, however, be too uncharitable, since without doubt there are cases in which quite severe pain is felt, now in one set of muscles and now in another, without any anatomical basis for such pain being discoverable. Nor should we ever forget that other diseases may have pain for their first symptom. It is not at all exceptional for the lancinating pains of locomotor ataxia to be for a long time regarded as "rheumatic." Lumbago may be confounded with insidiously developing tumors, diseases of the vertebræ, renal calculi, or with various hypogastric disorders (particularly in women). We should therefore never omit to make a careful physical examination.

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[*] Lately, S. Erben has sought to prove that lumbago and torticollis are not at all muscular diseases, but probably in most cases due to an affection of the joints of the vertebræ. He bases this belief upon the result of painstaking investigations. One who, like the author of this text-book, has often himself suffered from acute muscular rheumatism, will be scarcely inclined to adopt this view, for the subjective sensation of pain is referred decidedly to the muscles, or to the attachments and fasciæ of the muscles.

Treatment.—Acute muscular rheumatism has this in common with acute articular rheumatism, that it is usually very favorably affected by salicylic acid. In cases of genuine acute rheumatic myositis the employment of this remedy in the manner already described will often give surprising relief in as short a time as twelve or twenty-four hours. We also see good results from antipyrine or salipyrine, etc. Local treatment of the affected muscles also may be followed by great and speedy improvement. Massage is particularly valuable. It is not infrequently the case that a single properly conducted massage will cause a violent lumbago or omalgia to disappear almost completely, and like favorable results are witnessed when there is traumatic pain in the muscles. Most of the external applications which are so frequently prescribed for rheumatism—such as spirits of camphor or chloroform liniment—accomplish less through the cutaneous irritation they produce than by the massage incident to their employment. Next in value comes electricity. Both the constant and the faradic current may be employed. Often, also, the local application of warmth is very beneficial; thus hot dry cloths or hot poultices may be applied. Simple stimulation of the skin by mustard is often palliative, but less so than the first-mentioned remedies. Often great relief is given by sweating, by means of a hot wet-pack, or by a steam-bath, the employment of which is so common that patients often use it of their own accord.

In chronic muscular rheumatism the benefit of salicylic acid and antipyrine is merely temporary, and therefore is to be sought, if at all, only when there is an acute exacerbation. Massage and electricity are more effective, and, if persevered in for some time, will often accomplish good results even in obstinate cases. Treatment by sweating and bathing is frequently prescribed with advantage. Steam-baths are often beneficial, but their use requires great caution when the patient is corpulent and has a tendency to congestion or cardiac failure. There is also value in mud-baths, pine-needle-baths, and in the baths given at Teplitz, Wiesbaden, and other places.

In many case of chronic muscular rheumatism constitutional treatment is of great importance. Particularly when the patient is overfed, and intemperate in the use of alcohol, much benefit will often be accomplished by a proper regulation of the ingesta and the prescription of a sufficient amount of muscular exercise. Such patients may also be helped by a cautiously conducted cold-water treatment. This lessens their great sensitiveness to cold.

[In acute cases with localized pain I have found useful a thick flaxseed poultice, applied as hot as it can be borne, renewed once or twice, and followed by the application of a thick layer of cotton. A dry cup or two is also often productive of great relief. In chronic cases, plasters and the iodide of potassium are often of benefit.

Muscular rheumatism is a common and often very troublesome affection in those whose occupation calls for decided muscular exertion. A muscle is strained, pain settles in and is apt to recur in the part; and, while the general health is sufficiently good, the man is compelled to remain idle. Quack advertisements dwell so much upon pain in the back as a symptom of Bright's disease, that we are frequently consulted by those who, suffering from muscular pain and soreness, think themselves the subjects of serious disease of the kidneys.]

CHAPTER IV

ACUTE POLYMYOSITIS

FROM some recent observations (E. Wagner, Unverricht, and others) we have obtained knowledge of a disease which consists essentially in an acute inflammation of most, or even of all, the muscles of the body. The disease appears chiefly in persons in early or middle life, but it has also been repeatedly observed in the elderly. The disease may begin either quite suddenly or gradually. Sometimes polymyositis is a sequel of some other disease, most often, as it seems, of septic infections, such as puerperal fever and erysipelas. This circumstance seems to indicate that polymyositis is associated with severe constitutional intoxication.

The disease usually begins with decided constitutional disturbance, including languor, headache, anorexia, and vomiting. Very soon the characteristic muscular pains commence, there are dragging pains, and often cramps in the arms, legs, and trunk, and these sooner or later decidedly impede motion. If we examine the painful places we find a marked tenderness upon pressure and upon passive motion. Soon we can perceive a distinct swelling of the parts involved. This depends somewhat upon the inflammatory swelling of the muscle, and still more upon a firm inflammatory œdema of the corresponding skin, which usually appears very early. As a rule, the upper extremities are most severely attacked, and in them, too, the inflammatory œdema is most marked. The forearm swells, particularly in the region of the belly of the muscles, and so comes to have a characteristic spindle shape, provided the region of the wrist-joint is not involved. The face, the trunk, and the lower extremities also present similar though less marked swellings of the skin, or of the different soft parts, particularly on the extensor surfaces. At the same time, the skin is apt to be distinctly reddened with inflammation, so that it almost suggests erysipelas ("dermatomyositis"). Other exanthems also may appear—viz., erythema, urticaria, and herpes. If the disease lasts some time the swelling gradually disappears, and there may be distinct atrophy of the muscles. The patellar reflex is usually abolished from the start. The electric excitability of the muscles rapidly diminishes, and is finally destroyed. In some few muscles we sometimes observe the reaction of degeneration, but a careful electrical examination is always very difficult because of the great pain it causes. The sensibility of the skin in cases of polymyositis (*vide infra*) remains unimpaired.

The general condition becomes very much aggravated if the muscles of deglutition and respiration are attacked; the ingestion of food is rendered more and more difficult, and there is severe dyspnœa. Soon bronchitis and lobular pneumonia develop, and these are the more distressing to the patient because expectoration grows increasingly difficult and finally quite impossible. Nutrition is often interfered with by stomatitis. Tonsillitis has been repeatedly observed. Perspiration is decidedly increased, and the urine may contain a small amount of albumen. Splenic tumor has been repeatedly found in the acute cases. The bodily temperature is often elevated, 101°–103° (38.5°–39.5° C.); the pulse-rate may rise to 100 or 120 beats per minute.

With regard to the clinical course of the disease, we should make a distinction between the acute and the more chronic cases. The acute may end in recovery after two or three weeks. Often, however, after a shorter or longer course of from three weeks to three months, a fatal termination occurs, probably in most instances the result of respiratory disturbances, secondary pneumonia, and the like. Chronic cases may last a year or more.

In the cases observed thus far there has been found on examination a true acute inflammation of the muscles. Not only do the muscular fibers show all forms of degeneration and destruction, but also in the interstitial connective tissue we find true inflammatory foci (accumulation of nuclei about the vessels, etc.). In a case which we examined these changes were apparently to be found in all the muscles, even in the tongue, the ocular muscles, etc. In genuine polymyositis the peripheral nerves are perfectly normal. Yet recently there have been repeated observations of cases in which a genuine polyneuritis was demonstrated along with genuine polymyositic lesions (*neuro-myositis*). These cases indicate a close ætiological relation between polymyositis and polyneuritis (*q. v.*). At the bedside the appearance of polyneuritis as a complication may be recognized by slight disturbances in sensation, and by tenderness along the nerve-trunks.

The **diagnosis** of polymyositis is not difficult, on the whole. The pain in the muscles, their swelling, the impairment of motion, the inflammatory changes in the skin, etc., present a characteristic clinical picture. It is important to exclude a possible trichinosis. In this regard we must consider the ætiology, the initial gastro-intestinal symptoms, and the œdema of the face. [It is not yet certain whether eosinophilia is peculiar to the myositis occasioned by trichinæ or common to all forms of it.—V.] That there does not seem to be any sharp dividing line between polymyositis and polyneuritis has been already noted. We would like to point out, also, that a group of symptoms very similar to polymyositis is sometimes excited by a peculiar disease first described by Kussmaul and Maier, which is as yet very imperfectly understood. It attacks the small arteries and is termed "periarteritis nodosa." If the smaller arteries of the muscles are attacked, with resultant inflammation of the vascular wall, thrombosis, and similar changes, then degeneration of the muscular fibers ensues, with consequent swelling, pain, and impairment of function. At the same time there are usually severe constitutional symptoms, such as fever, anæmia, nephritis, and severe abdominal pain.

Treatment.—In early cases of acute polymyositis trial should be made of salicylic acid and its compounds, antipyrine, and similar remedies; beyond this, treatment must be purely symptomatic, including inunctions of chloroform oil, the administration of narcotics, and the like. Some cases may be benefited by galvanism.

CHAPTER V

RACHITIS

(*Rickets*)

Ætiology.—The first accurate description and the now universal name of "rachitis" (from ῥάχις, the spinal column) is to be ascribed to the Englishman Glisson, who published a comprehensive monograph upon this disease in 1650. It was his opinion that it first appeared in England, in the beginning of the seventeenth century; and for this reason rachitis is still often called by Germans "the English disease."

Although the clinical and anatomical phenomena of rachitis have been often and accurately investigated since Glisson's time, its true cause still remains entirely unknown. It is certain only that its development is promoted by all unfavorable external circumstances affecting the nourishment and health of the child. It is therefore more frequent among the poor than the wealthy, in the damp and crowded quarters of large cities than in the country, and among artificially fed, and therefore weakly and anæmic children, than such as receive the mother's milk. Nevertheless, the essential cause of the disease is not to be sought among these various influences, for rickets undoubtedly does occur, although rarely, in children whose circumstances seem in every respect most favorable.

Guérin, Friedleben, E. Voit, Wagner, Baginsky, and many others have made very exhaustive experimental researches with regard to the development of rachitis. It has been found possible to produce certain changes in the bones of growing animals by giving them as little lime as possible in their ingesta, or by administering very large amounts of lactic acid, with the purpose of dissolving the calcium salts, or by giving small quantities of phosphorus. The changes thus caused have been, with more or less correctness, regarded as analogous to those of rachitis. These investigations are of great interest with regard to the physiology of bony structures in general, but in our opinion they throw little light upon the clinical question which here concerns us. It is indeed natural enough to suppose that rachitis in childhood may be due to an insufficient proportion of lime in the food; or to a defective absorption of the lime-salts, on account of intestinal catarrh; or to an abnormally abundant production of lactic acid, or even of carbonic acid, which may dissolve the lime-salts in the system; but every one of these theories is contradicted by the facts of experience, for it is wholly improper to assume that the food of children with rachitis contains less lime than the food of healthy children, and the hypothesis of a mere poverty in lime of the bony tissues is by no means adequate to explain the whole complicated rachitic process. In our opinion everything seems to indicate that some special, specific, ætiological factor is requisite for the development of rachitis. This factor, however, is as yet entirely unknown to us. The thought had occurred to many that the disease bears some relation to congenital syphilis; but this assumption has long since been proven to be entirely without foundation. It is also claimed that heredity plays an important part in rachitis. The proof of this is lacking. It is, however, noteworthy that quite often several children of the same family are attacked by the disease.

Rickets is most common in children two or three years old. According to Kassowitz, the disease usually begins in the first months of life, while the severe symptoms, of course, appear only at the age of two or three years. Congenital rachitic changes (fœtal rachitis) have been repeatedly observed, but the cases of so-called *rachitis tarda*, where the disease is said to develop in children of eight or ten years of age and over, are at least extremely rare.

Sex exercises no great influence upon the occurrence of the disease.

Pathology.—Rachitis consists of a peculiar disturbance of the processes connected with the growth of the bones. As a result of an increased absorption of the already formed bony tissue, and especially as a result of an insufficient or an almost wholly deficient deposition of lime-salts, the bones become or remain abnormally flexible and soft, so that they can easily be cut with a knife.

Upon minute examination, we find both the periosteum and the marrow much reddened and congested. If we try to detach the thickened periosteum from the bone, not infrequently a few bits of bone adhere to the membrane. The most striking changes, however, are exposed upon making a longitudinal section of the bone. They are located at the bases of the epiphyses, because here is the place where the normal, and therefore the abnormal, processes of ossification are most active. Under normal circumstances, the epiphyseal cartilage of the bones in childhood is separated from the main shaft by two narrow layers: first, an outer one, nearest the epiphyseal cartilage, of a bluish color, and one or two millimetres thick; this is the proliferative layer, or hyperplastic zone, where the cartilage-cells become divided and arrange themselves in rows. Secondly, an inner, dull yellow layer, only about half a millimetre thick, known as the ossific layer, or zone of calcification, in which the real process of ossification takes place—that is, blood-vessels grow into it, osteoblasts develop, lime is deposited, and medullary spaces are hollowed out. In healthy bone these two layers are parallel to each other, and are limited by perfectly straight lines. In rachitic bone, on the other hand, they are both much enlarged, and their naturally sharp boundaries are replaced by an irregular serrated edge, so that the two zones encroach mutually upon each other. These changes affect both layers, but are most marked in the proliferative layer. Upon microscopical examination, the details of which can not be given here, we can see most plainly the complete confusion, if we may be permitted to use the expression, into which the growth of the bone has fallen. The proliferation of the cartilage-cells has increased beyond all bounds, and the scanty matrix of the cartilage displays a fibrous character. In the bony layer are seen irregularly scattered foci, which are already undergoing incomplete calcification, or else marrow formation, breaking down the cartilage. The latter is due to an invasion of the vessels, which always undergo an active new growth; they permeate the cartilage like lacunar spaces, and are surrounded by a so-called osteoid tissue.

The periosteum presents analogous changes. The innermost osteoblastic layer of the periosteum is thickened; but the newly formed tissue does not become completely calcified, but remains in large part soft and spongy. Finally an increased absorption of bone takes place inside the bones. The bony partitions disappear, and the cortical layer of bone often becomes much thinner than normal.

These various processes furnish a direct explanation of the macroscopic changes presented by rachitic bones. The proliferative process causes marked swelling of the epiphyses of the long bones, and thickening of the flat bones of the skull. The abnormal softness of the bones is due to the increased absorption of bone, their insufficient calcification, and it in turn causes various deformities, which are, for the most part, very characteristic (*vide infra*). If recovery takes place, the whole bone becomes firm at last, but often remains permanently deformed.

The deficient development of rachitic bones can also be recognized upon chemical examination. While normal bones in a dry state contain about sixty-three to sixty-five per cent. of lime, rachitic bones have only about twenty to thirty per cent.

Clinical History.—Rachitis often begins so insidiously that it can hardly be detected. Attention is not called to the disease until the deformity of the bones becomes very obvious, or it is noticed that the child does not learn to walk as early as other children, or, having already learned, is no longer able to do so. At last the anxiety of the parents is excited; and, on seeking medical advice, they find their fears only too well grounded.

In other cases the appearance of the characteristic changes in the bones is preceded by certain prodromes, either inflammatory symptoms, referable to the respiratory tract, or, what is especially frequent, signs of acute intestinal disease. The child suffers from diarrhœa, has some fever, looks pale, and grows weak and wretched. At night he is restless, screams, bores the back of his head into the pillow, and evinces a tendency to excessive perspiration. Often there is a slight enlargement of the spleen even at this period (Oppenheim).

The diagnosis of rachitis can not be definitely established until the characteristic changes in the bones have been developed. These anomalies vary, of course, in their severity and extent in different cases. We append a list of the most important:

The head is often noticeable for its great size and somewhat square shape, due to the thickening of the parietal and frontal eminences. In striking contrast with this the occiput is often thin and soft, so that it can be pressed in like parchment (Elsässer's *craniotabes*). This is particularly noticeable in places near the lambdoidal suture. At the same time the occiput usually becomes bald, because of its pressure against the pillow. The fontanelles remain open till the second or third year of life, and their edges seem soft and yielding. There is often a peculiar change in the shape of the jaws, particularly of the lower jaw. This is not rounded, but angular, being sharply bent in the neighborhood of the canine teeth; so that the incisors stand in a perfectly straight line, besides being somewhat inclined inward. Fleischmann was the first to describe this condition, and referred it to the action of the mylohyoid and masseter muscles upon the soft bone. Dentition in rachitic children is usually tardy, irregular, and tedious. Moreover, the teeth are soft and easily worn away.

The thorax presents, even in the mildest cases, very characteristic and noticeable changes. There is a swelling at the junction of the cartilages with the ribs, which can be felt and seen through the skin, and produces what is called the "rosary of rickets." In severe cases the lateral portions of the thorax are often drawn inward, particularly at the parts which correspond with the insertion of the diaphragm. This change is due mainly to the action of the diaphragm during inspiration upon the abnormally soft and therefore yielding ribs. The changes are greatest when the respiratory efforts, and particularly abdominal respiration, are exaggerated because of bronchitis, pneumonia, or some other disease of the air-passages. In such cases the entrance of air into the lungs is impeded, so that it is possible that the external atmospheric pressure also contributes to produce the deformity of the thorax. Deep hollows may finally be developed on each side of the chest, while the sternum becomes unusually prominent, giving the whole chest that shape which has been termed pigeon-breast, or *pectus carinatum*. When once this deformity has been developed, of course it in turn contributes to render respiration difficult.

The clavicles are sometimes distorted, and may even be partially fractured (*vide infra*). The spinal column is usually unaffected if the child remains quiet in bed; but if it sits up, or is carried about, or tries to walk, the traction and pressure thus exerted often produce curvature of the spinal column (rachitic scoliosis and kyphosis). These deformities may become extreme. Changes in the bones of the pelvis are of no special clinical importance at this period of the patient's life; but in women the consequent shortening of the antero-posterior diameter of the pelvis may, as is well known, prove a great obstacle to childbirth.

The extremities not only present swelling of the epiphyses, but are liable to curvature. This latter change is most marked in the lower limbs, inasmuch as they have to support the weight of the body. The swelling is especially well developed at the lower ends of the bones of the forearm and of the tibia and fibula. The curvature is almost invariably greatest, and therefore most easily recognized, in the tibia, which becomes convex outward, giving the rachitic child its "bow-legs." Similar curvature of the femur is less often seen, although it may be obvious enough in severe cases. The same is true of the humerus. The deformity of the lower limbs causes that waddling gait which can be so often seen on the streets of any large city. Sometimes the limbs present a sharp bend, the result of partial fracture. These "green-stick fractures of rachitis" are invariably referable to some slight traumatism, and are most often seen in the lower third of the tibia, although sometimes visible in the clavicles, ribs, and bones of the lower arm. The infraction usually takes place upon one—generally the concave—side, so that it is often compared to the partial fracture of a quill or an osier rod.

Symptoms in Other Parts of the Body.—Apart from the changes in the bones, a rachitic child may seem to be perfectly well. Even the general nutrition may be unimpaired. As a rule, however, rickets is associated with anæmia and impaired nutrition. The muscles, in particular, are small and flabby. The child seems pale, thin, and feeble, and may present swollen lymph-glands and other "scrofulous" symptoms. Sometimes there is a tendency to profuse perspiration, especially from the scalp. Very frequently there is chronic intestinal catarrh, and sometimes there is chronic bronchitis or lobular pneumonia. The abdomen is usually prominent, the liver and spleen are frequently, but not invariably, enlarged. The reader should also be reminded that rachitic children are particularly liable to spasm of the glottis (laryngismus stridulus) and convulsions, due perhaps to the rachitic lesions of the skull.

The fæces and urine have been repeatedly subjected to careful chemical investigation in the hope of gaining some information as to the pathogenesis of the disease. The results have been, however, in many respects, contradictory; the most accurate of these investigations have shown that the absorption and excretion of the calcium salts ingested with the food are not much different in healthy and in rachitic children.

The disease almost invariably runs a chronic course. Usually months, or even years, pass before the process ends. Its termination is to be recognized by closure of the fontanelles, increase in the length of the bones, and, above all, by the fact that the patient becomes stronger and makes attempts to walk. Unfortunately, many results of the disease persist through life. The legs are crooked, the thorax deformed, the spinal column curved, and the pelvis narrowed. Even in the most favorable cases persons who have once had rachitis usually remain somewhat smaller than those who are perfectly healthy.

Some authorities describe an "acute rachitis," in which painful swelling of the epiphyses is said to be developed in the course of a few weeks. At the same time the child becomes emaciated, and it may also suffer from diarrhœa or ulcerative stomatitis. Recovery takes place in a few months. How far cases of this sort are related to genuine rickets has not yet been determined.

Rachitis does not involve direct danger to life, although many rachitic children fall victims to the attendant intestinal catarrh, or to such complications as catarrhal pneumonia or tuberculosis. The prognosis is, therefore, not unfavorable when the worldly circumstances of the child permit of good care and nourishment. The remote influences of the thoracic, spinal, and pelvic deformities can be readily inferred.

The diagnosis of rachitis is but seldom difficult if the characteristic changes in the bones exist. In case cranial changes have occurred, we should guard against confounding rickets with hydrocephalus, but we can usually avoid error. The rachitic child holds its head erect, and is free from mental or other functional nervous disturbances.

Treatment.—The most experienced specialists agree that the first aim in treating most cases of rachitis is to improve the general nutrition. It is often possible to bring about recovery simply by means of proper diet (milk, the yolk of eggs, and perhaps meat), good air (in the country), and baths (brine, malt, and medicated baths). Digestive disturbances should be corrected by such remedies as hydrochloric acid or tincture of rhubarb; and iron should be administered if the patient be anæmic.

It is very important that the child should be placed upon a good mattress, and should neither attempt to walk too early, nor be needlessly taken up and carried about. The best way to avoid the development of deformities in the bones is to avoid all such unfavorable mechanical influences.

Attempts have also been made to check the disease by specific remedies. Upon doubtful theoretical grounds, lime has been very frequently prescribed, in the form of phosphate of calcium, of which fifteen to forty-five grains may be given in powder several times a day; or in the form of lime-water, of which one or two teaspoonfuls are added to the milk which the child drinks. The benefit of these remedies is seldom very obvious. Kassowitz has given a fresh impetus to the employment of phosphorus. To support his belief he brings forward numerous clinical observations, as well as facts obtained from experiment. We may either dissolve the phosphorus in cod-liver oil (0.01–100), giving one or two small teaspoonfuls of this solution every day, or we may write for the following mixture, which is more elegant, but is also more apt to spoil:

℞ Phosphori	0.01;
Olei amygdalæ expressi	10.0.
Misce, deinde adde:	
Pulv. acaciæ,	
Syrupi simplicis	āā 5.0;
Aquæ destillatæ	80.0.
M. Sig.: Two to four small teaspoonfuls a day.	

We can state from our own experience that the remedy is usually very well borne, and, in fact, often shows its beneficial action after a few weeks, the fontanelles growing smaller and the bones becoming firm. Yet there are cases in which phosphorus does not seem to do any good.

It may be eventually necessary to resort to orthopædic or surgical treatment in order to correct the deformities of the bones.

[The comparative rarity of rickets, especially in its extreme degrees, in this country strikes all observers who have studied in Germany. With a fairly extensive experience among the poorer classes of the city, the writer can recall scarcely half a dozen cases of craniotabes. The colored race furnishes a large contingent of cases of rickets, although, as is shown by Haven, in attention to diet and fresh air its members are superior to the Irish laboring classes, as a rule. The more pure the negro blood, the greater does the liability to rickets seem to be in this latitude—an indication, perhaps, that a northern climate is unsuitable to the African race.]

CHAPTER VI

OSTEOMALACIA

Ætiology and Pathology.—Osteomalacia does not, like rachitis, consist in a disturbance of development. The growing bones are not prevented from ossifying, but, having already undergone normal development and acquired normal firmness, they afterward become soft. It is mainly a disease of adults, occurring between thirty and forty years * of age, but sometimes earlier. The female sex is noticeably predisposed to the disease, although occasionally it has been observed in men.

The true cause of osteomalacia has not yet been ascertained. It is a remarkable fact that the disease is much more frequent in certain regions than in others. It is very common along the Rhine, and in Westphalia, in eastern Flanders, and northern Italy. This suggests that there is some specific cause for the disease, endemic in certain localities. Among exciting causes, child-bearing is certainly the most important, for both the first signs of osteomalacia, and also fresh exacerbations of the disease, usually date from a pregnancy. There is no doubt, however, that occasionally a case of osteomalacia occurs in a woman who has never had children. In such instances the appearance of menstruation seems invariably to have an unfavorable influence upon the disease. In brief, the relations of osteomalacia to the sexual processes are so close that there is some justification for the supposition that osteomalacia is directly dependent for its development upon disturbances of metabolism in the ovaries. Moreover, certain therapeutic experiences (*vide infra*) can well be reconciled with this assumption. It should also be mentioned that unfavorable hygienic surroundings, damp houses, and the like, promote the development of the disease.

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* Rehn maintains that genuine osteomalacia may occur in children; but his statement has not yet been fully corroborated.

The anatomical process of osteomalacia consists of a disappearance of the earthy salts of the bone, which begins interiorly and spreads outward, and causes a corresponding softening of the bony structure. The marrow is at first extremely hyperæmic; and extravasations of blood are not infrequently found here and there. The bony substance surrounding the myeloid spaces and the Haversian canals becomes transformed into a soft fibrous tissue, while the irregularly arranged bone-corpuscles either are destroyed or lose their characteristic shape. The softening process gradually extends from the spongy substance outward to the cortex. The central cavity grows larger and larger, so that finally the cortical substance is as thin as paper, and the whole bone like an "inflated and dried coil of intestine." At this stage the original hyperæmia of the marrow has vanished. The marrow acquires a yellow color, and may finally be entirely transformed into a yellow, viscid fluid. The affected bones are now flexible and soft, they can be easily cut, and they are of less specific gravity than normal. The periosteum is also at first thickened and hyperæmic, as if inflamed. When it is removed, the surface of the bone beneath it is found to be rough and uneven. The attendant alterations in the shape of the bones which can be seen during life will be mentioned below.

Upon chemical examination of the bones in osteomalacia, we naturally find a marked diminution in the proportion of lime-salts. It is also stated that lactic acid has been discovered in the bones. This is an interesting fact, as it may be that the acid plays an important chemical part in the process of decalcification.

Clinical History.—Osteomalacia begins very gradually in most cases. Usually the first thing noticed is an ill-defined, deep-seated pain, most often felt in the sacral region, the nape of the neck, and the back and thighs. The affected parts are also sensitive upon pressure.

The pain is persistent. While it still keeps on, motion becomes gradually impaired. The patient experiences more and more difficulty in walking, partly because of the pain and partly because of muscular weakness. The gait is either uncertain and slow, or characterized by short painful steps, the lower limb and the pelvis being jerked forward as if in one piece. This peculiar gait in which, with every short, hobbling step, the body makes a slight twist inward, is so characteristic that often the disease may be immediately recognized by this alone. In other instances the gait is more waddling. After a shorter or longer time walking becomes absolutely impossible, and the patient is permanently bedridden. Even then severe pain persists in most cases. It is not precisely spontaneous, but is produced by the mere pressure of the mattress or the bed-clothes, and similar causes.

In the meanwhile, there has probably been distortion of many of the bones, sufficient to occasion a decided alteration in the appearance of the skeleton. Usually the first to be noticed is the deformity of the spinal column (see Fig. 78). As a rule, there is kyphosis, less often some other deformity, and the head generally becomes more and more bent forward upon the sternum. As a result, the patient grows decidedly shorter, and this very shortening may be an important factor in diagnosis. The sick woman is apt to remark it, because she has to keep shortening her gown in front. The thorax, also, is apt to be much distorted in severe cases. It is compressed laterally, while the sternum becomes very prominent, and is sharply bent. The change in the shape of the pelvis in osteomalacia is less obvious externally, but it can be detected on internal examination. It is, of course, of great importance from an obstetrical point of view. The pelvis, like the chest, is compressed laterally, while the symphysis is made to project forward like a beak. The sacrum and its promontory are also pushed forward, and the superior strait thus acquires somewhat of a heart shape. This change in the pelvis is apt to appear very early. It is probably the chief cause of the peculiar gait (*vide supra*).

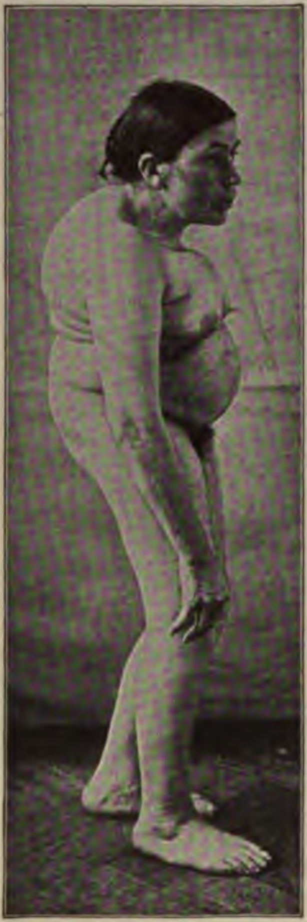

FIG. 78.—Puerperal osteomalacia. (Erlangen Medical Clinique.)

The extremities are less often distorted, particularly if the patient becomes bed-ridden at an early period. Manifold changes are, however, possible. Sometimes there is also fracture. In a few reported cases the softness of the bones of the extremities was so extreme that one could bend the limbs at will, like wax, and give them the most extraordinary positions. In cases so far advanced as these, the pain in the bones seems finally to cease. The bones of the head and face seldom undergo noticeable change, but we often find that the teeth are carious or lost. In the muscles, several observers have noticed trembling and fibrillary contractions, also paresis and complete paralysis. It is also said that sometimes even a slight irritation of the skin suffices to excite painful contractions of the underlying muscles. These phenomena have not yet been thoroughly investigated.

The general condition of the patient is often unimpaired for a long while, except for the pain and the impairment of motion. The internal organs perform their functions in a normal manner, and the appetite is good. Fever is observed only when the disease is undergoing some temporary exacerbation. With regard to changes in the urine, there have been a good many statements made, but their significance is extremely doubtful. It is said that the amount of phosphoric acid excreted is diminished. With regard to the amount of lime, no definite statement can be made. Lactic acid has been repeatedly detected in the urine, as has also albumen. Concretions of lime have been found in the bladder and the kidneys.

The disease runs a chronic course, occupying seldom less than two or three years, and sometimes even five to ten or even longer. Apparent improvement is not infrequently observed, but this is followed by fresh exacerbations, especially after confinement. The most frequent termination is in death. This results either from general debility, or, more often still, from the dyspnœa caused by the compression of the lungs, or by some such disease as lobular pneumonia. In milder cases, however, particularly if they receive prompt and suitable attention, there may be complete recovery; or in severe cases, even if deformity has occurred, there may be, at any rate, a permanent cessation of the disease.

Diagnosis.—In well-developed cases it is not difficult to recognize the disease, but at first a correct diagnosis is often impossible, unless the endemic frequency of osteomalacia suggests it. In the onset of the disease we may often be led to the false belief that there is incipient disease of the cord or the vertebræ. We ourselves, as well as other observers, have repeatedly seen cases in which women as a sequel to pregnancy, or even apparently spontaneously, have developed paresis in the lower extremities, particularly in the psoas and iliacus muscles, associated with pain and exaggerated tendon reflex, and in which diagnosis at first was very difficult. Not very infrequently the complaint of the patient at the start was regarded as hysterical. Later, the peculiar hobbling and waddling gait ("like a duck") would suggest the possibility of osteomalacia, and this diagnosis would be established by the results of a careful examination of the pelvis. At any rate, it is important to know that even before there are demonstrable changes in the bones, there may be paralysis, probably referable to an early involvement of the muscles in the morbid process. As the disease is almost entirely confined to adults, we are seldom in danger of confounding it with rachitis. Besides, osteomalacia does not produce swelling of the epiphyses, nor changes in the bones of the skull. It is said that osteomalacia is occasionally confounded with diffuse carcinosis of the bones, as this may produce similar symptoms and deformities.

Treatment.—As has been already implied, therapeutic efforts have thus far proved in severe cases almost unavailing in this disease. In the onset of the disease we can obtain a distinct improvement by the use of hygienic remedies, good air, proper food, etc. Internally we give cod-liver oil and also iron. Many patients praise warm baths, with or without salt. The exhibition of lime seems to be of no special use. On the other hand, personal experience leads us earnestly to recommend a trial of small doses of phosphorus, either in the form of phosphorized cod-liver oil (*vide supra*), or in pills of one sixtieth of a grain (gramme 0.001) several times a day. Particularly in the above-mentioned forms of incipient osteomalacia with paresis of the legs, and even also in cases with distinct changes in the pelvis already developed, we have seen, repeatedly, striking benefit from treatment with phosphorus. Yet sometimes even this remedy proves disappointing.

The changes in the bony pelvis produced by osteomalacia may eventually demand obstetrical interference, but we need not discuss such procedures here. We should invariably warn women who suffer from the disease of the dangers of becoming pregnant. Recently, therefore, castration has been repeatedly performed in cases in which an aggravation of the disease was invariably associated with fresh pregnancies. Under these circumstances, it has been shown that the removal of the ovaries has resulted in a remarkably rapid recovery from the disease, a fact which suggests an intimate connection between the processes in the ovaries and the symptoms of osteomalacia (*vide supra*). Certainly, these relations should receive the greatest consideration in the future.

DISEASES AFFECTING THE BLOOD AND TISSUE-METAMORPHOSIS

(CONSTITUTIONAL DISEASES)

CHAPTER I

ANÆMIA AND CHLOROSIS

1. General Preliminary Remarks as to the Classification and Ætiology of the Various Forms of Anæmia.—The word "anæmia" (or, more strictly, oligæmia) might properly be taken to signify diminution of the total volume of the blood, such as, for example, is directly brought about by a severe hæmorrhage. Usually, however, the word is employed to signify not so much diminution in quantity as deterioration in quality. The total volume of the blood is not liable to nearly so great variations as is the number of its most important constituents—the red corpuscles—inasmuch as the total volume is dependent merely upon the amount of watery constituents, and even after large hæmorrhages the water is quite rapidly replaced by absorption. This is undoubtedly the case in most instances of sudden loss of blood. Even in chronic anæmia there is usually no reason to assume that the total amount of blood is diminished, although it may be when there is general emaciation, or diminished supply of liquids (persistent vomiting, dysphagia), or large watery discharges (as in cholera). The essential element in anæmia is, therefore, a diminution in the number of the red blood-corpuscles, or so-called oligocythæmia. Changes in the character of the red blood-corpuscles are not taken into consideration here. On the other hand, however, we shall see later that, even in spite of a normal number of red blood-corpuscles, their abnormal condition, particularly their poverty in hæmoglobin, may lead to well-marked "anæmic" conditions.

The circumstances under which anæmia is observed are manifold. They admit, however, of our distinguishing two great classes of anæmia—primary and secondary. Primary anæmia is developed as an apparently primary and idiopathic disease in persons previously healthy, while secondary anæmia is merely a symptom of some already existing disease. However simple this theoretical distinction, yet in actual practice it is often quite difficult to determine whether the particular case before us should be regarded as primary or secondary. A secondary anæmia may occur where the true primary cause can not be at all readily determined. There are, nevertheless, quite a large number of cases which would seem to deserve the name of primary or essential, in which we feel compelled to assume that some pathogenic influence acts directly upon the blood and the hæmatopoietic processes.

In the first place, we would class as primary, cases which may best be described as "simple constitutional anæmia." These often stand close to the borderland between health and disease. There are not a few individuals who present a striking pallor for a large part, if not all, of their lives. These persons may feel so well and vigorous that we scarcely have a right to regard the existent anæmia (usually moderate) as an actual disease. Sometimes, however, such individuals do betray some diminution of energy, are easily fatigued, and are subject to headache. We may then regard the conditions as certainly pathological. In many instances the cause of this simple anæmia is found in the general hygienic surroundings of the patient, for such cases are most often met with among the poorer classes. Deficient nutrition, bad air, unhealthy occupation in factories or the like, not only affect the general health, but more especially interfere with the processes of normal blood-making. Other cases of constitutional anæmia, apparently primary, occur in individuals who are entirely beyond the reach of such influences as have just been mentioned, in whom the anæmia has developed and persists despite the best of food and air. Here we are forced to the conclusion that the organs engaged in the manufacture of the blood are in some way prevented from performing their proper functions. The trouble often seems to be congenital, for such individuals may present the symptoms of anæmia in their earliest infancy. There are persons who have always been pale and feeble. Or, again, anæmia does not develop until later on, in which case it not infrequently associates itself with certain phases of physiological development, as when growth is particularly rapid, or when adolescence occurs. Virchow has directed attention to another factor, which he regards as potent in many of these cases of congenital anæmia. He has found that the arteries may be congenitally small, or that the whole arterial system may be imperfectly developed. The condition may be associated with a congenitally weak and small heart. The importance of this factor has not yet been fully determined. Possibly the condition of the circulatory system just mentioned may be the result rather than the cause of the anæmia.

A second division of primary anæmia includes cases which present a far more definite and distinct group of symptoms. They quite often appear in persons previously healthy, last for a certain length of time, and then end in complete recovery. The typical form of this variety is chlorosis (χλωρὸς = greenish yellow), or "green sickness." This well-known disease is especially frequent in young girls fourteen to twenty years of age—that is, at puberty. It often comes on quite rapidly without any ascertainable cause. There are not infrequently predisposing influences in the outward circumstances of the patient. Thus it is promoted by an unhealthy sedentary mode of life, as in seamstresses; bad air, as in factory operatives; mental and physical over-exertion, as in teachers, governesses, and students; and, finally, by mental influences (excitement, etc.). It is, nevertheless, true that chlorosis also appears in girls who have lived under the most favorable hygienic conditions possible. Sometimes the disease seems to be merely a temporary exacerbation of a simple constitutional anæmia which has existed a long time; but it may also appear in young women who have been previously healthy, and even robust.

The true cause of chlorosis is unknown. Perhaps the interesting investigations of Bunge with regard to the amount of iron in milk and in new-born animals afford at least a starting-point for some future theory as to chlorosis. Bunge found that the constituents of the ash of milk, and the constituents of the total ashes of young suckling animals, correspond precisely in their proportions. The suckling animal, therefore, receives with his mother's milk all the salts that are needed for his body, in a proper amount—the single exception being the iron compounds. The amount of iron in milk is very small compared with the amount of iron in the suckling. Indeed, the proportion of iron in the new-born animal is the internal processes of metabolism which relate to the matter. S facts and conditions which we have mentioned explain to a certain de fact that genuine chlorosis is found exclusively in the female sex and, precisely at the time of puberty—i. e., the time of preparation for the se Perhaps, also, the fact that disturbances of menstruation are so frequent ciation with chlorosis is connected with the conditions above mentione is also possible that the menstrual disturbances may be a result of chlorosi

A third form of primary essential anæmia is the so-called pernicious This, likewise, appears independently of other diseases, and is characte its steady progress, as a rule leading finally to death. We do not regard p anæmia as a very severe form of chlorosis, but as an entirely separate It is distinguished from chlorosis not merely by the much more definite logical changes in the blood, but still more by the circumstance that it is fined to women, but appears also in men, both in early and advanced lif of " severe essential anæmia " also have many points in common with cert diseases, such as pseudo-leukæmia and splenic anæmia. These will be later.

The forms of secondary anæmia offer a contrast to the forms of pr essential anæmia just described in the much greater number of thei Under this head come cases of anæmia which do not occur idiopathicall a result of other abnormal processes. The simplest variety is anæm hæmorrhage. This is produced by profuse loss of blood, whether from t ach, lungs, uterus, intestines, kidneys, or some wound. Repeated small rhages finally produce the same result as a single large one. Thus the found anæmia may be observed where there is a very frequent epistaxis rhagic diathesis, contracted kidney), or where cancer of the womb gives constant oozing of blood.

Other cases of secondary anæmia may be divided into two great gro one class the anæmia is a symptom of impaired nutrition. This is seen every severe disease, acute or chronic, and is usually associated with mo emaciation and loss of strength. The bad appetite, the lack of fresh air cise, and perhaps an impairment of digestion, or fever, or some abnorm upon the system, as in suppuration—these injure the entire body. It is prising that the blood shares in the universal misfortune. This is w chronic invalids seem pale, particularly if they suffer from disease of the kidneys, chest, or nervous system. In the second class, the anæmia is the greatest emaciation, with a subnormal temperature and slow pulse. Of course, such a patient appears pale and wretched, but he does not present that peculiar waxy pallor which is the sign of genuine anæmia.

The exact mode of origin of specific secondary anæmia is often obscure. We have already reported, in a preceding chapter, a very instructive example. In cancer of the stomach we find usually emaciation and pallor. This is natural enough; but sometimes the carcinoma is complicated by an extraordinarily profound anæmia, comparable only to the pernicious variety. In one such case we found, at the autopsy, an extensive secondary carcinosis of the bone-marrow. Here, therefore, the anæmia certainly was not the result merely of the general impairment of nutrition occasioned by the gastric carcinoma, but of the disease of the marrow, which tissue undoubtedly plays an important part in the manufacture of the blood.

Some cases of specific secondary anæmia deserve special mention, although it is seldom possible to demonstrate their precise cause. In the first place come such cases as develop after certain acute diseases, usually of an infectious character. For example, there may be great anæmia after typhoid fever, or less often after acute articular rheumatism. A peculiar form of anæmia is often observed during the secondary stage of syphilis, although nutrition seems to be otherwise well maintained. This is known as "syphilitic chlorosis." Tuberculosis, chronic malarial poisoning, chronic septic infection, and other forms of chronic poisoning (lead), as well as amyloid disease, renal disease, etc., may also be attended by anæmia of such intensity as to justify the conclusion that there is some special disturbance of secondary origin affecting the manufacture of the blood or the blood itself. Of great importance is the striking anæmia which occurs in association with many intestinal parasites (*vide supra*, page 512 *et seq.*), sometimes as a result of actual loss of blood, but probably more often because of the action upon the blood of certain poisons generated by the entozoa.

[Chronic arsenical poisoning from wall papers, dress fabrics, furniture covering, and the like, is a not infrequent cause of anæmia and debility, which resists treatment as long as the exposure to the toxic influence lasts. The classical symptoms of chronic arsenical poisoning are not necessarily present. The number of cases in which prompt recovery has taken place after the removal of the poison which was proved to exist in the surroundings as well as in the urine of the patient is too large and too striking to explain by simple coincidence. Recovery may, however, be slow in those whose toleration of arsenic is slight, or in whom the cause of the symptoms has remained long undetected.]

We shall now proceed to describe the symptoms directly dependent upon the anæmia, and therefore common to all its forms. Upon this will follow a sketch of chlorosis. A special chapter is assigned to the grave form of essential anæmia known as progressive pernicious anæmia; and in the same connection will be set forth the little that is known with regard to the relations of anæmia to pathological changes in the hæmatopoietic organs.

2. **Symptomatology of Anæmia.**—The first symptom which attracts the attention of the physician in any case of anæmia is the altered appearance, the pallor of the skin and visible mucous membranes. This is especially striking in the face, but it is sufficiently evident everywhere. Special value is usually assigned to pallor of the mucous membranes, for example, of the lips and conjunctivæ, inasmuch as their color is not liable to be interfered with by pigmentation or opacity of the epidermis. The degree of pallor, of course, varies greatly. The whole body may present a waxy appearance. A pallor of this kind implies, of course, a very considerable diminution in the amount of coloring matter in the blood—that is, of hæmoglobine. The diminution of hæmoglobine in simple anæmia is usually a direct result of diminution in the number of red blood-corpuscles,

decidedly large, but it diminishes gradually as the creature develops. This indicates that the animal comes into the world with a supply of iron, which can originate only in the maternal organism; and in the first part of his life, so long as he has milk for his exclusive nourishment, he draws upon this supply. If, therefore, we consider that it is a function of the female organism in some way to store up iron for the next generation, there might be some disturbance of this function, either an imperfect assimilation or an abnormal conversion of the assimilated iron, and this might very likely rob the blood of hæmoglobine. It must be confessed that as yet we have no thorough understanding at all of the internal processes of metabolism which relate to the matter. Still, the facts and conditions which we have mentioned explain to a certain degree the fact that genuine chlorosis is found exclusively in the female sex and, indeed, precisely at the time of puberty—i. e., the time of preparation for the sexual life. Perhaps, also, the fact that disturbances of menstruation are so frequent in association with chlorosis is connected with the conditions above mentioned, but it is also possible that the menstrual disturbances may be a result of chlorosis.

A third form of primary essential anæmia is the so-called pernicious anæmia. This, likewise, appears independently of other diseases, and is characterized by its steady progress, as a rule leading finally to death. We do not regard pernicious anæmia as a very severe form of chlorosis, but as an entirely separate disease. It is distinguished from chlorosis not merely by the much more definite morphological changes in the blood, but still more by the circumstance that it is not confined to women, but appears also in men, both in early and advanced life. Cases of "severe essential anæmia" also have many points in common with certain other diseases, such as pseudo-leukæmia and splenic anæmia. These will be discussed later.

The forms of secondary anæmia offer a contrast to the forms of primary or essential anæmia just described in the much greater number of their causes. Under this head come cases of anæmia which do not occur idiopathically, but as a result of other abnormal processes. The simplest variety is anæmia from hæmorrhage. This is produced by profuse loss of blood, whether from the stomach, lungs, uterus, intestines, kidneys, or some wound. Repeated small hæmorrhages finally produce the same result as a single large one. Thus the most profound anæmia may be observed where there is a very frequent epistaxis (hæmorrhagic diathesis, contracted kidney), or where cancer of the womb gives rise to a constant oozing of blood.

Other cases of secondary anæmia may be divided into two great groups. In one class the anæmia is a symptom of impaired nutrition. This is seen in almost every severe disease, acute or chronic, and is usually associated with more or less emaciation and loss of strength. The bad appetite, the lack of fresh air and exercise, and perhaps an impairment of digestion, or fever, or some abnormal drain upon the system, as in suppuration—these injure the entire body. It is not surprising that the blood shares in the universal misfortune. This is why most chronic invalids seem pale, particularly if they suffer from disease of the stomach, kidneys, chest, or nervous system. In the second class, the anæmia is secondary to some other disease, but assumes especial prominence as a symptom, independently of any general impairment of nutrition. Of course, it is often associated with emaciation, but nevertheless its extraordinary intensity offers a striking contrast to the condition of the rest of the body. This "specific secondary anæmia" must, like essential anæmia, be due to a special lesion of the blood itself, and is, therefore, to be regarded as in a certain sense a special complication or localization of the primary disease. General malnutrition never produces anæmia of this grade directly. This fact is illustrated in stenosis of the œsophagus. Here the ingestion of food is very greatly, if not completely, hindered, and there is the greatest emaciation, with a subnormal temperature and slow pulse. Of course, such a patient appears pale and wretched, but he does not present that peculiar waxy pallor which is the sign of genuine anæmia.

The exact mode of origin of specific secondary anæmia is often obscure. We have already reported, in a preceding chapter, a very instructive example. In cancer of the stomach we find usually emaciation and pallor. This is natural enough; but sometimes the carcinoma is complicated by an extraordinarily profound anæmia, comparable only to the pernicious variety. In one such case we found, at the autopsy, an extensive secondary carcinosis of the bone-marrow. Here, therefore, the anæmia certainly was not the result merely of the general impairment of nutrition occasioned by the gastric carcinoma, but of the disease of the marrow, which tissue undoubtedly plays an important part in the manufacture of the blood.

Some cases of specific secondary anæmia deserve special mention, although it is seldom possible to demonstrate their precise cause. In the first place come such cases as develop after certain acute diseases, usually of an infectious character. For example, there may be great anæmia after typhoid fever, or less often after acute articular rheumatism. A peculiar form of anæmia is often observed during the secondary stage of syphilis, although nutrition seems to be otherwise well maintained. This is known as "syphilitic chlorosis." Tuberculosis, chronic malarial poisoning, chronic septic infection, and other forms of chronic poisoning (lead), as well as amyloid disease, renal disease, etc., may also be attended by anæmia of such intensity as to justify the conclusion that there is some special disturbance of secondary origin affecting the manufacture of the blood or the blood itself. Of great importance is the striking anæmia which occurs in association with many intestinal parasites (*vide supra*, page 512 *et seq.*), sometimes as a result of actual loss of blood, but probably more often because of the action upon the blood of certain poisons generated by the entozoa.

[Chronic arsenical poisoning from wall papers, dress fabrics, furniture covering, and the like, is a not infrequent cause of anæmia and debility, which resists treatment as long as the exposure to the toxic influence lasts. The classical symptoms of chronic arsenical poisoning are not necessarily present. The number of cases in which prompt recovery has taken place after the removal of the poison which was proved to exist in the surroundings as well as in the urine of the patient is too large and too striking to explain by simple coincidence. Recovery may, however, be slow in those whose toleration of arsenic is slight, or in whom the cause of the symptoms has remained long undetected.]

We shall now proceed to describe the symptoms directly dependent upon the anæmia, and therefore common to all its forms. Upon this will follow a sketch of chlorosis. A special chapter is assigned to the grave form of essential anæmia known as progressive pernicious anæmia; and in the same connection will be set forth the little that is known with regard to the relations of anæmia to pathological changes in the hæmatopoietic organs.

2. **Symptomatology of Anæmia.**—The first symptom which attracts the attention of the physician in any case of anæmia is the altered appearance, the pallor of the skin and visible mucous membranes. This is especially striking in the face, but it is sufficiently evident everywhere. Special value is usually assigned to pallor of the mucous membranes, for example, of the lips and conjunctivæ, inasmuch as their color is not liable to be interfered with by pigmentation or opacity of the epidermis. The degree of pallor, of course, varies greatly. The whole body may present a waxy appearance. A pallor of this kind implies, of course, a very considerable diminution in the amount of coloring matter in the blood—that is, of hæmoglobine. The diminution of hæmoglobine in simple anæmia is usually a direct result of diminution in the number of red blood-corpuscles,

each one remaining normal; but the red corpuscles themselves may show a diminution in the amount of hæmoglobine, although their number is normal. In order to determine these important facts we must in every case of decided anæmia find out not merely the relative number of blood-corpuscles—i. e., the number of blood-corpuscles in a cubic millimetre of blood—but also the proportion of hæmoglobine in the blood (the simplest way being by the well-known methods of Gowers and Fleischl), and compare the two.

Besides the alteration in complexion, there is always a group of symptoms referable ultimately to an impairment of the normal processes of innervation resulting from a lack of arterial blood. First among these phenomena comes general weakness of the motor system. The voluntary muscles are easily fatigued, and the patient suffers from constant languor. When the anæmia is very great, as after severe hæmorrhage, this weakness may be so pronounced that the patient can neither walk nor stand; but even in moderate anæmia there is more or less impairment of strength.

This diminished nervous energy is shown also by the mental condition. There is no intellectual vigor. The patient is incapable of any great mental exertion, and experiences a constant feeling of weariness and sleepiness. In cases of severe anæmia there is not infrequently a decided tendency to faintness, or actual syncope, as the result of cerebral anæmia.—It is very common to have such subjective symptoms as impaired vision, specks before the eyes, and pain in the eyes. In occasional instances, particularly after a great loss of blood (for example, after hæmatemesis), total amaurosis develops. Examination with the ophthalmoscope shows nothing abnormal, beyond the anæmia of the retina.—Impairment of hearing is exceptional; on the other hand, there is often great annoyance from marked tinnitus aurium, which of course is due to the irritated condition of the auditory nerve, or perhaps also of the auditory center.

The processes of secretion are much less affected. It is true that there is often dryness of the tongue and mouth, but this is mainly subsequent to the loss of large amounts of blood, and probably, therefore, is occasioned less by a diminution in the activity of the mucous and salivary glands than by the desiccation of the tissues. As relates to other secretory functions, the only one which has been carefully investigated is the formation of gastric juice. The earlier experiments on animals caused the opinion to be held for a long while that in anæmia the proportion of hydrochloric acid in the gastric juice was diminished. Numerous more recent experiments, however, have shown that this assumption is subject to exceptions. Although the amount of hydrochloric acid in the gastric juice may be small in anæmia and chlorosis, and although there may be complete absence of free hydrochloric acid in the gastric juice; yet, on the other hand, it is a very frequent experience to find a fully normal or even excessive amount of hydrochloric acid; nor are the motor functions of the stomach, as a rule, disturbed; but it may be that the constipation which is so often present in anæmia is, at least in part, connected with an impairment of the energy of intestinal peristalsis.

The symptoms thus far described are referable partly to diminished functional activity. On the other hand, anæmia gives rise to certain symptoms of irritation in the nervous system. These are, in all probability, expressive of the irritation excited in certain portions of the nervous system by the products of abnormal metabolism, perhaps due to incomplete oxidation. Some of these symptoms, such as the annoying tinnitus, the specks before the eyes, and the vertigo, have been already mentioned. We should also note especially eructations and vomiting as anæmic symptoms; these are in most cases of central origin, and in severe anæmia they may be very annoying. In the same way may be explained the occasional occurrence of annoying hiccough, the frequent spasmodic yawning, and similar symptoms. Anæmic headache may be very severe. It is usually referred either

ıe entire head or to the forehead; there is a painful feeling of pressure, which
attain great severity.

)ther important evidences of irritation are to be found in the behavior of the
e and the respiration. The changes here are apparently in part of a compen-
ry character. The pulse is accelerated in most cases of any severity, reaching
› 100 beats per minute, or even more. It is also very excitable, so that com-
tively slight external influences suffice to increase its rapidity for the time
g. This increase in frequency would not, of course, necessitate an increase of
ı-vascular tension, or of the rapidity of the circulation; but it may exert a
rable influence in this direction, and so be compensatory. Respiration also is
lly accelerated in anæmia. In cases of great severity the breathing may be so
and noisy as to justify the term "anæmic dyspnœa." This is the direct ex-
ion of the body's hunger for oxygen. It is obvious that this increase in the
ber of respirations favors, to a certain extent, the absorption of that gas.

'here are still other symptoms referable to the circulatory system. It has
already stated that the total volume of the blood is not diminished in anæmia
ss, of course, there has just been an actual hæmorrhage. This explains why
pulse in anæmia is often comparatively full and strong. Quite frequently
e is a peculiar quickness of the pulse. This seems to be due to a vigorous
iac systole, in conjunction with a low intra-arterial tension. Akin to these
ıomena is the fact that in severe anæmia it is not exceptional to hear a dis-
pistol-shot sound in the femoral artery, or, in rare cases, even a distinct
ıle sound, just as in aortic insufficiency.

t has long been known that anæmia may produce functional cardiac mur-
s, often called "anæmic murmurs." Their mode of origin is not yet fully
ained. They are usually heard loudest over the base of the heart, in the
hborhood of the pulmonary valves, and at the apex of the heart. As a rule,
are purely systolic in time, but we certainly heard in one case of pernicious
mia a loud diastolic murmur of anæmic origin. The murmurs are of a blow-
character. Sometimes they are so rough as to simulate pericardial friction-
murs; it has therefore been suggested that many anæmic murmurs are ac-
ly due to the rubbing upon each other of the abnormally dry folds of the peri-
ial sac. Another explanation of their occurrence is, that the movements of the
iac valves are interfered with, as a result, perhaps, of fatty degeneration of the
cardium (*vide infra*). It is also possible that there may be sometimes relative
fficiency of the valves—e. g., due to dilatation of the heart or to imperfect
ɔn of the papillary muscles. Still, the question has not yet been settled
her genuine dilatation of the heart is at all frequent in anæmia. Increase in
cardiac dullness is observed, but it is due mainly to retraction of the lungs.

Iurmurs in the large veins of the neck are very often heard in anæmia, either
or without cardiac murmurs. They are often called *bruit de diable*. No
an authority than A. Weil has maintained that murmurs in the jugular veins
just as frequent in healthy persons as in the anæmic; but our experience
pels us to differ from this view. We believe that the loud venous murmurs are
frequent in the anæmic than in other persons, so that they possess a certain
nostic value.

'he processes of metabolism in profound anæmia are of great interest, but, un-
ınately, they have not yet been thoroughly investigated. *A priori*, we should
ct that the absorption of oxygen would be diminished in severe anæmia; but
s not yet been possible to confirm this surmise, at least in the cases of medium
rity, doubtless because the compensatory acceleration of the respiration and
e (*vide supra*) equalizes matters. Whether, even in severe anæmia, there
y diminution in the absorption of oxygen, has not yet been proved; yet many
s seem to favor this idea. One is that in severe anæmia there is usually a

somewhat increased destruction of albumen, as compared with the amount of nourishment ingested. This can be recognized by the increase in the excretion of nitrogen in the urine. This circumstance, which accords with the results of experiments as to the consequences of a diminution in the supply of oxygen, assumes the more importance if we associate it with certain anatomical results of anæmia, there being almost always a marked fatty degeneration of many organs, particularly the heart and kidneys. This fatty degeneration is the direct result of the abnormal destruction of albuminoid structures. The fat represents the non-nitrogenous remnants of the decomposed albuminoids. The reason the fat itself does not undergo oxidation is the lack of oxygen. It is perhaps for the same reason that the panniculus adiposus is for a long while preserved in many cases of anæmia.

It is evident that the fatty degeneration once produced must, in its turn, lead to unfavorable results. It has already been suggested that the fatty degeneration of the heart may be the cause of certain irregularities in its functional activity; but it should be said that this is not invariably the case, for often the heart exhibits a surprising energy despite marked fatty degeneration of its muscular tissue. The corresponding changes in the walls of the blood-vessels are, however, of great importance, as they frequently occasion disturbance, above all by hæmorrhage. In many instances (for example, in leukæmia, *vide infra*) an actual hæmorrhagic diathesis is developed; but we shall find later (see the chapter on pernicious anæmia) that other factors may also play a part in producing capillary hæmorrhages in severe anæmia. There is strong evidence in many cases of an abnormal permeability of the vascular walls. From this results the mild grade of œdema frequently seen in anæmic patients, although in some few cases this œdema may be ascribed to passive congestion, resulting from the cardiac debility (*vide supra*). The increased permeability of the renal blood-vessels is sometimes shown by polyuria.

The urine is usually rather light-colored, if the anæmia be at all marked. It is evident that there is a diminished production of urinary pigment, the material for which is the coloring matter of the blood. Another reason for the light color of the secretion is the polyuria above mentioned. There may be fifty to seventy ounces (1,500–2,000 cubic centimetres), or more, secreted in twenty-four hours. The specific gravity may nevertheless be comparatively high, and higher than one would expect from the appearance of the urine, not infrequently ranging between 1015 and 1021. This indicates, of course, that the amount of solid constituents is comparatively large; and in fact we find a corresponding amount of urea, perhaps four or five hundred grains (grammes 25–32) in twenty-four hours. This is a large figure when we consider the amount of ingesta. As to the other constituents of the urine, we possess as yet little definite knowledge. The amount of phosphoric acid is sometimes surprisingly small, compared with the amount of nitrogen. Albuminuria is exceptional in cases of simple anæmia.

The bodily temperature is very often affected in anæmia. An "anæmic fever" is very frequently associated with pernicious anæmia, and even with cases of profound secondary anæmia, as after a large hæmorrhage from the stomach. There are irregular elevations of temperature, usually occurring in the evening. and attaining 101°–102° (38.5°–39° C.), or even still higher figures. This fever is not due to inflammatory organic changes, but, in all probability, is rather the result of the presence in the blood of matter due to the disintegration of the blood-globules (perhaps fibrine ferment).

Clinical History of Chlorosis.—Chlorosis, or "green sickness," is a definite form of primary essential anæmia, which appears exclusively in the female sex and in the years of adolescence, mainly from the fifteenth to about the twentieth year of life. The disease either may appear quite suddenly in a previously healthy girl, and in such a case may completely disappear again after a few weeks or months; or the entire course may be a chronic one without sharp limits, so that the condition approaches more nearly to a constitutional anæmia. In many cases, also, it would be proper to say that there are recurrences of chlorosis, for severe attacks of the disease may be seen more than once in the same girl. As a rule, there is no definite cause to be found for the appearance of chlorosis, except that sometimes it seems to be promoted by an unhygienic mode of life, too many social demands, or long-continued mental excitement.

The various symptoms of chlorosis are almost all direct results of the anæmia, so that they have already been described. Their intensity and variety are, however, very different in different cases. There are mild cases where the patient can hardly be called ill. She feels perfectly well, but is "a little pale." From these cases there is a gradual and unbroken transition to the other extreme of severity.

A constant and essential symptom is the greater or less pallor of the face as well as of the rest of the surface of the body, and of the mucous membranes so far as visible. This pallor sometimes really has a greenish tint. There is almost invariably general languor; the patient is easily fatigued, and has neither the desire nor the ability to make any great bodily or mental exertion. There is also a tendency to headache and vertigo. Chlorotic patients often complain of dyspepsia. The appetite is usually diminished, and there is often a sense of pressure in the epigastrium after meals. There may be severe cardialgia. It is not always easy to determine its cause, often it seems to be purely nervous. Sometimes there may be myalgia. Moreover, chlorosis may be associated with a genuine ulcer of the stomach, as has been already mentioned. We believe, however, that this combination is much less frequent than is commonly supposed, for many physicians make a diagnosis of gastric ulcer upon insufficient data. The earlier assumption that the dyspeptic symptoms of chlorosis are associated with a lack of hydrochloric acid in the gastric juice is entirely incorrect. If we make a careful investigation of the gastric functions in chlorotic girls, we shall find that the amount of hydrochloric acid in the gastric juice is normal or even excessive. That gastroptosis may often be found in conjunction with chlorosis is not surprising (*vide supra*, page 462). An attempt has even been made to regard chlorosis as a result of gastroptosis, although of course this is entirely incorrect. The bowels are usually constipated in chlorosis, which is explained by the impaired appetite and the sluggish intestinal peristalsis.

Over the cervical veins we often hear a loud murmur, which has been above mentioned as the *bruit de diable*. Examination of the heart often shows a slight increase in the area of cardiac dullness, although this does not seem due in most cases to an actual dilatation of the heart, but merely to a retraction of the borders of the lung. Anæmic cardiac murmurs are often heard, the pulse is accelerated and easily excited. Otherwise physical examination does not reveal any abnormal condition of the internal organs. It is a very great exception to find any indications of disturbance in the spleen or lymph-glands, or the bone-marrow. Fever is rare in mild cases of chlorosis, but in severe cases we sometimes see, especially at night, a slight rise of temperature, up to 101° F. (38.5° C.). The urine is usually pale, but seldom differs greatly, either in amount or constituents, from the normal character. It is noteworthy that chlorotic girls are very apt to suffer from disorders of menstruation. The menses may not appear until late, or they are very scanty. Even complete and long-continued amenorrhœa is often seen in chlorosis. All these disturbances seem to point to an essential relation between the appearance of chlorosis and the phenomena of sexual life. It is only in exceptional instances that chlorotic patients have menorrhagia.

Examinations of the blood have been made, in the hope of gaining a clearer insight into the true character of chlorosis. The blood is usually pale. Upon microscopic examination, we notice that the red blood-corpuscles do not tend to form rouleaux so much as normally, and sometimes we can perceive that the globules are comparatively light-colored, and are not all of the same size. Some are of the normal dimensions, others are notably small (microcytes), while there are a few which are unusually large (macrocytes). Irregularities in the shape (poikilocytosis) are also seen, particularly in severe cases. In mild chlorosis all these changes in the form of the red blood-corpuscles are slight, or scarcely appreciable. The white blood-corpuscles are present in normal amount, or sometimes somewhat above normal, so that there is a slight leucocytosis. In occasional instances there are considerable numbers of "granular bodies" in the blood. These are usually regarded as products of the disintegration of white blood-corpuscles.

Malassez, Hayem, Thoma, and others have endeavored to determine the number of blood-corpuscles in chlorosis and allied diseases by means of special methods of counting. In general, we may say that in many cases of severe chlorosis the number of red blood-corpuscles is decidedly diminished. In a cubic millimetre of blood we find perhaps only 3,000,000 to 3,500,000 red blood-corpuscles, instead of the normal number of 5,000,000. The diminution may be even greater than this. On the other hand, it should be noted that Duncan, Hayem, and Laache have met with cases where the number of corpuscles was not diminished; but the amount of hæmoglobine in the blood-corpuscles was diminished. Not infrequently, when the number of red blood-corpuscles is but slightly diminished, perhaps the amount of hæmoglobine in the blood is much reduced, to thirty or forty per cent. of normal. This justifies the conclusion that in chlorosis the number of red corpuscles is less affected than is their constitution, particularly the proportion of hæmoglobine contained in them. How this lack of hæmoglobine in the red corpuscles comes about we do not yet know.

We have already intimated that there is great variety in the general course of chlorosis. Many cases which seem severe at first terminate in complete recovery by the end of four to six weeks, or within a few months. Other cases are much more obstinate, resist all modes of treatment, and undergo frequent relapses; so that with its manifold variations, improvements, and exacerbations, the disease may occupy one or two years or more. The prognosis may be regarded as favorable on the whole, but it should always be given with a certain degree of reserve. It is true that ordinary chlorosis never involves direct danger to life. We shall, however, find that, so far as symptoms go, there is no sharp dividing-line between "simple chlorosis" and "pernicious anæmia"; and in any individual case it may not at first be possible to decide which variety of anæmia is before us.

There are hardly any true complications of chlorosis. If other organic diseases, such as pulmonary tuberculosis or gastric ulcer, exist along with chlorosis, one should always consider that the anæmia may have developed secondarily, or that the diseases may be present from mere coincidence. All the varieties of nervousness and hysteria are frequently found in association with chlorosis, and yet it can not be assumed in every case that there is an essential causative relation between them.

Diagnosis.—The diagnosis of chlorosis is, in a certain sense, easy, for the characteristic symptoms, the pallor, and the ordinary phenomena consequent on constitutional anæmia, are always easy to appreciate; but, on the other hand, the diagnosis is not perfectly simple, for the anæmia must be actually a primary essential anæmia in order to justify the name chlorosis. We therefore have no right to declare the diagnosis of chlorosis until we have made a careful and thorough physical examination, and have found that no factors are present which might produce a secondary anæmia. We should, above all, bear in mind the possibility of incipient tuberculosis, and examine the lungs, the expectoration, and the general condition of the patient, and also consider hereditary and other predisposing influences. We should bear in mind the possibility of some organic disease of the stomach, such as ulcer, catarrh, or dilatation, or chronic renal disease, or possibly constitutional syphilis, giving rise to the syphilitic chlorosis before mentioned. In many cases it is easy to exclude all these secondary forms of anæmia, but occasionally the task is a very difficult one. In making a diagnosis of chlorosis it is very important, also, to consider the entire course of the disease, particularly the rapid onset of the anæmia at the time of adolescence in young girls, and the greater or less disturbance of menstruation.

Treatment of Anæmia and Chlorosis.—The first indication in treating chlorosis, as well as every other form of anæmia, is to promote the regeneration of the blood. There are two ways in which we can endeavor to fulfill this indication: first, by general hygienic and dietetic measures, and, secondly, by employing certain internal remedies.

First among general measures comes a care for pure air, sunlight, and proper food. Many a pale city girl regains her ruddy cheeks after a few weeks spent either in the country, or on the mountains, or at the seashore. The choice of a place must, of course, depend mainly upon the circumstances of the patient. In many instances, boarding in any suitable country place answers as well as a long and expensive journey. If the seashore be chosen, some bathing resort on the North Sea will usually be preferable. There are other places, where the patient can combine the advantages of pure forest air and ferruginous mineral water; these will be mentioned below.

The diet of anæmic patients should be easily digestible and rich in albumen; carbohydrates and fat should be given in but limited amounts to patients in whom the panniculus adiposus is already well developed. Lean patients, on the other hand, require such ingredients in their diet, and should be recommended to take simple puddings, extract of malt, butter, and cod-liver oil. Milk, if well borne, is certainly not an unsuitable food for anæmic patients, but we do not ascribe any special value to the drinking of large amounts of milk as a cure for chlorosis. In the first place, milk contains an extremely small amount of iron, and secondly, it is very apt to cause troublesome constipation. Formerly it was not uncommon to order "milk-cures," but we regard this as decidedly unsuitable. On the other hand, we would especially advise the use of the yolks of eggs, provided eggs are well borne by the patient, and are not the object of strong aversion. The yolk of egg is comparatively rich in hæmatogen, which is a nucleine-like compound of iron from which it is probable that hæmoglobine is formed; emphasis should likewise be laid upon the use of an abundance of green vegetables, such as spinach, for these all contain a large amount of iron. Some authors ascribe an exaggerated value to alcoholic beverages. They may be allowed in moderate amount if the patient asks for them and finds the appetite improve under their use. The best to choose for an emaciated patient are porter and other varieties of beer rich in extractive principles, but wine is often ill borne by chlorotic girls, being apt to cause palpitation, etc.

Many physicians also insist upon "abundant exercise in the open air." This can, however, be carried too far. A chlorotic girl is often made to take long walks in spite of her own reluctance to do so, with the result of becoming more languid and exhausted than she was before. We even believe that a case of profound anæmia will be benefited by a certain degree of bodily rest. Thus any needless tax upon the muscles is avoided. The most brilliant results we have ever observed in the treatment of chlorosis have been within the hospital. The patients, who are usually factory operatives and servant girls, are often kept in bed for the first two or three weeks. We admit that pure country air may be very beneficial, but we would strongly advise moderation in bodily exercise. If the patient begins to feel more vigorous, she will of herself take more exercise.

Of internal remedies, the preparations of iron have long occupied the first place in the treatment of all varieties of anæmia. How these exert their influence is at present quite uncertain, for the body needs so small an amount of iron that the iron contained in ordinary food seems to be quite sufficient for the demands of the system; still, the therapeutic efficiency of iron, particularly in chlorosis, has been established by so many instances that there is scarcely room for doubt. On the other hand, in simple constitutional anæmia the efficacy of preparations of iron is much more uncertain and doubtful.

The number of iron preparations that are recommended and are used is very large, and is continually being increased by the commercial manufacturers of drugs, although there is no actual need of such increase. Almost every physician has his favorite prescription, to which he ascribes the greatest efficiency. We ourselves employ most frequently in chlorosis the long- and well-known Blaud's pills. We can assure the reader that they are almost always well borne, and often cause a surprisingly rapid and decided improvement in chlorosis. We usually prescribe them in the following form: Dried sulphate of iron, pure carbonate of potassium, each one hundred and fifty to two hundred and twenty-five grains (grammes 10–15); tragacanth, q. s. This makes one hundred pills, of which two to four are to be given three times a day after meals. Other ferruginous remedies which are likewise often useful are reduced iron, which is absolutely pure iron in a very finely divided powder, and is prescribed as such or in pills, from one to three grains (grammes 0.05 or 0.2), to be given several times a day; two preparations, ferri carbonas saccharata (U. S. P.), and ferrum oxydatum saccharatum solubile (Ph. G.), are prescribed as loose powders, of which one half to one teaspoonful may be given in water three times a day. The last named has a special advantage in that it does not blacken the teeth. Another good preparation of iron is the lactate, which may be given in powders and pills, in the dose of five to twelve grains (gramme 0.3–0.75). The tinctures of iron are, in general, scarcely to be recommended, and are usually employed only for children. The liquor ferri albuminati (Ph. G.) deserves praise; a teaspoonful of it is given three times a day. It often has a good effect upon the appetite. Of late there have been repeated trials of the internal administration of organic compounds of iron. First, hæmoglobine itself was tried (Pfeufer's hæmoglobine tablets or hæmoglobine extract); also Schmiedeberg's "ferratine"; hæmagallol, which was recommended by Kobert; hæmatogen, hæmalbumen, and many others. All these preparations have found their champions, but it is by no means certain that they act better than the old approved inorganic forms of iron. Often iron is given in combination with other remedies, of which quinine is chief. This is said not only to increase the tonic action, but to lessen the headaches to which anæmic patients are subject. Stomachic tonics, such as gentian, or laxatives, such as aloes, are also combined with iron.

Exceptionally iron is not well borne, and causes digestive disturbances or diarrhœa. We must then try another preparation or diminish the dose. It is usual to forbid the ingestion of sour articles of diet while taking iron, but the injunction is due mainly to prejudice.

The use of mineral waters containing iron is quite common, although the amount of iron thus introduced into the system is so small that it is hard to understand their therapeutic value. Of those artificially produced, that which contains the pyrophosphate of iron is the best, and seldom irritates even a delicate stomach. The natural chalybeate waters are also bottled and sent to distant places. They are often more effective if drunk upon the spot, but merely because, in that case, the general hygienic surroundings of the patient are better than they would be at home. The best-known and most popular ferruginous springs in Germany and Switzerland are situated at Cudowa, Rippoldsau, Elster, Schwalbach, Pyrmont, Steben, Kohlgrub, Driburg, Liebenstein, and St. Moritz. "Steel-baths" are also much employed; but the effect is due not to the iron contained in the water, but to the temperature of the water and the carbonic-acid gas it contains. Baths generally seem to be of decided service in chlorosis, and hence we often prescribe simple baths or salt-baths, giving a bath of fifteen or twenty minutes' duration, at a temperature of 90°–95° F., two or three times a week, adding five or six pounds of salt to the bath.

Some other remedies besides iron are occasionally employed in chlorosis. Of these, the chief is arsenic. Its effects are often very beneficial, and it deserves a trial, especially in severe cases. It may be given alone or in combination with iron. We have repeatedly seen cases of chlorosis which showed no improvement after the use of iron, in which the combination of iron with small amounts of arsenic, a thirtieth to a fifteenth of a grain (gramme 0.002–0.004) three times a day, had a strikingly beneficial effect. Scholz and Strübing recommend the administration of sulphur in chlorosis—purified sulphur, ten parts; sugar of milk, twenty parts; dose, as much as can be lifted on the point of a knife, three times a day. Other physicians extol manganese. No especial advantage, however, has been shown to pertain to these remedies.

Although the constitutional treatment is always the main consideration, yet sometimes special symptoms demand attention. Violent headache may be relieved by antipyrine, or similar remedies; dyspeptic symptoms are to be treated chiefly by careful injunctions as to diet. The patient should avoid pressure from without upon the region of the stomach, and should have easily digested food. The internal administration of hydrochloric acid is proper only when the examination of the gastric juice has shown that there is a diminution in hydrochloric acid; as a rule, there is an abundance of hydrochloric acid secreted (*vide supra*), and if so, alkalies are preferable.

If there be constipation, it is desirable to remedy it rather by suitable diet than by laxatives. Thus we may prescribe fruit, Graham bread, and other foods which promote peristaltic action by the mechanical irritation which they exert; also gentle massage of the abdomen, and only when these fail, injections or mild laxatives.

CHAPTER II

PROGRESSIVE PERNICIOUS ANÆMIA

(Grave Form of Essential Anæmia)

Definition and Ætiology.—We apply the term pernicious anæmia to a severe and rare form of anæmia which is usually fatal, and which develops as a primary disease, without demonstrable cause, in both men and women. We must hold fast to this conception of the disease, especially as the attempt has been repeatedly made of late to degrade pernicious anæmia from the rank of an idiopathic disease, and regard it as merely a grave form of secondary anæmia which may result from any one of the most various causes.

Of course, the diagnosis of primary anæmia may be wrongly made in cases where a more careful search will detect some special cause for the condition. Such a mistake may become evident at the autopsy. Thus, in repeated instances, cases which have been during life regarded as "pernicious anæmia" have been shown to be cancer of the stomach (*vide* page 448). In other cases, however, it is more difficult to discover the primary cause of the anæmia. Thus the profound anæmia which attacked the men at work upon the St. Gothard Tunnel was at first regarded as idiopathic, but more careful investigation showed that the disease was really due to anchylostoma (*vide* page 578), the ravages of which parasite have in other cases also been confounded with the symptoms of pernicious anæmia. Reyher and Runeberg have lately called attention to the fact that the presence of bothriocephalus latus in the intestine may produce an affection resembling severe and apparently primary anæmia. Of late there have been a number of cases reported where the post-mortem examination showed that the progressive marasmus and anæmia of the patient had resulted from an extensive atrophy of the coats of the stomach and—what is more important—of the intestine, associated in some instances with well-marked lesions of the sympathetic plexus. These cases are very interesting, but have nothing whatever to do with pernicious anæmia. Often the symptoms even during life differ not a little from those of pernicious anæmia, so that we can not think it right to establish what is called a "gastro-intestinal variety of pernicious anæmia." It is proper to group and classify the forms of secondary anæmia according to their various causes, but not the cases of primary idiopathic anæmia, which are all due to some trouble with the manufacture of the blood, or with the blood itself, and form a distinct clinical group.

The credit of having been the first to study pernicious anæmia as a special form of disease belongs to Biermer (1868), although occasional instances of the disease had long before been noticed. Its occurrence during pregnancy was first pointed out by Gusserow. [The puerperal form of pernicious anæmia was described by Walter Channing in 1842.]

With regard to the special ætiology of progressive pernicious anæmia, it must be confessed that we have as little definite information as about the ætiology of chlorosis. Often it has been thought that the disease might be infectious, but the discoveries thus far reported of specific micro-organisms in the blood—for instance, the so-called cercomonads of Klebs and Frankenhäuser—lack confirmation. There do not usually seem to be any special exciting causes. It has been maintained that unfavorable hygienic surroundings and insufficient nourishment promote the development of the disease; and this would apply perhaps to some cases, but certainly it does not to all. We are inclined to regard it as characteristic of this disease, as well as of chlorosis, that the anæmia may develop despite the most favorable outward circumstances. There is, however, one factor which seems to have a decided influence, and that is the condition associated with pregnancy and childbirth. These are comparatively often the occasion of the development of the first symptoms of the disease in women. Still, pernicious anæmia has this essential difference from chlorosis, that, as we have already said, it is not confined to women. Most cases occur in middle life, from about twenty-five to forty years of age. It is a very interesting but somewhat puzzling fact, that the disease may appear as a sequel to a great loss of blood, whether from one or several hæmorrhages. It seems as if the body were sometimes unable to recover from the effects of large hæmorrhages of this sort, so that the acute merges into a chronic and progressive anæmia, which, despite nursing and treatment, advances to a fatal termination. It is quite doubtful, however, whether such cases should be classed as genuine pernicious anæmia.

Pathology.—Pernicious anæmia so often terminates fatally that there has been abundant opportunity to make accurate anatomical investigations of the lesions it produces. We shall not here discuss the changes in the blood itself, inasmuch as they will more properly be described among the clinical phenomena. The changes in the internal organs are divisible into two groups: First, such as are secondary and the result of the anæmia; and, secondly, such as may perhaps be primary and characteristic. All the internal organs are, of course, anæmic. Another striking change is fatty degeneration. This is usually best marked in the heart, but it also affects the kidneys, the liver, and the walls of the stomach and intestines, as well as the intima of the blood-vessels. We have already pointed out that this fatty change is to be regarded as the direct result of the anæmia, probably because of the diminished supply of oxygen in the tissues.

The second important lesion of pernicious anæmia is the hæmorrhages, usually small, but occasionally large; these are usually numerous, and are found in all sorts of locations. The most important of these hæmorrhages are into the retina, because they can be demonstrated by means of the ophthalmoscope during life. They are very frequent. We may also find minute hæmorrhages in the serous membranes, such as the pleura or pericardium, in the brain and in the mucous membranes. Cutaneous ecchymoses are also occasionally found. We shall revert later to the ætiology of these hæmorrhages.

Still another secondary symptom is an abundant deposit of iron in the cells of many organs. This can usually be determined only by the aid of the microscope or micro-chemical tests. It is most abundant in the peripheral zone of the hepatic lobules, but it also occurs in the kidneys, pancreas, and other organs. Quincke has studied this point very carefully, and finds that the total amount of iron contained in the liver in pernicious anæmia is much larger than normal. The most natural and most probable interpretation of this fact is that the iron originates from the destruction of large numbers of red blood-corpuscles.

The changes thus far described are undoubtedly the result of anæmia, and hence are the same in all severe anæmias, whatever their cause. On the other hand, search has been made for specific changes which could be regarded as the primary disease, and thus, of course, our attention has been especially directed to the organs engaged in the formation of the blood. The lymph-glands do not usually present any special changes in pernicious anæmia. If they are much altered, it is probable that the disease is a different one, which we shall later have an opportunity to study (*vide* pseudo-leukæmia). The spleen is in many cases normal. In exceptional instances, however, it is decidedly enlarged, although never enormous. Even when the organ is enlarged there are no important histological changes to be detected. Cases of pernicious anæmia with marked splenic tumor are often termed splenic anæmia (*vide infra*), but we do not ourselves see how such cases differ essentially from others in which the spleen is not enlarged. The changes in the bone-marrow are by far the most constant. This structure plays an important part in the formation of the blood. C. Wood, and after him Cohnheim, called attention to the fact that the bone-marrow is almost invariably affected in pernicious anæmia. Instead of its normal yellow color, it has a dark-red color, like raspberry jelly. This is due mainly to the fact that the numerous fat-cells of the marrow are all, or nearly all, destroyed. This is the more surprising as the fatty tissues in other parts of the body are often very little affected in pernicious anæmia. The specific cellular elements of the marrow also exhibit certain changes. There is a decided hyperplasia, and, as a rule, there are found large numbers of nucleated red blood-corpuscles. Cohnheim is inclined to regard the disease of the bone-marrow as specific and primary, but it must be confessed that there are many reasons which strongly oppose this view, and suggest the possibility of this change in the marrow being merely a secondary phenomenon, a sign of the extremely active formation and regeneration of the red blood-corpuscles. Neumann's investigations would lead to the belief that the nucleated red blood-corpuscles are young blood-cells in the process of development. Furthermore, these vigorous processes of regeneration, and the corresponding changes in the bone-marrow, are often found in cases of profound anæmia which are beyond a doubt of secondary origin.

We can not, therefore, feel certain that the lesion of the bone-marrow is the primary anatomical disturbance, and hence we are obliged to assume that essential anæmia may be a disease of the blood itself—that is, some specific but unknown process which does direct injury to the red blood-corpuscles.

Symptoms.—As already stated, the symptoms of pernicious anæmia usually begin, independently of any demonstrable cause, in individuals previously healthy, and develop so slowly and gradually that it is hardly ever possible to determine the precise date of the commencement of the disease. This is still more likely to be the case if the trouble occurs, as indeed it may, in individuals who were previously feeble and pale without being actually ill. Occasionally a more acute onset is observed in pregnant women.

The first symptoms are almost invariably traceable to the incipient anæmia. They include the subjective disturbances and the objective changes which are seen in any ordinary anæmia. The patient feels languid and is easily fatigued, is liable to headache, vertigo, palpitation, and tinnitus aurium; there are anorexia, frequent nausea, and, above all, a striking pallor of the skin and mucous membranes, but while these symptoms in ordinary anæmia and chlorosis are moderate, in the severe forms of essential anæmia they are extreme.

In every well-marked case of pernicious anæmia the weakness of the patient is so great that he is permanently confined to bed, and standing or sitting for any length of time is impossible. Usually the patient lies upon his back, with his head rather low, and presents a countenance of waxy pallor. Very frequently a slight but distinct yellowish hue may be detected. Occasionally there are cutaneous ecchymoses, but these are exceptional. The mucous membrane of the lips, the gums, and the conjunctivæ are likewise pale and colorless. The intellect is unimpaired, but all answers to questions are slow, apathetic, and delivered in a low and feeble tone of voice. The patient is usually incapable of any great mental exertion. He is sleepy, and often yawns. If the body be moved, and especially if an upright position be substituted for a horizontal one, there is great liability to syncope. This may result from any slight physical exertion, and is often accompanied by a peculiar spasmodic rigidity of the body. The main subjective symptoms are weakness, and more especially intense headache; this often assumes a pulsating character, and is located mainly in the temples or forehead. There is also an annoying ringing in the ears, described as a singing, rushing, or roaring sound. There are certain other subjective sensations, namely, nausea, a sense of thoracic oppression, and pain in the bones, which will be considered later on.

If we now proceed to a systematic physical examination, we are struck, in the first place, by the condition of the eyes. The pupils are often somewhat enlarged, but react in a normal manner. Vision is often disturbed by spots before the eyes. The anæmic amaurosis seen after a sudden large hæmorrhage has not yet been observed in pernicious anæmia. The ophthalmoscopic examination of the fundus has very great diagnostic importance. We find in a majority of cases, although not in all, retinal hæmorrhage. There may be either one or many hæmorrhages. If they are extensive enough to involve the macula lutea or the disk, they may disturb vision greatly. Retinal hæmorrhages invariably signify that the anæmia is profound, and are, with considerable justice, regarded as distinguishing pernicious from simple anæmia.

Respiratory Symptoms.—Breathing is usually accelerated, and, in the most advanced cases, is often remarkably deep and noisy (anæmic dyspnœa, *vide supra*). Sometimes there is a very annoying and almost painful sense of thoracic oppression, which is evidently connected with the dyspnœa. There is a “hunger for air.” Physical examination of the lungs gives negative results. Sometimes there is a little cough, and there may be sufficient hæmorrhage into the mucous membrane of the air-passages to give rise to a slight hæmoptysis. Even in this case no anætomical changes can be detected during life. We may also mention in this connection that epistaxis is not very infrequent.

Phenomena referable to the circulatory system are of still greater clinical importance. The area of cardiac dullness is usually normal, although sometimes slightly increased. Upon palpation, we often find that the heart's action is exaggerated, and that its beat is felt over a larger area than normal. The pulse is usually decidedly rapid (100–120), but it is regular, and sometimes, but by no means invariably, small. It is often surprisingly strong. The loud "anæmic murmurs" are very characteristic. They can be appreciated at the apex of the heart, but are usually heard still better at its base. We generally hear a loud *bruit de diable* in the veins of the neck.

Digestive Organs.—The tongue is usually pale, smooth, and dry. The appetite is often very poor. The most prominent disturbances, however, are not due directly to the condition of the stomach, but they result from the cerebral anæmia —that is, they are symptoms of the irritation of the nervous centers. We refer to eructations and vomiting, which may be very frequent and distressing. There is generally a tendency to constipation. Occasionally there is diarrhœa.

The liver is usually normal; as is also the spleen in many instances, while in others it is shown by percussion and palpation to be enlarged. It is sometimes possible, as we can ourselves bear witness, to observe an enlargement of the spleen increasing as the anæmia grows more profound, and again decreasing if improvement occurs (*vide infra*). On ordinary examination, the urine does not usually differ essentially from normal. With few exceptions it is free from albumen, and it never contains sugar. As already set forth, however, accurate quantitative analysis often furnishes important evidence of changes in tissue-metamorphosis resultant upon the anæmia (cf. page 714). We will merely mention once more the comparative increase in the amount of urea excreted, and the occasional excess of uric acid. Sometimes the urine gives a vivid reaction for indican.

In regard to what has already been said about the marrow of the bones, it is an interesting fact that they are very sensitive to pressure in many cases of pernicious anæmia. The sternum in particular is painful upon light percussion; and sometimes pressure will cause pain in the bones of the extremities. In rare instances swelling of the knee and other joints has been observed. A series of very interesting observations have been recently made by Lichtheim, Minnich, and others, which show that persons with primary or even secondary pernicious anæmia (e. g., anæmia due to the bothriocephalus) may quite suddenly develop well-marked nervous symptoms. These consist chiefly in paræsthesiæ in the hands and legs, in slight disturbances of sensation, unsteadiness and ataxia of the legs, weakness or complete abolition of the tendon reflexes, etc.—in brief, a group of symptoms resembling tabes. The pupils have been observed to become irresponsive to light. Upon the post-mortem examination of such cases, there is found, particularly in the posterior columns of the spinal cord, a well-marked disease which consists for the most part in small scattered foci of degeneration, and in a more systemic (secondary) degeneration of the columns of Goll. Sometimes small capillary hæmorrhages are also found. Slight changes of this sort may be observed in the spinal column, even when no special spinal symptoms had been present. Probably these conditions are due to toxic action.

The blood has been made the subject of numerous and careful investigations; nevertheless, no characteristic change has been discovered. The changes presented in pernicious anæmia likewise occur in cases of profound secondary anæmia. This seems the more readily intelligible to us because of the view which we have already expressed on page 710 with regard to the origin of secondary anæmia. To the naked eye the blood seems extremely pale and watery. The number of red blood-corpuscles is sometimes so diminished that it seems incredible that life should persist. It is not at all unusual, in the worst stages of the disease, to find only 500,000 to 1,000,000 red blood-corpuscles per cubic millimetre, or even 250,000—that is, even less than one tenth of the normal amount. The proportion of hæmoglobine in the blood is consequently much diminished, but comparative determinations of the number of blood-corpuscles and the amount of hæmoglobine seem to indicate that the individual blood-corpuscles have not lost any of their coloring matter. The red blood-corpuscles are found to present striking varieties in size and form (*vide* Fig. 79). While some corpuscles have a normal appearance, others may be of an unusually large size (macrocytes). These "giant corpuscles" appear normal except in dimensions, and some observers (Laache) have thought that they were even possessed of an unusual amount of hæmoglobine. It is therefore surmised that they represent an effort on the part of nature toward compensation. In contrast with these large cells are found a varying number of minute red cells of a spherical shape: these were first described by Vanlair and Masius, and called microcytes. Finally, there are numerous red blood-corpuscles of abnormal shape. These Quincke was the first to notice. They present remarkable forms, being biscuit-shaped, hammer-shaped, or anvil-shaped, and so on, as illustrated in the accompanying cut. These "poikilocytes" are found in perfectly fresh undiluted blood, so that there is no reason to suppose that they are artificial products. Both the microcytes and the poikilocytes are regarded as degenerative forms of the blood-corpuscles. They are the products of the division and constriction of old blood-corpuscles. Careful histological examination discloses several definite and histologically distinct forms of degeneration in the red blood-corpuscles. We have already pointed out that the microcytes and poikilocytes are not essentially characteristic of pernicious anæmia, but occur in other severe forms of anæmia; but besides these products of degeneration, we find in the blood, also, indications of an increase in the formation of new elements. We refer to the nucleated blood-corpuscles. Ehrlich, to whom we are indebted for the best investigations with regard to this matter, distinguishes the so-called normoblasts and megaloblasts. The normoblasts are nucleated red blood-corpuscles of ordinary size, which correspond to the youthful forms of red blood-corpuscles normally present. The megaloblasts, on the other hand, are nucleated structures which exceed the ordinary red blood-disks in size, two to four fold. Megaloblasts are found normally in the embryo alone, so that Ehrlich conceives the appearance of megaloblasts to be a sort of reversion to the embryonic state. The appearance of megaloblasts in the blood is very characteristic of pernicious anæmia. The white blood-corpuscles are not usually increased in number; in occasional instances, however, a temporary leucocytosis has been found. "Granule-masses" are often found in considerable abundance. Chemical examination of the blood has not as yet brought to light any facts of great importance. It has been already stated that there is a great diminution in the total amount of hæmoglobine. The amount of albumen in the blood-serum remains nearly normal.

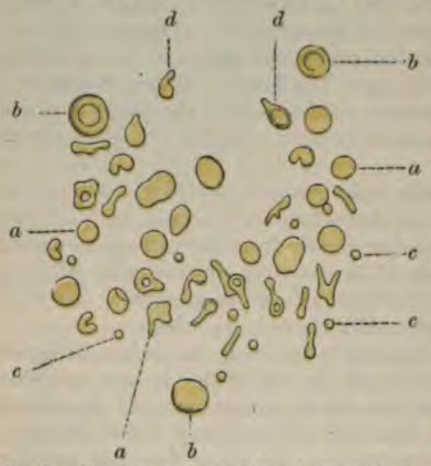

FIG. 79.—Changes in the red blood-corpuscles in pernicious anæmia. (From QUINCKE.) *a*. Normal blood-corpuscles. *b*. Macrocytes. *c*. Microcytes. *d*. Poikilocytes.

We observe in pernicious anæmia the tendency to fever common to all varieties of profound anæmia. In many cases the evening temperature will for weeks reach 100°–101° (38°–38.5° C.), or even higher. Previous to death, however, the temperature may become subnormal, falling to 86° (30° C.), or even lower.

As to the origin of all these symptoms we must, as we have said, regard the impairment of the blood, especially its poverty in red blood-corpuscles, as the essential morbid process. How this impairment is produced, whether by injury to the blood-corpuscles themselves or to the organs where they are formed, we do not know. A large part of the other morbid symptoms—the pallor of the skin, the weakness, the "anæmic cerebral symptoms," tinnitus, vertigo, nausea, faintness, etc., are due directly to the anæmia. In regard to certain other important symptoms, however, another factor is probably of great significance, namely, an auto-intoxication of the body with fibrine ferment or some similar substance.

By the destruction of many red blood-corpuscles, probably a certain amount of hæmoglobine is always set free, and enters the blood plasma. There is then a hæmoglobinæmia. From many experiments, however (Dorpat school of Alexander Schmidt, Ponfick, Silbermann, etc.), we know that hæmoglobine dissolved in the blood destroys the white blood-corpuscles, and in some way produces from them fibrine ferment. Chronic "ferment intoxication" of the body causes certain symptoms, which are almost never absent in pernicious anæmia—capillary hæmorrhages and fever. The hæmorrhages are usually associated with embolism of the smaller vessels, seldom with local thrombi. Many severe nervous symptoms (*vide supra*) may perhaps also be referred to this intoxication.

General Course, Duration, and Prognosis.—As the very name "pernicious" indicates, the disease generally terminates unfavorably. Death usually seems to be the direct result of the extreme anæmia; special complications are exceptional. The disease often maintains a slow but gradual progress to the end. Its duration, reckoning from the appearance of the first symptoms, may not exceed three to six months. It may even run its course in a still shorter time. It seldom lasts more than a year. Sometimes its course is interrupted; there may be an arrest of the process, or improvement, or even apparent recovery. Usually, however, there are fresh relapses. In a certain class of cases the disease lasts two or three years, and is marked by a number of "attacks of anæmia" so intense that the subsequent improvement of the patient seems simply marvelous. It is in cases of this sort that splenic tumor has been made out at the time the anæmia was at its height; yet we do not perceive the necessity of establishing "splenic anæmia" as an affection essentially different from progressive pernicious anæmia. It is merely a clinical variety of the disease under discussion. Apparently, it likewise has an invariably fatal termination.

Permanent recovery may occur in cases of idiopathic anæmia so profound that we are at first inclined to regard them as pernicious. These cases are unfortunately very rare, and even when there is a marked improvement the danger of a relapse is to be borne in mind. The prognosis is therefore always very grave, if not absolutely unfavorable. Of course, general hygienic surroundings and good care may exert some influence upon the course of the disease. It is noteworthy that if pregnancy be complicated by profound anæmia there is a great liability to premature delivery, after which there is often a rapid change for the worse. There are exceptions to this rule.

Diagnosis.—It is seldom difficult to make out the existence of a profound anæmia, or to determine the degree of danger which the consequent symptoms indicate. We have, however, the same difficulty here as in chlorosis in proving that the anæmia is primary and idiopathic. The factors essential to this diagnosis have been already indicated. We should bear in mind the possibility of insidious tuberculosis, organic diseases of the stomach, or such parasites as the anchylostoma and bothriocephalus.

Treatment.—For treating progressive pernicious anæmia we have only the same remedies as for the benign variety of anæmia. We must provide the patient with nourishing and digestible food, and regulate all other hygienic matters; and in the way of medicine the greatest prospect of benefit lies in arsenic, while the efficiency of ferruginous preparations in pernicious anæmia is dubious. We may, however, try a combination of iron with arsenic. The best form in which to administer the arsenic is pills, but subcutaneous injections, also, may be tried, in the manner which will be fully described in the following chapter. Besides arsenic, some physicians recommend phosphorus.

If the case be not too far advanced, baths may prove useful adjuvants to the internal treatment. Salt baths or artificial carbonic-acid baths may be employed. Symptomatic treatment is often indicated: the dyspepsia may call for dilute hydrochloric acid, or the troublesome vomiting may require bits of ice, bromide of potassium, or opium.

The transfusion of blood has been employed in pernicious anæmia. Sometimes the effect seems to be favorable. Experience thus far, however, would not lead one to expect very great benefit from it. Von Ziemssen has lately recommended in all severe forms of anæmia the subcutaneous injection of blood. It is done by injecting under the skin, by thoroughly disinfected instruments, fifty cubic centimetres of defibrinated human blood, divided into two parts, twenty-five cubic centimetres being injected into each thigh. The point of injection is vigorously rubbed, and thus the injected blood is forced into the lymph-channels. In severe cases this procedure should be frequently repeated. Experience of this method has thus far been very favorable. Not only the subjective symptoms, but the objective condition of the blood, the number of red blood-corpuscles, sometimes show a striking improvement after the injection.

[In the experience of American and English clinicians, arsenic, long continued and in as large doses as are tolerated, is of far greater value than iron.

The inhalation of oxygen is expensive, but seems to be of distinct service in some cases of grave anæmia of all forms.]

CHAPTER III

LEUKÆMIA

(*Leucocythæmia*)

Definition and Ætiology.—Virchow, in 1845, was the first to obtain an insight into the disease leukæmia ("white blood"). He detected the great increase of white corpuscles occasioned by it, and from this time these constituents of the blood were subjected to observation in all sorts of diseases. It was soon found that there may be a temporary increase of the white corpuscles in various primary diseases, and that in certain instances this increase may actually constitute the essential symptom. In this latter case the increase is due to a depraved condition of certain internal organs. In the temporary cases, in which the increase of white blood-corpuscles is usually not very great, we find one white corpuscle to one hundred red, or even one to fifty—the normal ratio being one to six hundred or more. This is usually termed leucocytosis, in distinction from leukæmia proper. If we take for normal a count of about 6,000 to 10,000 leucocytes in a cubic millimetre of blood, the term leucocytosis is applied to conditions in which the number of leucocytes rises to 15,000 or 20,000, or even still higher up to and beyond 40,000. A leucocytosis of this sort is found, under normal conditions, at the time of digestion, also in pregnant women and in the new-born; under pathological conditions, chiefly in certain acute febrile diseases, particularly croupous pneumonia, erysipelas, and sepsis.

Genuine leukæmia is a rather rare disease. Its characteristics are well marked in most cases, but of its true nature we remain entirely ignorant. In a majority of the cases the change in the blood is associated with marked changes in the spleen and the bone-marrow, and often also in the lymph-glands. The organs just enumerated being concerned in the manufacture of the blood, and particularly of the white corpuscles, it is very reasonable to suppose that leukæmia is a disease which primarily affects these organs, and that the increase in white corpuscles results from the disturbance thus occasioned. The cause of the disease in the organs mentioned is as yet unknown. Various authors have suggested that there may be some specific infection, but they have not been able thus far to produce any evidence of the truth of their surmise. In few cases can we discover even any exciting cause. The illness seems to develop spontaneously in perfectly healthy persons. In some cases, on the other hand, leukæmia does seem to be a sequel of some other disease. Thus it occasionally follows a tedious attack of intermittent fever. It has also been asserted that syphilis and other infectious diseases, such as typhoid fever, may give rise to leukæmia, but this is not very probable. Finally, trauma of the spleen or bones has in repeated instances been regarded as the occasion of the disease. The hygienic surroundings of the patient have also been regarded as a cause of leukæmia. The disease is somewhat more frequent among the poorer classes than among the wealthy; but there are numerous exceptions to this rule. Ætiological importance has also been ascribed to anxiety, trouble, and mental depression in general. Still, all these apparent exciting causes are scarcely ever of real importance, and in most cases of leukæmia there is, as we have said, no evidence of any of them.

Leukæmia is most common in middle life, between thirty and forty-five years of age, but well-marked cases have been observed repeatedly even in childhood, as also, though less frequently, in old age. Men are somewhat more liable to the disease than women. It has been repeatedly stated that in female patients the disease is sometimes referable to sexual derangement, but apparently this was mistaking effect for cause.

Pathological Anatomy.—The essential change in leukæmia is a great increase in the number of white blood-corpuscles. The characteristics of the blood can be studied during the life of the patient, and upon them the diagnosis is mainly based. They will therefore be discussed under symptomatology, while we shall here confine ourselves to the lesions presented by the spleen, bone-marrow, and lymph-glands.

Of the organs just enumerated, the spleen is the one most frequently affected (splenic leukæmia). It is often greatly increased in size, and may attain a weight of six to eleven pounds (3–5 kilogrammes) and a length of a foot (30 centimetres). There is a true hyperplasia of the whole organ: all the histological constituents are increased. The cut surface is usually a rather vivid red in early cases, but later on it often has a lighter, yellowish color. The consistency is usually diminished, but in the later stages it may be greater than normal. Upon microscopic examination, we find enlargement of the blood-vessels and a great increase in the cells of the pulp and of the follicles. Sometimes the hyperplasia of the follicles predominates, giving the spleen a spotted appearance, like marble. In such cases the pulp usually presents retrograde metamorphosis, with atrophy and fatty degeneration of its cells and deposits of pigment. In advanced cases a considerable amount of firm connective tissue may be present. There are often hæmorrhagic infarctions, presenting the appearance of circumscribed spots, dark red, or in the later stages brownish yellow, in color.

Lesions of the bone-marrow are next in frequency to those of the spleen (medullary or myelogenous variety of leukæmia). Neumann and a few other authorities regard changes in the bone-marrow as an essential lesion, and hold that they can be demonstrated in every case of leukæmia. There would certainly seem to be exceptions to this last rule, but nevertheless in a majority of cases the marrow does present a peculiar yellowish or almost puriform appearance. By means of the microscope we can detect a great increase in the lymphoid cells of the marrow and the presence of a considerable number of nucleated red blood-corpuscles.

In many cases the lymph-glands remain perfectly normal; but in others they become considerably enlarged, and form actual tumors in various parts of the body, such as the axilla, neck, and groin, and sometimes the internal lymph-glands. Such cases are examples of lymphatic leukæmia. Histologically, the change here is simple hyperplasia of the glandular tissue.

These three forms of leukæmia—splenic, myelogenous, and lymphatic—can not be regarded as distinct diseases, inasmuch as cases occur which present all sorts of combinations of these different lesions. Purely myelogenous cases are very rare, if they occur at all; and we rarely meet with cases which are purely splenic or purely lymphatic. Most cases present lesions of the spleen and marrow conjointly. In less frequent instances splenic disease is associated with that of the lymphatic glands. The fact that these combinations exist indicates that there is one common cause for the disease, which assails sometimes one, sometimes two, or sometimes all three of the organs named.

Just what the connection is between the changes in the blood and the lesions of these various organs is an unanswered question. The view which seems to us most plausible regards the disturbance in the spleen, marrow, or lymph-glands, as the case may be, as the primary one, and the alteration in the blood as a result of this primary disturbance; there is an increase in the number of colorless corpuscles formed, and a consequent increase in the number of them introduced into the circulatory fluid. It is true, however, that the number of red blood-corpuscles is usually diminished in leukæmia. This diminution in number might be ascribed either to scantiness of supply or to increase in the processes of destruction, but which factor is in reality the important one must remain undetermined.

Changes in Other Organs.—Leukæmia sometimes causes new growths of a lymphatic character in certain organs other than those already mentioned. The growths may either be diffuse or circumscribed. They are observed in the tonsils, Peyer's patches, and the intestinal follicles. They are also found very frequently in the liver, kidneys, and retina, and more rarely in the lungs and the pleura. These various lesions may be regarded as in a certain sense analogous to the metastatic tumors of cancer or sarcoma, and suggest the possibility of the diffusion of the pathogenic poison throughout the whole body. In one or two cases a well-marked leukæmia has been found independent of any demonstrable organic lesions. It is impossible, at present, to explain such occurrences. Leube and Fleischer have reported a case of this sort, and are inclined to believe that the blood itself was diseased.

There is little known as yet as to changes in the chemical composition of the blood and viscera in leukæmia. Glutine, xanthine, and hypoxanthine have been found in the blood, as have also lactic acid and formic acid. It is also interesting that nucleo-albumen is found in the blood serum of leukæmic patients, for this fact points directly to destruction of the blood-corpuscles (Matthes).

It is noteworthy that octahedral crystals are often found after death in the blood, spleen, and marrow, and in other parts. These are known as Charcot's crystals, and have already been described as occurring in the sputum in bronchial asthma (see page 184).

Symptoms.—The clinical phenomena of leukæmia are in many respects similar o those of progressive pernicious anæmia, and need not be enumerated again ere. In leukæmia, however, we have, in addition, symptoms referable to the pleen, lymph-glands or bone-marrow (as the case may be), and also the charcteristic alterations in the blood. The blood-changes, being pathognomonic, emand a full description.

The pallor and watery character of the blood in leukæmia are noticeable even o the naked eye in advanced stages of the disease. They can not, however, be istinguished without the aid of the microscope from the changes present in grave næmia (*vide* Fig. 80). In this way it is often possible to determine by the first lance that there is an enormous increase n the white corpuscles, their number eeming of more significance because the umber of red globules is apt to be coniderably diminished. If an accurate count s made we find in a cubic millimetre of lood about 2,500,000 to 3,500,000 of red lood-corpuscles and about 300,000 to 500,00, or even 700,000 leucocytes. The roportion of white to red blood-corpusles is one to twelve, or even one to eight nd one to seven. The earlier reports of atios of one to two and one to one are robably due to inaccurate counting. Still more interesting than these ratios re the special forms of leucocytes; even in he fresh unstained preparation it is easy o appreciate that there are decided differences in the size and character of the individual leucocytes; but all these ifferences become far more distinct in the stained preparation* (Fig. 81). In his we see that a very large proportion of the leucocytes consists of the ordiary polynuclear cells, but we also find small mononuclear cells, called lymphoytes, which probably originate in the lymph-glands, so that an increase in the umber of them is regarded as especially characteristic of the lymphatic form of eukæmia. We also see in most cases numerous large mononuclear cells, almost wice as large as a white blood-corpuscle, so that they can be distinctly recogized even in the fresh unstained specimen. These cells are at present usually ermed marrow-cells, or myelocytes, because they are found in large numbers in he bone-marrow, both in normal conditions and particularly also in leukæmic ubjects, so that it is regarded as probable that the myelocytes which are present n leukæmic blood originate in the bone-marrow. Finally, we ordinarily find in very microscopic field several so-called eosinophilous cells—that is, cells whose arked granulation is noticeable even in the fresh specimen, but becomes still ore striking when the granules are stained red with eosine. As to the origin bone-marrow?) and the significance of eosinophiles, we have as yet no certain nowledge, nor can we make absolute statements as to the source of the lymphoytes and myelocytes. Many investigators regard the mononuclear, small eucocytes, or lymphocytes, as merely the immature form of the polynuclear and arge nucleated cells, into which they are supposed to develop. The red bloodorpuscles in leukæmia often undergo certain changes in size and shape (poikiloytosis), but seldom to such a degree as in grave essential anæmia. We should

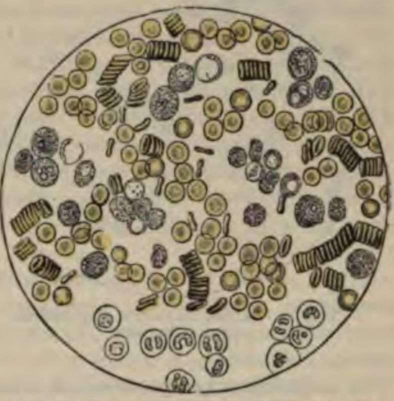

FIG. 80.—Leukæmic blood. (From FUNKE.)

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* For particulars with regard to the various methods of staining the blood (which are chiefly due o P. Ehrlich), the reader is referred to text-books on methods of clinical examination.

mention, however, that in leukæmia we almost invariably find nucleated red blood-corpuscles, and these are exclusively normoblasts (see the preceding chapter). Granular masses are usually present in leukæmic blood in large numbers.

Splenic tumor is the most frequent and important of the organic lesions produced by leukæmia. It is rarely possible to observe its development. In most instances the spleen is already large when the patient first comes under observation. It projects from under the ribs as a firm, hard mass, the lower and anterior extremity of which often extends to the median line of the body. The inner edge of the tumor is somewhat characteristic; it is rather sharp, and presents one or two notches. At first there is little subjective disturbance or pain referred to the spleen. When the enlargement is very great, there is often an annoying or even distressing feeling of distention and fullness in the abdomen. Respiration may also be interfered with by the crowding up of the diaphragm.

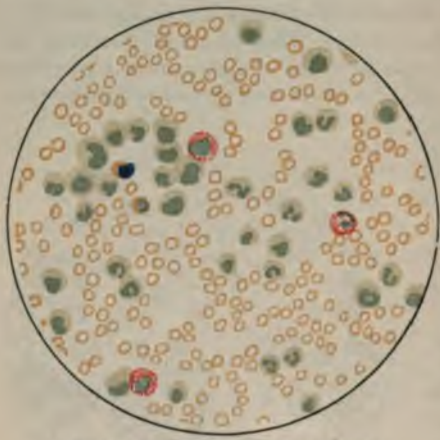

FIG. 81.—Mixed leukæmia (myelæmia). (From RIEDER.)

The lesion of the bone-marrow can be determined during life mainly if at all by the myelocytes (*vide supra*). The only direct symptom which renders its existence probable is pain in the bones, but even this is not an infallible symptom. There is seldom pain except upon pressure. It is usually brought out by percussion of the sternum, but there may be well-marked disease of the marrow without this "sternal pain."

As already stated, the lymph-glands often remain perfectly normal. If they also are affected, the disturbance is betrayed by their increase in size. Not only may the glands in the neck, axilla, and groin be enlarged, but occasionally also those of the mesentery and retroperitoneum, as can be demonstrated upon palpation of the abdomen. The enlarged lymph-glands rarely cause severe pain, if any at all. If the blood were found to contain large numbers of small mononuclear lymphocytes, this would suggest, as has already been said, involvement of the lymph-glands. Unfortunately, however, our knowledge as to the significance of all the various forms of leucocytes is too imperfect as yet to justify absolute diagnostic conclusions.

We have already referred to leukæmic new growths in other internal organs. These possess, for the most part, merely a scientific interest, as they cause no special symptoms. Sometimes hepatic enlargement occurs, as the result of a diffuse leukæmic infiltration. The changes in the retina associated with leukæmia are of importance, as they can be detected by means of the ophthalmoscope. The retina presents white spots or stripes running parallel with the blood-vessels. They are due to collections of lymphoid cells, or to actual lymphoid growths. These lesions have been inappropriately called leukæmic retinitis. Retinal hæmorrhages occur also in leukæmia, as in grave cases of idiopathic anæmia.

All the other clinical phenomena of leukæmia result from the abnormal condition of the blood, meaning thereby the anæmia. The ability of the blood to perform its normal functions is impaired mainly through the loss of red blood-corpuscles; the resulting symptoms are therefore precisely the same as in essential anæmia, and we do not need to describe them again. They usually are the most prominent symptoms of the disease, and include noticeable pallor of the skin, equal to that seen in pernicious anæmia; anæmic murmurs over the heart and the veins of the neck; general debility; anorexia, and digestive disturbances; palpitation and dyspnœa; and, finally, the whole group of the "cerebral symptoms of anæmia," that is, headache, vertigo, syncope, and tinnitus aurium. Sometimes there is a troublesome pruritus. Provided he has no great anæmia, the general condition of a leukæmic patient may be fairly good. A symptom that is observed remarkably often in men suffering from leukæmia is persistent priapism. This symptom may, as we ourselves have known, be the first one noticed by the patient. Its cause is to be sought not in conditions of nervous irritation, but in the formation of white thrombi, rich in leucocytes, in the corpora cavernosa (Kast).—In a few cases paralysis of the cranial nerves has been observed, such as the facial, hypoglossal, and acoustic. This paralysis is due sometimes to small hæmorrhages, and sometimes to degenerative changes in the medulla oblongata.

We would also call attention once more to the frequent hæmorrhages. These must be due to impaired nutrition of the vascular walls, and sometimes they justify our speaking of a "hæmorrhagic diathesis." Obstinate epistaxis is particularly frequent. Less often we have hæmorrhage from the intestine, stomach, kidneys, or into the skin or muscles, etc. We may have cerebral hæmorrhage, with hemiplegia, or sometimes immediate death, consequent upon it. Severe cases may present a slight œdema of the skin and serous effusions into the various cavities of the body.

The urine in leukæmia is essentially like that excreted in pernicious anæmia. Fleischer and Penzoldt have shown that in leukæmia as well as in pernicious anæmia there is increased destruction of albuminoids, and a consequent relative increase in the excretion of nitrogen. There is often also a considerable increase of uric acid. These rules, however, do not seem to be without exceptions.

The temperature is apt to undergo slight elevations, as in severe cases of anæmia. In advanced stages there may be quite high fever of an intermittent character, reaching 103°–104° (39.5°–40° C.). The fever is sometimes accompanied by chills, and when the temperature falls there is often a profuse and debilitating perspiration.

Complications are, on the whole, rare. Sometimes we observe pulmonary tuberculosis, or some acute intercurrent disease such as pneumonia. We saw one case end in death from hæmorrhagic angina and œdema of the glottis.

Clinical History.—Leukæmia is almost always chronic in its course. The disease begins insidiously and progresses gradually; the patient grows pale, feels languid, and has slight and apparently insignificant symptoms, which gradually give place to alarming phenomena. The patient may himself notice the organic lesions incident to the disease. If the leukæmia be of the lymphatic type, he is apt to be struck by the swelling of the lymph-glands, while in splenic leukæmia his attention is attracted by the feeling of tension and pressure in the abdomen, the increasing prominence of the left side, and the unusual sense of resistance present in that part of the abdomen. Sometimes it is obstinate epistaxis, or hæmorrhage from some other source, which first attracts attention and leads to a careful examination of the blood and spleen.

The entire duration of the disease is usually one or two years or perhaps more. Many cases pursue a rather benign and slow course, while in others there is a more rapid advance of all the symptoms, even justifying the term of acute leukæmia. Attention has been particularly directed to such cases by Ebstein. In them the changes in the spleen and lymph-glands may be only moderate. Usually, however, there is a distinct splenic tumor. The lymph-glands are almost invariably involved, as is found at the autopsy, if not before. The constitutional symptoms are very severe and grow rapidly worse, and, in particular, there is apt to develop an acute hæmorrhagic diathesis, with hæmorrhages into the skin and the internal organs, including the brain. Often the temperature rises to 104° (40° C.). Although there may be many fluctuations in the course of the disease, it leads, apparently invariably, in a few weeks to death. For diagnosis a microscopic examination of the blood is essential. It is characteristic of acute leukæmia, as A. Fränkel has pointed out, that the leucocytes in the blood are, in an overwhelming proportion, the small mononuclear lymphocytes (immature form, *vide supra*). These cases of acute leukæmia give one the decided impression of an acute infectious disease.

In ordinary cases of leukæmia we see frequently an apparent arrest of the disease, or temporary improvement, and fresh relapses. The final termination is, however, almost invariably unfavorable. Recovery from leukæmia is not absolutely impossible; still, it is very rare, and at the most can be hoped for only during the early period of the disease. In almost all the advanced cases the prognosis must be described as absolutely unfavorable. Death usually takes place with symptoms of profound anæmia, and progressive bodily weakness. Sometimes the end is hastened by the occurrence of dangerous hæmorrhage from the nose or into the brain, or by some intercurrent disease.

Diagnosis.—Leukæmia can be easily and unmistakably recognized by a microscopic examination of the blood. In a very early stage of the disease the increase of white blood-corpuscles may be so slight that a definite decision can not be made; but the later developments will afford absolute certainty in any typical case.

We can not fail to recognise a case of leukæmia if the blood be examined. Such an examination is therefore demanded in every case of obstinate anæmia, and, above all, in such patients as have a chronic enlargement of the spleen, or swelling of the lymph-glands in various parts of the body. The enlarged lymph-glands are readily recognizable. The splenic tumor can usually be diagnosticated from its characteristic position and shape, and especially from its inner, rather sharp edge, usually notched. It may be simulated by hydronephrosis and other diseases causing enlargement of the kidneys, and in women by ovarian tumors. In cases of doubt the blood should be examined, and if the result be a positive one, we may feel certain of our diagnosis. If there is a chronic enlargement of the spleen without a leukæmic change in the blood, we must consider all the possible causes of such an enlargement; thus there may be a passive congestion of the spleen, with enlargement, as the result of hepatic disease, portal thrombosis, or disease of the heart, or a splenic tumor from malarial poisoning. Again, there are cases where we have the signs of a gradually progressive anæmia, apparently idiopathic, and a chronic enlargement of the spleen, or still more frequently enlargement of the lymph-glands in various parts of the body, without increase in the number of white blood-corpuscles. Such cases are termed pseudo-leukæmia, or splenic anæmia, and are described in the next chapter.

Treatment.—Nearly the same remedies are employed in leukæmia as in idiopathic anæmia. Of course the greatest attention should be paid to the general condition. The patient should be well nourished, and perhaps he may be sent into the country or to some health-resort. Of internal remedies, the preparations of iron have been mainly employed. They seldom produce any brilliant or permanent effects. We have much more confidence in the administration of arsenic, and this remedy should certainly be tried in large doses. It may be given either in the shape of pills, or, with still more advantage, subcutaneously. Ziemssen recommends that fifteen grains (gramme 1) of arsenious acid should be boiled with seventy-five minims (5 cubic centimetres) of a fifteen-per-cent. solution of sodic hydrate, and the solution then diluted to make three ounces and a third (grammes 100). Of this one-per-cent. solution of arsenate of soda, at first one quarter, then one half, and, finally, an entire Pravaz's syringeful, is injected once or twice a day. [A Pravaz's syringe holds about thirteen minims.—V.] Of course, the only hope of permanent benefit from this treatment would be in incipient cases. We have seen repeatedly, however, most satisfactory although temporary improvement follow the persistent use of arsenic in leukæmia.

What are called " splenic remedies " have been often employed, but they do not seem very effectual in leukæmia. Mosler obtained good results from the long-continued use of quinine (5–8 grains = 0.30–0.50 gramme, or more, in twenty-four hours). He also recommends a trial of piperine and oil of eucalyptus:

℞ Olei eucalypti .... gtt. 100;
Piperini,
Ceræ albæ .... āā ʒj (gramme 4.0);
Pulv. altheæ .... ʒij (gramme 7.5).
M. et fiant pilulæ no. c.
S. Three to five pills three times a day.

Local treatment of the spleen has been attempted. If an ice-bag be constantly kept on the splenic region, it will sometimes diminish the size of the tumor, and it may also relieve pain. Botkin has recommended faradization of the spleen, but we can scarcely expect any great benefit from such a procedure. Injections of quinine, arsenic, and other remedies, have been made into the substance of the spleen. We do not believe that this is advisable, because it may cause dangerous hæmorrhage. The splenic tumor of leucæmia has actually been extirpated surgically; but the proceeding is so ineffectual and so fatal that it is now universally abandoned. The transfusion of healthy human blood has also been tried, but without satisfactory results. Subcutaneous injections of blood (*vide supra*) have been tried in only a few cases, but they may, perhaps, have good results. In some recent cases inhalations of oxygen are said to have improved the symptoms.

CHAPTER IV

PSEUDO-LEUKÆMIA

(*Hodgkin's Disease. Adenia. Malignant Lympho-sarcoma. Pseudo-leucocythæmia*)

It was mentioned in the preceding chapter that there are cases in which the organic lesions are apparently the same as in genuine leukæmia, and yet there is little if any increase in the number of white corpuscles in the blood. There is generally, however, a diminution in the number of red corpuscles. These cases usually receive the name which Cohnheim gave them of pseudo-leukæmia. It is nevertheless doubtful whether they are to be regarded as a special form of disease, and there are various facts which indicate that they are at least very closely allied to genuine leukæmia. There is a great similarity in most of the symptoms of the two diseases and in their general course, as well as in the organic changes they produce. Furthermore, a case of pseudo-leukæmia may finally assume the character of genuine leukæmia, with its characteristic blood changes.

The purely splenic type of pseudo-leukæmia is the least frequent one. As yet very few such cases have been reported. There is a gradually increasing anæmia with the usual symptoms, and associated with these increasing enlargement of the spleen. It is impossible to draw any sharp dividing line between such cases and cases of pernicious anæmia attended with moderate enlargement of the same organ (splenic anæmia). It may be said to be a matter of taste which name the physician shall give to a case of this sort. The bone-marrow seems to present the same characteristics in splenic pseudo-leukæmia as in pernicious anæmia.

Pseudo-leukæmia Lymphatica.—Pseudo-leukæmia of a lymphatic type is a much more frequent and well-defined disease. It was first described in 1832 by the Englishman Hodgkin, and is sometimes called Hodgkin's disease. Wunderlich was the first in Germany to study the disease thoroughly; he described it in 1858 under the name of "progressive multiple hypertrophy of the lymph-glands"; and later Billroth termed it "multiple malignant lymphoma." Trousseau gave it the name of "adenia."

Little is known about the ætiology of lymphatic pseudo-leukæmia. The tendency of late is to assign adenia to the group of infectious tumors, although the reasons for this belief are as yet purely theoretical. Many cases seem to be directly referable to tuberculosis, in the form of tubercular adenitis without other tubercular disease. Whether this is true as regards all cases of pseudo-leukæmia is as yet very doubtful, but this point should receive especial attention in the future. Pseudo-leukæmia is most common in young and middle-aged persons, and is apparently rather more frequent in men than in women.

Pathological Anatomy.—The hyperplasia of the lymph-glands may be very great, producing large tumors of varying consistency. These have been called lymphoma, lymphadenoma, and lymphosarcoma. On section, the tumors display a white or grayish-red surface, and are seen to be made up of a number of swollen glands fused into nodular masses. Upon microscopic examination, we find a very abundant proliferation of lymph-cells, sufficient to obscure the reticulum of the gland completely. The new growth may even escape beyond the capsule of the gland and invade surrounding structures. Inflammatory adhesions often take place between the tumor and the overlying skin. There does not seem to be any essential difference between the harder and the softer varieties of these tumors.

These changes in the lymph-glands are often, though not invariably, associated with a swelling of the spleen. Its increase in size is usually slight. Lymphomata may also develop in the tonsils, the intestinal lymphatics, liver, kidneys, and other organs. Whether there are changes in the bone-marrow has not yet been determined.

The **symptoms** are very gradually developed. It is almost invariably the swelling of the lymph-glands which first attracts the attention of the patient or his physician. The glands upon one or both sides of the neck are usually the first to be enlarged, and they may finally grow to tumors the size of the fist, or even larger, producing great disfigurement. To the changes in the neck succeed swelling of the axillary, inguinal, and perhaps also the internal lymph-glands. The changes are gradual, and vary in their rapidity and extent.

At first the general health is hardly at all affected, but as the disease progresses its constitutional effects become more and more marked. The patient grows pale and languid, and finally presents all the symptoms of profound anæmia. We may also have certain symptoms due to mechanical compression occasioned by the growth of the lymphomata. The tumors in the neck may cause dysphagia, from compression of the pharynx and œsophagus; dyspnœa, from compression of the larynx and trachea; and perhaps alarming cardiac disturbance, from interference with the vagus. Hypertrophy of the bronchial glands sometimes occasions great difficulty in respiration; enlargement of the abdominal glands may produce ascites or jaundice; and enlargement of the glands in the groin may give rise to œdema in the lower extremities. In advanced stages we may have "cerebral symptoms of anæmia" precisely similar to those seen in genuine leucæmia or in pernicious anæmia. There may be a tendency to hæmorrhage, pruritus, and occasionally prurigo, and the urinary secretion and temperature may be abnormal. For particulars the reader is referred to the preceding chapters on leucæmia and pernicious anæmia.

Upon examination of the blood, there are usually found the changes characteristic of ordinary anæmia, without increase in the number of white blood-corpuscles. Sometimes, however, there may be a slight leucocytosis; and sometimes, as already said, lymphatic pseudo-leukæmia may merge into genuine leukæmia. The examination of the blood must therefore be repeated from time to time. The spleen should also be examined. It is usually somewhat enlarged, and in some cases the enlargement may be considerable. Such cases might be properly denominated splenic-lymphatic pseudo-leukæmia. We may also discover a tenderness of the sternum or other bones.

The disease often lasts but a few months; it may, in rare instances, extend over two or three years or more. Recovery is not completely impossible in the early stages of the disease (*vide infra*), but at a later period the prognosis is absolutely unfavorable. The fatal termination results either from increasing debility and anæmia, or from the effects of mechanical compression, or from hæmorrhage, or from some intercurrent disease.

A peculiar form of pseudo-leukæmia has lately been described by Ebstein and Pel. In this there are, at intervals of about ten to fourteen days, periods of marked fever, which last, also, about the same length of time. The condition has received the name chronic relapsing fever. Death occurs with symptoms of anæmia; and the autopsy shows firm swelling of the liver, spleen, and the retroperitoneal, mesenteric, and bronchial lymph-glands. Less often the external lymph-glands also are swollen, so that they can be felt during the life of the patient. The character of the blood is not leukæmic, so that these cases must be provisionally regarded as pseudo-leukæmia.

The diagnosis of pseudo-leukæmia is usually easy. It is to be based upon the objective signs and the condition of the blood. The disease is most apt to be confounded with swelling of the lymph-glands occasioned by tubercular infection; but in this latter case the changes are seldom seen in so many parts of the body, and the patient usually presents other indubitable evidences of tuberculosis. (See also under ætiology.)

Treatment.—We possess only one remedy capable of promoting absorption of the lymphomata, namely, arsenic. We have ourselves, in common with a great number of observers, had the most convincing evidence of the favorable influence of arsenic. It must, however, be given in sufficient doses: for example, a pill containing one fifteenth of a grain (gramme 0.004), or even a larger amount of arsenious acid, three times a day; and its use must be persisted in for a long time. Still more efficient are subcutaneous injections of the solution of arseniate of soda which we have described quite fully in the preceding chapter. We have also seen apparent benefit from associating with the arsenic inunctions of iodoform (iodoform one part, vaseline fifteen parts) over the tumors.

In the early stages decided benefit may be expected from this mode of treatment. At a later period we may obtain a decrease in the size of the tumors, but we can hardly hope for any permanent improvement. Operative interference is out of the question, except at the very beginning of the disease. Later on, it would be perfectly useless, and it could seldom be carried out.

Other suggestions with regard to treatment may be obtained from the chapters on anæmia and leukæmia.

CHAPTER V

HÆMOGLOBINÆMIA AND HÆMOGLOBINURIA

Definition and General Ætiological Considerations.—If any cause produces a solution of the red blood-corpuscles in the blood-serum, hæmoglobine is excreted through the kidneys. The hæmoglobinæmia—the presence of free hæmoglobine in solution in the blood—excites hæmoglobinuria, i. e., the excretion of hæmoglobine in the urine. The causes of hæmoglobinæmia and its correlative hæmoglobinuria are manifold. In the first place, there is a whole series of poisons which, if introduced into the blood in sufficient amount, exercise a directly destructive influence upon the red blood-corpuscles, and thus excite hæmoglobinuria. To this list belong chlorate of potash (Marchand), pyrogallic acid and naphthol (Neisser), sulphuric acid, glycerine, toluylendiamine, and many other substances. Distilled water is also in this sense a poison. Boström has discovered a fact of practical importance which deserves mention in this connection. It is, that a certain kind of mushroom (*Helvella esculenta*), when fresh, contains a poison which is capable of producing intense hæmoglobinuria, and such grave symptoms as jaundice, delirium, drowsiness, and tetanic convulsions, with perhaps a fatal termination. This poison is, however, so evanescent and so readily soluble in hot water that the mushroom becomes perfectly harmless if thoroughly soaked and then boiled, or if it has been dried.

Secondly, hæmoglobinuria may be developed in connection with infectious diseases. In this case, also, it is probably referable to the action of poisons created within the system. Thus hæmoglobinuria has been observed in the course of a severe attack of scarlet fever or typhoid fever. Possibly malarial poisoning and syphilis may give rise to paroxysmal hæmoglobinuria. This question will be discussed later on.

There is a third mode of origin which also possesses practical importance. If blood from one animal be injected into another of a different species, hæmoglobinuria is almost sure to result. Not only do the injected blood-corpuscles undergo solution, but also the injected serum acts as a poison upon the original blood-corpuscles, destroying and dissolving them. This transfusion-hæmoglobinuria has been described by Prévost, Dumas, Ponfick, and Landois. It can be produced in human beings, as there was only too good opportunity to observe during the brief period when the transfusion of lamb's blood was in vogue. The practical deduction is evident, that we should not use for injection into the circulatory system of a patient anything but an unirritating salt solution, or human blood.

A fourth and very important ætiological factor is exposure to extremes of temperature. Hæmoglobinuria follows extensive burns. The blood-corpuscles in that region of the periphery exposed to the heat are destroyed. Cold is capable of producing precisely analogous results. This is particularly evident in the cases of so-called paroxysmal hæmoglobinuria described by Wickham Legg, Lichtheim, Murri, and Küssner. [Raynaud's disease (see page 900) and hæmoglobinuria are associated in a sufficiently large proportion of cases to show some relationship between the two.]

Pathology and Symptoms of Hæmoglobinæmia, particularly the Paroxysmal Variety.—In most of the cases above enumerated, hæmoglobinuria is the result of an obvious or easily demonstrable cause. There is, however, another variety which appears paroxysmally in individuals who are otherwise perfectly well. Its symptoms are extremely characteristic. Although not a very frequent disease, there has been abundant opportunity to study it.

As just intimated, the disease is paroxysmal. Very often an attack is ushered equent and persistent yawning. To this symptom are soon added pain in s, headache, nausea, vomiting, and coolness of the periphery. The tem- speedily rises to 102° (39° C.) or more. With this is often associated a chill. Sometimes there is a violent pain in the hepatic region. There is t enlargement of the spleen. Then the temperature falls again, perspira- pears, and the patient, although languid and depressed, soon recovers. icteric hue can almost invariably be detected toward the end of the at- ordinary duration of which is from two to twelve hours. An eruption of a has been repeatedly seen in connection with the attacks.

most interesting phenomenon of all remains to be described. We refer to ition of the urine during and directly after the paroxysm. This secretion a dark brownish-red color resembling blood; it may even appear almost Its reaction is almost invariably acid; its specific gravity is usually w, say 1008–1012. On boiling the urine, the hæmoglobine is decomposed rown coagulum of albumen formed. If the fresh urine be examined a spectroscope, we find the bands characteristic of hæmoglobine, and es also the narrow bands indicative of methæmoglobine. It is therefore ble to doubt the existence of hæmoglobine in the urine; and yet, upon pic examination, we find no red blood-corpuscles in the urine, or, in ords, no "hæmaturia." Frequently there are large numbers of opaque ules in the urine, the shape of which is extremely irregular. These are, s, granules of hæmoglobine. Some of them are free in the urine, some ched to casts. Of the latter, we find hyaline and a few epithelial casts Sometimes masses of hæmoglobine assume the appearance of casts. The t may also contain a few cells of renal epithelium. The presence of this yaline casts indicates that the kidneys have been slightly irritated by the n of hæmoglobine. Hence, we sometimes observe a persistence of slight uria after the hæmoglobinuria has ceased. In mild attacks, also, there merely albuminuria, without hæmoglobinuria.

w we examine the blood during a paroxysm, we shall find that hæmoglo- is associated with hæmoglobinuria of the paroxysmal type, as well as t occasioned by the action of various poisons. Küssner obtained blood atient during a paroxysm, and found that its serum had a ruby-red color, tained hæmoglobine in solution. This proves that the destruction of blood- es takes place within the circulatory system. Indubitable tokens of this ive process are to be seen upon microscopic examination of the blood paroxysm, especially when the paroxysm has been produced artificially anner described below. The red blood-corpuscles have little tendency to leaux. They are pale, and many of them are irregular in shape (poikilo- Irregularly shaped flakes of hæmoglobine are also present, and often large of decolorized red blood-corpuscles are to be seen. To these latter Pon- given the name of "shadows." The exciting cause of the individual of paroxysmal hæmoglobinuria is, in most instances, catching cold. A n is usually due to being outdoors in a cold storm, and perhaps being wet . In summer the attacks cease completely in patients of this sort; still, nbach showed by experiment, an attack may even then be artificially ex- any time by exposing the patient's skin on purpose to a decided chill—e. g., ng the patient to take an ice-cold foot-bath, or the like. In order to show re is merely a local action of cold in these cases, Ehrlich and Boas (each dently) arranged the experiment in the following manner: They tied an igature firmly around the finger of a subject, and had him immerse it for a of an hour in ice-water. In every sample of blood taken from the finger e-described changes could be most distinctly demonstrated, while blood om other parts of the patient's body showed scarcely any abnormality.

The influence of cold is not the only exciting cause for such attacks, although it is the most frequent one. Paroxysms of hæmoglobinuria have been seen after great bodily exertion, particularly after a rather long walk; in women sometimes at the menstrual period; and also after mental excitement, and the like. No explanation has as yet been given why some few human beings have this remarkable sensitiveness of their red blood-corpuscles, while most persons undergo the same influences without similar consequences. In this regard should be noted the statement of Murri, that paroxysmal hæmoglobinuria is particularly apt to appear in persons who have a syphilitic taint.

Finally, with regard to the origin of the other symptoms of the hæmoglobinuric attack, such as fever, at first it was thought that there was a sort of uræmic condition, inasmuch as the urinary tubules are often so choked with granules of hæmoglobine that the excretion of urine might well be hindered. Probably, however, another circumstance is still more important—viz., the toxic influence of the fibrine ferment which is formed because of the hæmoglobinuria. The hæmoglobine, as it is set free, destroys numerous white blood-corpuscles, and in this way fibrine ferment is produced, as we have already mentioned (see page 725).

Another important point is that it is not the kidneys alone which serve for the reception of the constituents of the disintegrated and dissolved blood-corpuscles. Ponfick has been led by the result of certain experiments to believe that the spleen and liver are also affected. The spleen appropriates the undissolved remnants of the corpuscles, and, as a consequence, it may undergo considerable enlargement. The liver absorbs a large part of that portion of the hæmoglobine which has undergone solution, and converts it into bile. As a result there seems to be an increased secretion of bile. Jaundice is probably due to local biliary stasis and biliary absorption in the liver itself ("hæmo-hepatogenous jaundice" of Afanassiew). No proof has been furnished that a part of the hæmoglobine dissolved in the blood can be changed into bile pigment (true "hæmatogenous jaundice").

Prognosis.—When hæmoglobinuria is merely a symptom of other abnormal processes caused by poisoning or by some specific infection, the future of the patient depends entirely upon the severity of the primary disease. An attack of paroxysmal hæmoglobinuria would not seem to involve any direct danger to life. Recurrence of the paroxysms is always to be feared if the patient be exposed to those influences which produce it. There are no certain means of decreasing the patient's liability to attacks. In a few cases, however, where there had been syphilis, the paroxysms are said to have been permanently banished by mercurial inunctions. Likewise, if we suspect malarial influences, quinine should be tried.

No special treatment is required during the paroxysm itself. The patient must escape as soon as possible from the exciting cause; it is then advisable for him to lie quietly in bed, and to drink a large amount of fluid, so as to wash out the masses of hæmoglobin from the kidneys.

CHAPTER VI

SCURVY

(Scorbutus)

Prefatory Remarks.—Scurvy is one of a group of diseases which may be termed "hæmorrhagic." They all have one predominant symptom, namely, a decided hæmorrhagic diathesis, respectively associated in the different diseases with various other more or less pronounced disturbances. This tendency to spontaneous hæmorrhage is in many cases, particularly the milder ones, confined more or less exclusively to the skin, but in numerous other instances hæmorrhage also takes place into the underlying tissues, such as the muscles or joints, as well as into the mucous membranes.

The distinctions between these various diseases are founded upon the manner in which the hæmorrhages occur and the symptoms which attend them. We may mention scorbutus, purpura simplex, purpura hæmorrhagica, and peliosis. It should, however, be stated that, although it is possible to distinguish several varieties of disease, each one of which presents a tolerably characteristic picture, there are innumerable transitional forms. It may, indeed, be almost a matter of taste in any particular case what name shall be applied to it. The existence of so many intermediate forms renders it evident that the various members of this group of diseases are at least closely related if not actually identical. We shall even find, upon careful consideration, that certain other diseases not usually regarded as hæmorrhagic are nearly akin to the group now under consideration. We refer to certain skin diseases, which are characterized mainly by inflammatory and exudative lesions of the skin. Chief among these should be mentioned erythema exsudativum multiforme, which not very infrequently exhibits some tendency to hæmorrhages, and thus presents external appearances closely simulating the forms of purpura. In the last-mentioned forms of disease, however, there appears still another clinical relationship—viz., to the ordinary acute articular rheumatism (*q. v.*). All of the hæmorrhagic diseases are distinguished by a frequent involvement of the joints, while, on the other hand, acute articular rheumatism is sometimes associated with hæmorrhagic lesions in the skin.

In order to understand the underlying connection between these various disorders, a precise knowledge of their ætiology is requisite. Already considerable evidence has been gathered pointing to the importance of infectious influences in their production (*vide infra*), but no absolute proof has yet been obtained. Still, it must be said that various observations already made indicate that we are likely to find some ætiological relations between the hæmorrhagic and the septic diseases. In the meanwhile, we must be guided mainly by the purely clinical phenomena. These, again, indicate that sharp distinctions between the various hæmorrhagic diseases would be purely artificial. In this and the following chapters we shall discuss the two main types of hæmorrhagic disease.

Ætiology of Scurvy.—Scurvy is sometimes sporadic, and sometimes epidemic and endemic in its occurrence. There were formerly very extensive and fatal epidemics of the disease, at a time when the laws of health with regard to large aggregations of human beings were little regarded. The disease was prone to attack armies, or the inhabitants of besieged cities, or, especially, seamen. It was, and to a certain extent is still, one of the diseases most dreaded by the mariner. It has often swept away an entire ship's crew. To-day endemics of scurvy are by no means infrequent, although not so extensive as formerly. They are most apt to occur in prisons and similar institutions, and in barracks.

These facts, under the light of our present views in regard to such matters, would almost force us to seek some organic infectious poison as the origin of the disease. Formerly men were inclined to direct their sole attention to such circumstances as the character of the food, the dwelling, the climate, and similar conditions; nor can it indeed be denied that these hygienic factors do exert a decided influence upon the spread of the disease. It is, however, evident that they can not be its proper cause, for, beyond a doubt, scurvy may occur independently of any of the factors usually regarded as essential to its development. These causes must therefore be regarded as simply predisposing influences.

Great ætiological importance has long been ascribed to certain errors in diet.

These include the use of bad or insufficient food, the undue predominance certain kinds of food, and in particular of the salt meats so much employed o shipboard; or, again, the deficiency of certain varieties of food, in particular th lack of vegetable food, and still more of fresh vegetables. Much industry a acuteness have been expended in defending the theory that the lack of vegetab food is injurious because of the deficient supply of potassium salts under su circumstances (Garrod). Nevertheless, this view does not reach the heart of t matter, for in numerous epidemics of scurvy there has been no such lack of ve table nourishment; and in some instances the diet employed has contained unusual abundance of potassium compounds.

A like predisposing but not specific influence is exerted by the other factors to which ætiological importance has been assigned. They are indeed often present in epidemic as well as in sporadic cases, but, as previously stated, they may not exist at all. To this class belong damp and unfavorable quarters, cold, moisture, persistent heat, and excessive muscular exertion.

Age and sex exert no great influence upon the disease. Weakly persons seem to be somewhat more liable to be attacked than are the vigorous. The possibility of contagion has been maintained repeatedly, but contagion has not been proved to exist, and unprejudiced observation would incline one strongly to doubt its existence.

[It seems probable that the dietetic causes of scurvy lie rather in a want of variety in the food than in the absence of any one class or order of foods. Wales says (Pepper, System of Medicine): "No single natural order contains plants that supply all the elements essential to the nutrition of the body and the right composition of the blood. The graminaceous and leguminous articles of food, for instance, are numerous but not various; they all afford the same or analogous albuminous elements, which have about the same nutrient value as the corresponding substances in animal food, and hence health and vigor can not be sustained on a diet of flesh combined with wheat, rice, and oatmeal, or with beans and peas, or with all of them together. Outbreaks of scurvy have occurred on shipboard where the ration is made up principally of these articles—as in Anson's ship when supplied with an abundance of fresh animal, farinaceous, and leguminous foods. It is clear, therefore, that, in order to obtain a variety of materials required in nutrition, we must resort to several of the natural groups, those particularly which comprise the succulent vegetables and fruits."

It is certain that scurvy is a disease which we can produce artificially, and that it is preventable in the vast majority of cases. It is now as rare among seamen as it was formerly common—a change which is the result chiefly of care to vary the diet, especially on long voyages. The United States law requires that lime- or lemon-juice, sugar, and vinegar shall be carried by all sailing vessels bound on ocean voyages or engaged in the fisheries, specifies the circumstances and minimum doses under which the antiscorbutics are to be given, and provides penalties for violation or neglect.]

Clinical History.—The disease does not usually begin suddenly. There is a gradual onset, marked by certain constitutional symptoms. The chief of these are languor and debility, a sense of thoracic oppression, palpitation, and usually a "rheumatic, dragging pain" in the loins and extremities, especially in the lower extremities. The patient is obliged to take to his bed if the case be at all severe; he is very sensitive to cold, and often is drowsy and apathetic. These somewhat indefinite premonitory symptoms last for a few days or a week, when other and more characteristic phenomena appear.

Among these new appearances are spontaneous hæmorrhages, occurring chiefly in the lower extremities. There are cutaneous hæmorrhages, producing dark-red macules of varying size, most of them with a hair-follicle in their center, and

are almost invariably hæmorrhages into the deeper tissues also. The sub-
ous connective tissue and muscles, and sometimes the periosteum, are
d. These deeper extravasations are a peculiarity of scurvy. They can
mes be felt as hard, painful swellings in the parts affected, and are some-
discernible from the discoloration of the skin, which soon results from the
n and diffusion of the blood-pigment. The patches present a diffuse bluish
merging into greenish or yellowish at the periphery, and they are often
large. They have a precisely similar appearance to "black-and-blue" spots
ng from injury. Of course, the more abundant and the more superficial
travasation, the more extensive and darker is the macule. Similar appear-
may sometimes be observed in the upper extremities and trunk, mainly in
cases. The face and scalp rarely present ecchymoses.

metimes a hæmorrhage results in the necrosis and sloughing away of a por-
f the skin. The necrosis is succeeded by ulceration ("scorbutic ulcers").
unfavorable hygienic influences this process may assume a grave signifi-
It should also be stated that we may observe other cutaneous disturbances,
s erythema, wheals, vesicles (the contents of which may be tinged with
—"scorbutic pemphigus"), papules, and pustules. These eruptions are more
nt in some epidemics than in others; they may either be associated with or
e the cutaneous ecchymoses.

the ordinary sporadic cases of scurvy which occur among us, hæmorrhages
he mucous membrane, except of the gums, are very rarely seen. The same
of hæmorrhages from the stomach and other internal organs. In severe
during epidemics and under bad hygienic surroundings, it is otherwise;
rrhage may take place from the nose, stomach, intestines, bronchi, and kid-
and blood may be effused into the serous membranes.

xt in importance to hæmorrhage is another peculiar symptom, presented by
ucous membrane of the mouth, and particularly that of the gums. In order
ablish a diagnosis of scurvy in sporadic cases, we must demonstrate the
nce of these two main symptoms—namely, the hæmorrhage into the skin
scles, and the changes in the gums now to be described.

e scorbutic changes in the gums usually appear quite early in the course of
sease, being in many cases simultaneous with the hæmorrhages, although
nay either precede or follow the latter. The gums assume a bluish hue,
e swollen and spongy, are painful, and have a tendency to bleed. The
es are usually most pronounced in the salient parts of the gums between
eth. It is a remarkable fact that they are hardly visible at all at places
there are no teeth; and the gums of very young children and of aged
ts remain almost intact. In severe cases the gums are not only swollen but
ed; the change is at first a superficial one, but it may extend inward and
ce dirty-looking ulcers. Other parts of the mouth are liable to become
ed in the ulceration, producing a diffuse ulcerative stomatitis, and giving
eath a most offensive odor.

rtain other local and constitutional phenomena are not infrequent, though
aracteristic than the hæmorrhages and the alterations in the gums. Chief
the general disturbances is scorbutic anæmia. This is often referable in
o the unfavorable hygienic influences surrounding the patient, but the dis-
tself seems to impair the general nutrition. The patient looks pale, has a
in, and loses flesh rapidly. The temperature is often normal. Sometimes
may be an occasional rise of temperature, either in the beginning or in the
r course of the disease. If complications occur, they are not infrequently
panied by considerable fever.

nong more localized symptoms should be mentioned the premonitory sore
which sometimes occurs. It is usually of the ordinary catarrhal variety,

but it may assume a hæmorrhagic character. Bronchitis also may occur. Lobular pneumonia and genuine lobar pneumonia have been repeatedly seen in severe cases. Pleurisy, pericarditis, and inflammations of other serous membranes occasionally complicate the disease. They may likewise display a hæmorrhagic tendency in the exudations to which they give rise. Disturbances in the joints are sometimes seen, and are characterized by an effusion of liquid into the articular cavities, which effusion may be either serous or hæmorrhagic. This is a favorable opportunity to call attention to a peculiarity common to all the hæmorrhagic diseases and allied affections (*vide supra*)—they are apt to be associated with articular swelling.

The pulse may be somewhat accelerated, or may be slower than normal. It is usually small and compressible. Endocarditis may occur, but it is very exceptional. The blood does not present any constant and characteristic alterations in scurvy. The spleen may be decidedly enlarged, particularly in severe cases. Albuminuria has been repeatedly observed, but it is almost wholly confined to severe cases, in which, indeed, a typical acute nephritis may be developed.

Course of the Disease. Varieties of Scurvy. Prognosis.—The sporadic cases usually met with in this region almost invariably pursue a favorable course. The symptoms are confined mainly to constitutional disturbance, ecchymoses in the lower extremities, and the affection of the gums, the grave complications above mentioned being rarely met with. The average duration of the disease is, nevertheless, some weeks. Recovery is deferred in proportion as the hygienic surroundings are bad, but even then the termination is almost sure to be favorable. Scurvy has, however, a noteworthy tendency to relapse, so that the course of the disease may be very much prolonged. Even when the illness has terminated in recovery, there remains behind a certain liability to fresh attacks of scurvy, just as acute articular rheumatism is apt to recur. We are acquainted with a patient who has been severely ill with scurvy some five or six times within the last few years, and this without any demonstrable cause.

The prognosis in grave cases, occurring under unfavorable hygienic influences, and aggravated by the lack of proper food and attention, is far otherwise. Here death is not infrequent, sometimes as a result of progressive cachexia, sometimes because of pneumonia, pericarditis, cerebral hæmorrhage, or a similar intercurrent disease.

Anomalous or rudimentary cases of scorbutus may occur. They are most apt to be seen when the disease is epidemic or endemic. As a rule, the symptoms are mild. We find, for example, a scorbutic gingivitis and stomatitis without hæmorrhage, or, on the other hand, hæmorrhages into the skin and mucous membranes unattended by alteration in the gums. There have even been cases reported of simple scorbutic anæmia without any local symptoms.

[The experience of army surgeons during our civil war deserves mention in this connection. Hammond, Woodward, and others state that many cases classed in the sick reports as "general debility" were cases of incipient or imperfectly developed scurvy, hæmorrhage from mucous membranes or into the skin being absent.]

Diagnosis.—The diagnosis of scorbutus is almost self-evident when the two chief symptoms of hæmorrhage and alteration in the gums are both present. If, however, one or the other of these symptoms is suppressed or imperfectly developed, it may be difficult to determine what disease we have before us, or to exclude ordinary stomatitis, rheumatic peliosis, and similar diseases. One characteristic sign should be emphasized again in this connection. The hæmorrhage of scurvy is scarcely ever confined to the skin, but almost invariably there are large extravasations deeper in, in the muscles and fasciæ. These occasion, secondarily, the characteristic bruise-like bluish-yellow spots seen through the skin.

Treatment.—The essential requisites in the treatment of scurvy are proper hygiene and diet. Fresh air, suitable nourishment, and good nursing, if promptly supplied, are usually of themselves sufficient to induce recovery, while the physician possesses no remedies which compensate for their absence.

The belief that a main cause of scurvy lies in a deficiency of fresh vegetables has given rise to a practice, still in vogue, of prescribing a great abundance of fresh vegetables, such as lettuce, spinach, and sorrel, fruit, lemonade, and other drinks prepared from fruit syrups. There is no reason to deviate from a course to which experience has given its sanction, although we have repeatedly had opportunity to see that the administration of fresh vegetables is by no means essential to rapid recovery. Patients supplied with any other proper nourishment thrive equally well. Certain varieties of plants have attained a special reputation as "antiscorbutics," such as the spoonwort (*Cochlearia officinalis*), so frequently mentioned in accounts of early polar expeditions. None of these plants, however, possess the specific properties assigned to them. The administration of vegetable acids and the salts of potassium (bitartrate and nitrate of potassium), in a chemically pure form, has also been repeatedly tried, but it has not gained popularity.

The drugs most employed are the bitters and tonics. They have no specific value, but are perhaps as good remedies to prescribe as any. We may give a decoction of cinchona, to which may be added a small amount of dilute sulphuric acid and syrup, or some preparation of gentian or a similar bitter. It was once believed that the internal administration of the mineral acids exerted a specially favorable influence upon the hæmorrhagic diathesis; but this is very doubtful.

Certain symptoms may demand attention; in particular, the affection of the mouth and gums. It is of great importance to cleanse the mouth frequently with disinfectants and mild astringents, such as chlorate of potassium or sage tea. It is also advisable to paint the inflamed and spongy gums at short intervals with tincture of myrrh or tincture of rhatany. The absorption of the ecchymoses in the lower extremities will be promoted by cautious massage. Inunctions of linimentum chloroformi and the like give relief from the pain caused by the extravasations into the deeper tissues. In severe cases stimulants are often demanded, such as camphor, ether, and alcohol. Such complications as appear may also demand special treatment.

Convalescence is promoted by continued attention to diet, bathing, and the administration of iron and quinine.

APPENDIX

BARLOW'S DISEASE

In this connection we desire to mention briefly a peculiar symptom-complex which has been associated with scurvy, in regard to the real nature of which we do not yet possess any definite knowledge. The disease is, therefore, usually named after the person who discovered it (1883), as Barlow's disease.

The disease has been seen thus far in children alone, and most often between the ages of six and eighteen months; almost all the children had previously shown definite signs of rickets. There is little, if any, fever, but an extremely painful swelling of the bones, particularly of the epiphyses of the long bones, though the lower jaw, cranium, and other parts may be involved. The epiphyses may become loosened, but the joints themselves are not affected. Upon careful examination it is found that the swelling is due to subperiosteal hæmorrhages. In association with these there may be other indications of a hæmorrhagic diathesis, such as hæmorrhage into the skin, and into the serous membranes, and hæmaturia. In mild cases the hæmorrhages along the bones are absorbed and do not recur. In severe cases, however, the hæmorrhages are constantly repeated, and the child may finally die of exhaustion.

The prognosis is consequently to be expressed with some reserve. Treatment consists chiefly in suitable regulation of the diet. If the hæmorrhages are quite large it is advantageous to make a surgical incision, and allow the blood to escape.

CHAPTER VII

PURPURA. MORBUS MACULOSUS WERLHOFII. PELIOSIS

As already stated in the preceding chapter, the various "hæmorrhagic diseases" are so intimately related to one another that it is quite impossible to make a rigid categorical division of them. The numerous names which have been introduced into the literature of this subject certainly contribute more to obscure than to elucidate the attendant phenomena.

From a clinical standpoint this fact is the all-important one—namely, that there are cases in which the foremost symptom is the spontaneous occurrence of hæmorrhage. There are cutaneous ecchymoses, and there may be at the same time hæmorrhages in the internal organs and into the mucous membranes. In the milder cases of this sort these hæmorrhages constitute almost the only symptom of disease; but they may be associated with considerable general disturbance, indicated by fever and weakness, or with certain local complications. The true cause of these diseases has not yet been discovered. There is seldom any evidence of an exciting cause, and the disorder may attack either the well-nourished or the poorly nourished, the old or the young, men or women. There is, however, an indisputable relationship between these diseases and certain others—namely, scurvy, erythema exsudativum, and perhaps acute rheumatism and endocarditis. This similarity indicates that the process is of an infectious, or toxic, character. Such an assumption promotes greatly a proper understanding of the phenomena under consideration. It is also said that subcutaneous injections of blood from patients with morbus maculosus may cause a like affection in rabbits (Petrone and others). In some few cases the weight of evidence would seem, however, to point to an antecedent impairment of nutrition in the walls of the blood-vessels. A good example of this is seen where cutaneous ecchymoses occur in old and marantic individuals (*peliosis senilis*). There is some doubt whether these exceptional cases belong with the others.

The mildest forms of the diseases under discussion are termed **purpura**. The hæmorrhages are seen mainly in the skin of the lower extremities, and are apt to take place in the follicles. There may also be ecchymoses upon the trunk and upper extremities, but the mucous membranes and the deeper tissues remain intact. A means of distinguishing purpura from scorbutus lies in the fact that in purpura there are no hæmorrhages into the muscles and no lesions of the gums, although it should be confessed that transitional forms between the two occur. The disorder is called *purpura simplex* if the cutaneous ecchymoses constitute the only symptom, or, at any rate, the only important one. These cases almost invariably terminate in recovery, and are over at the end of ten days or three weeks. Sometimes elevations of the skin are formed resembling wheals, and hæmorrhages take place here and there into them. This subvariety has been called by some *purpura urticans*. It forms a connecting link between purpura simplex and those cases of erythema exsudativum which are associated with hæmorrhage. Further particulars may be found in special works upon dermatology.

Quite often the hæmorrhages are attended by "dragging rheumatic pains"; such cases are termed **purpura rheumatica** or **rheumatic peliosis** (Schönlein). There may also be constitutional disturbance, slight fever, anorexia, and indisposition to either bodily or mental exertion. There may sometimes be actual arthritis, with an inflammatory effusion into the joints. The knee and other joints of the lower extremities are most apt to suffer in this way. The gums are usually normal; nor is there, as a rule, hæmorrhage into the mucous membranes or the viscera. These cases may last but two or three weeks. Often, however, they are more tedious, being marked by the recurrence of the ecchymoses and articular pain. Most of them get well at last.

No sharp dividing line can be drawn between the forms of purpura thus far described and certain graver cases. These latter are most of them grouped under the name of *purpura hæmorrhagica*, or its preferable because more distinctive synonym—every purpura being hæmorrhagic—**morbus maculosus Werlhofii.** The cutaneous ecchymoses in this class of cases are usually extensive; and, furthermore, we have hæmorrhages into the mucous membranes of the nose, mouth, soft palate, stomach, and intestinal canal, as well as into internal organs (the brain and kidneys), and also into the serous membranes. The constitutional disturbance is apt to be severe. The condition may be distinctly "typhoidal." Fever may be entirely absent, even in grave cases, although sometimes there is a considerable rise in temperature.

There are usually no local symptoms beyond those already mentioned. In typical cases the gums remain intact. Swelling of one or more joints has been repeatedly observed, as have also endocarditis and acute hæmorrhagic nephritis. If marked cerebral symptoms are developed, suggesting an apoplectic shock, we may surmise that a cerebral hæmorrhage has taken place. It should also be stated that marked gastro-intestinal disturbance may occur. Cases of this sort have been observed by Henoch in children. They may also occur in adults. In rare instances there may be intestinal ulceration, with perforation and consequent peritonitis. The spleen may undergo acute enlargement.

All the symptoms which have been enumerated render distinct the similarity of the clinical picture, in severe cases of morbus maculosus Werlhofii, to that of severe septic infection. In fact, it is not impossible that continued investigation will deprive morbus maculosus of its individuality as a specific form of disease, and show that it is merely a special form of profound sepsis.

The prognosis in purpura hæmorrhagica should always be a guarded one; the patient is in danger both from the general depression and anæmia, and from certain special lesions. Even a severe case may, however, recover. The disease sometimes proves very tedious; it may occupy several months.

Treatment.—The general regimen to be prescribed is similar to that directed in scurvy. The physician must strive to support his patient's strength by proper nourishment. A great many internal remedies have been recommended, most of them on purely theoretic grounds. It is difficult to say whether they actually exert a favorable influence upon the course of the disease. The following drugs are chiefly employed: Ergotine, perchloride of iron, dilute sulphuric acid, and cinchona. If there be swelling of the joints or endocarditis, we should advise a trial of salicylic acid or antipyrine. Such special symptoms as demand attention, particularly heart-failure, should be treated according to general principles.

CHAPTER VIII

HÆMOPHILIA

Definition and Ætiology.—Hæmophilia is the term used to denote a pecu constitutional anomaly, exhibited in a remarkable tendency to spontaneous traumatic hæmorrhage. The condition is probably in every instance congeni and is usually hereditary; the existence of families of "bleeders" has long b known. Generation after generation displays frequent cases of hæmophilia, b among the direct descendants and the lateral branches. Bleeders are very ap have a numerous progeny. Not all of the children, however, fall victims to th disease. Grandidier has pointed out two facts which are of interest in thi connection, as they might aid in deciding as to the marriageability of certain persons.

If a man belonging to a family of bleeders marries a healthy woman, neither a bleeder herself nor inheriting a predisposition to hæmophilia, his children are almost certain to be healthy, even though the father himself be a bleeder. On the other hand, a woman belonging to a family of bleeders, even though she herself be healthy, will almost always have some children who are subject to hæmophilia. In other words, hereditary predisposition is transmitted much oftener through the female than through the male members of the family. Hæmophilia itself is, on the contrary, much more frequent in the male sex than in the female; at least this is true of the pronounced cases. Hössli, who has lately published a very careful family tree of bleeders of Tenna (Canton Graubünden), comes to the following conclusions: "The inheritance of hæmophilia is often from the father, through the daughter, to the grandson, also from the mother, through the daughter, to the grandson, and most rarely directly from the father to the son." It is doubtful whether race and place of residence are of ætiological importance. So far as is known, hæmophilia appears to occur in all countries, although it is fortunately rare everywhere.

[A similar transmission through the females, who themselves usually escape, is seen in color-blindness and in pseudo-hypertrophic paralysis.]

The real causes of hæmophilia are entirely unknown to us. We can make one or two steps toward finding the source of the hæmorrhage, but we are unable to proceed further. It would seem that the bleeding must depend, in the first place, upon an abnormal delicacy of the walls of the vessels predisposing them to rupture, and, secondly, upon deficient coagulability of the blood. This latter abnormality is evident from the fact that in hæmophilia it is difficult to check even the most insignificant hæmorrhage. Thus far all attempts to discover any anatomical or chemical explanation of this imperfect coagulability have been vain. It has not been possible to detect any variation in the saline constituents of the blood, or in the amount of albuminoids, such as fibrinogen, that it contains, or in its morphological elements. And likewise no anatomical change in the vascular walls or the heart has yet been reported which throws light upon the character of the disease. Various authorities have laid stress upon the small diameter of the arteries and the thinness of the intima, but these conditions may occur independently of hæmophilia. Fatty degeneration of the intima is, to be sure, often found in connection with this disease, but it is doubtless rather a result of the coincident anæmia than the cause of the hæmophilia. The observations with regard to the heart are very contradictory; sometimes it is found to be very small, sometimes of normal size, and again actually hypertrophied.

The subjects of hæmophilia do not present any distinctive constitutional peculiarities. It has been stated that they are very apt to be blondes with a delicate white skin, and superficial and abnormally distended cutaneous veins; but the exceptions to this rule are not a few.

Clinical History.—Hæmophilia does not display equal malignity in all cases. If we have opportunity to obtain thorough information with regard to families of bleeders, we shall find that quite often rudimentary varieties occur side by side with typical and severe cases. There is, to be sure, a striking tendency to hæmorrhage even in them; but the hæmorrhage never assumes threatening proportions. By perseverance and industry it is possible to collect an almost unbroken series of cases, varying in degree from extreme mildness to extreme severity. The following sketch applies mainly to typical and severe cases.

That hæmophilia is a hereditary constitutional disease is shown by the fact that it sometimes appears in earliest infancy. Many cases of umbilical hæmorrhage in the new-born are referable to hæmophilia. Of course this does not apply to all cases. In Jewish children the disease may betray itself for the first time when the rite of circumcision is performed. In many cases the disease is not recognized at so early a period; but this is no proof that the disease is not already developed, inasmuch as the young child is not much exposed to traumatism and other causes which naturally occasion hæmorrhage.

The most striking symptom in a fully developed case of hæmophilia is the occurrence of severe hæmorrhage as a result of the most insignificant causes. A slight blow produces a " black-and-blue spot " such as is ordinarily seen only after a very violent injury. The prick of a pin, a slight cut on the finger, or the extraction of a tooth, may give rise in hæmophilia to an obstinate and alarming hæmorrhage. Epistaxis may be caused by blowing the nose, hæmorrhage from the gums by brushing the teeth, and so on. Whether there is ever a perfectly spontaneous hæmorrhage is uncertain. It is true that in severe cases hæmorrhages take place independently of any visible cause. This may be seen in the skin and mucous membranes (nose and gums); and in rare instances we may even have free hæmorrhage from the stomach, intestines, or urinary passages. Yet it may be doubted whether these occurrences are not the result of comparatively insignificant mechanical injuries which escape our notice. At any rate, we scarcely ever find hæmorrhage taking place into the parenchyma of the viscera, in places where injury from external sources is entirely out of the question. This fact constitutes an important point of distinction between hæmophilia and the acquired hæmorrhagic diathesis.

The second important symptom of hæmophilia has been already referred to: it is extremely difficult, and may even be impossible, to check by artificial means any free hæmorrhage which may occur. It is this which makes the disease so dangerous, and prevents most patients from reaching old age. It has frequently happened that an apparently trifling wound of the skin, some insignificant operation, or a leech-bite, or in women childbirth, has started up a hæmorrhage, which eventually became fatal. In other cases the hæmorrhage is finally checked, but not until it has caused profound anæmia. Bleeders are apt to recover with remarkable rapidity from the effects of excessive hæmorrhage; yet continually repeated hæmorrhages may lead to a persistent and profound anæmia, attended by all the symptoms described in the preceding chapters.

We see, therefore, that the general condition in hæmophilia varies with the severity of the individual case, and with the more or less fortuitous circumstances which develop its dormant characteristics. If no special accident occurs, the patient may maintain the appearance of perfect health for years. In the worst cases, however, such a state is very temporary, if it exists at all, because the hæmorrhages can be so easily excited. As a consequence, the skin almost always presents a greater or less number of ecchymoses, while hæmorrhages from the internal organs contribute from time to time to the general debility and anæmia.

Certain complications may occur in hæmophilia, but they are little characteristic. There is a noticeable tendency to "rheumatic" inflammation of the muscles and swelling of the joints, wherein is seen a striking analogy to the "hæmorrhagic diseases." Often there is an actual effusion of blood into the joints. This may cause considerable functional disturbance of the joints, and eventuate in anchylosis. Various writers have also called attention to the comparative frequency of neuralgia, especially in the trigeminus.

Prognosis.—In only too many instances the victims of hæmophilia die in childhood; in other cases the patient attains an advanced age. A fact of great practical importance is that often, although not invariably, hæmophilia grows gradually milder with advancing years. If, therefore, the patient have survived the period of adolescence, we may believe that his prospects are gradually improving. The prognosis of hæmophilia is obvious. The amount of danger at any given time depends upon the severity of the hæmorrhage and the consequent anæmia. The comparative severity of the case must be judged from its previous history; as has just been said, the prognosis grows more favorable as the patient grows older.

Treatment.—Prophylaxis assumes a very important place in the treatment of hæmophilia. First, children who inherit a tendency to the disease, or who have given evidence of its existence, should be treated with a view to improve their general constitutional condition, so as to check the development of the disease as far as possible. The means to this end need not be described at length. They comprise good nourishment, fresh air, cautious endeavors to harden the system, baths, and tonics. Secondly, when hæmophilia already exists, the patient should be guarded as much as possible from any mechanical injury, such as might excite hæmorrhage. Thus caution is demanded in performing vaccination and other apparently trifling operations.

As regards direct treatment of the disease, no effectual remedy is known. The general tonic treatment already referred to should not be neglected; but the administration of ergotine, acetate of lead, and similar drugs is indicated, if at all, only when hæmorrhage is actually taking place, and even then it is very apt to fail. The only way to stop the hæmorrhage is by surgical methods, and these need not be described here. They do not differ essentially from those employed when hæmorrhage occurs independently of hæmophilia. If mechanical efforts to check the bleeding fail, we can expect nothing from the remedies above mentioned, nor from sulphate of sodium and the other laxatives which have been recommended. For the symptomatic treatment of the anæmia and of its results, we may refer to the first chapter of this section.

CHAPTER IX

DIABETES MELLITUS

Definition and Ætiology.—Under normal circumstances the blood always contains a slight amount of sugar; but this ingredient does not usually pass over in appreciable quantities into the urinary excretion. If, however, the amount of sugar in the blood exceeds certain limits—that is, if there exists an abnormal "glycæmia"—then sugar is excreted in the urine, and we have glycosuria. This is seen as a more or less temporary phenomenon under the most varied conditions. The amount of sugar in the urine is usually comparatively slight, and it soon disappears again. Its presence does not imply any persistent abnormal condition.

This phenomenon has been termed glycosuria or melituria, in contrast with the peculiar disease which has for its chief symptom a persistence of sugar in the urine, and has therefore received the name of diabetes mellitus. It is not necessary in this connection to enumerate all the causes of glycosuria, i. e., of a temporary appearance of sugar in the urine. It may be briefly mentioned, however, that glycosuria is observed even in healthy persons after excessive indulgence in sugar. If a healthy person takes at one time about seven ounces (grammes 200) or more of grape-sugar dissolved in water, there will probably be after an hour or two a demonstrable, although not a very great, amount of sugar in the urine. This is the so-called alimentary glycosuria. In this case the blood is so overburdened with sugar that the sugar can not be oxygenated fast enough. A portion of the sugar, therefore, escapes change and is excreted with the urine. If the same amount of sugar is given in divided doses at intervals, there will be no glycosuria. If glycosuria appears upon the administration of small amounts of sugar (two to three ounces, grammes 50–100) we are justified in speaking of a morbid alimentary glycosuria, the power of the body to assimilate sugar being subnormal. An abnormal glycosuria of this sort is observed, for example, not infrequently in obese beer-drinkers, and in patients with severe traumatic neuroses, or with exophthalmic goître. Temporary glycosuria is also observed not infrequently in certain kinds of poisoning, particularly after severe poisoning with carbonic oxide, morphine, hydrocyanic acid, mercury, nitrite of amyl, and curare. Von Mehring has lately discovered in phloridzine, a glucoside in the bark of the roots of apple and cherry trees, a substance which causes a very large amount of sugar in the urine if given to dogs, rabbits, etc. A marked glycosuria may be produced in man also by giving phloridzine, without other disturbances of the general condition. It is not improbable that the phloridzine-glycosuria is different from the other toxic glycosurias, in that it depends upon a change in the renal epithelium, as a consequence of which the cells become permeable to the sugar which is circulating in the blood. Temporary glycosuria has also been seen occasionally in connection with the acute infectious diseases—for example, malignant pustule, cholera, typhus or typhoid fever, scarlet fever, diphtheria, and malarial poisoning. A far more frequent cause is disturbance of the nervous system. Thus, glycosuria may result from severe concussion of the brain, fracture of the skull, cerebral hæmorrhage, cerebro-spinal meningitis, and after epileptic fits. It is especially apt to occur when there is disease of the medulla; and we need hardly point out how close is the connection between this clinical fact and the famous discovery of Claude Bernard, who found by experiment that, when certain injuries are inflicted upon the floor of the fourth ventricle, glycosuria inevitably follows. This glycosuria is explained by assuming that, as a result of nervous influences, the liver discharges its supply of glycogen into the blood in the form of grape sugar. The blood is overwhelmed with the sugar, and a portion escapes into the urine.

In some other oganic diseases, also, there may be a temporary and symptomatic glycosuria. Formerly, special attention in this regard was directed to hepatic diseases, but it is to be noted that, as a rule, even when there is extensive disease of the liver—for example, in hepatic cirrhosis—no glycosuria is observed, even if the patient is given large amounts of sugar in his dietary. On the other hand, the combination of persistent diabetes mellitus and hepatic cirrhosis is not very exceptional. As we shall point out again later on, we regard both diseases in these cases as co-ordinate effects of the same cause, chronic alcoholism. We shall likewise refer again to the relation between diabetes and renal disease. Especially great interest attaches to the question of the relation between glycosuria and diseases of the pancreas, since Minkowski and Von Mehring made the important discovery that we can excite severe diabetes in dogs by extirpation of the pancreas. The ligation of the excretory duct of the gland, or the diversion of the pancreatic juice into a fistula leading to the surface of the body, does not excite diabetes, nor does the diabetes occur if the pancreas is partially and not wholly extirpated. Pancreatic diabetes, therefore, implies a disturbance of a special function of the pancreas, of which we have as yet no intimate knowledge, but the abolition of which prevents the complete splitting up or oxidation of sugar in the normal organism. Diseases of the pancreas in man (carcinoma; pancreatic calculi, with secondary atrophy of the gland) occasion glycosuria sometimes, but by no means invariably. The atrophy of the pancreas observed in many cases of genuine diabetes will be discussed later on. We should mention in this connection, also, the glycosuria which sometimes appears in association with exophthalmic goître, and finally the interesting and frequent combination of acromegaly (*q. v.*), and the persistent excretion of sugar in the urine.

In contrast with the temporary phenomenon of glycosuria, we apply the name diabetes mellitus to a chronic morbid state, in which a permanent disturbance in the metabolism of the carbohydrates is indicated by a persistent excretion of sugar in the urine, and in which this anomaly of metabolism is the main symptom of the disease. If the excretion of sugar in this disease is not absolutely persistent, at least it is sure to occur under certain conditions of nutrition. It is doubtful whether diabetes mellitus is to be regarded from an ætiological standpoint as a single, distinct disease. With regard to most of the "pure" cases, particularly the severe form of diabetes which usually occurs in youthful individuals without any demonstrable cause and also without any primary organic lesion, there is great probability in the assumption that the morbid process is a single and definite one, although we are as yet entirely unacquainted with its real nature. On the other hand, with regard to the so-called milder forms of diabetes mellitus, particularly those which occur in middle and advanced life, there are probably various causes which may excite the disease. Still, it must be emphatically stated that no sharp dividing line can be drawn between the "severe" and the "milder" forms of diabetes. The physician is, therefore, bound in every individual case to search for certain causative factors and harmful influences, which sometimes, at least, play a part in the development of the disease. These causative factors are the following: First, heredity: diabetes has been repeatedly observed to occur in several generations of the same family, or in several brothers and sisters. It is noteworthy that the disease may also occur in families where there is a hereditary predisposition to nervous diseases. For example, we observed once in the same patient the remarkable combination of diabetes and progressive muscular atrophy of spinal origin. Second, improper mode of life: by this is meant chiefly unsuitable diet, especially the persistent overindulgence in starchy foods and sugar; sedentary habits are also considered harmful, especially if associated with overeating. This is said to be the reason why diabetes is more frequent among the wealthy classes, and why it is comparatively common in corpulent persons (*vide infra*). Our personal observations lead us to ascribe to long-continued and excessive beer-drinking (four or five litres and more per day) an influence upon the development of diabetes. In such cases it may be that we have a combined action of the excessive ingestion of carbohydrates in a state of solution, and of chronic alcoholism. It can, in our opinion, be no mere coincidence that diabetes so often attacks brewers, inn-keepers, and the like, in Bavaria. Furthermore, the complication of diabetes with other results of chronic alcoholism, such as hepatic cirrhosis, chronic nephritis, and alcoholic neuritis, is an argument for the correctness of our supposition. The "diabetes of obesity" has been long recognized, but in many cases it is really a "beer diabetes." Third, taking cold and getting wet are said, in occasional rare instances, to determine the appearance of diabetes. The author has never seen a clear case of this

Fourth, emotional disturbances, excessive mental exertion, anxiety, and ion, are sometimes thought to occasion the disease; and apparently with reason. Fifth, it is very remarkable that sometimes the same factors which ave already seen to be possible causes of temporary glycosuria, may also occa- a chronic diabetes mellitus; thus, cases of diabetes have been known to w injuries to the head, and such acute infectious diseases as typhus, typhoid, scarlet fevers, cholera, and malarial poisoning. Sixth, a few authors (Schmitz others) have hinted at the possibility of the disease being transferred by con- on—for example, in the case of married people. This interpretation of the tered instances which have been reported is, however, at least open to doubt. nth, a very important matter is the occurrence of diabetes in association , or as a sequel to, certain other constitutional diseases. In this category be- the above-mentioned diabetes of the obese; also the simultaneous appearance iabetes and genuine gout (*q. v.*); and finally, the extremely important com- tion of diabetes with general arterio-sclerosis. That all these combinations do uently occur can not be doubted, but it is very difficult to explain the connec- between them; often both conditions may be the simultaneous effect of the cause—for instance, congenital anomalies of constitution, unhygienic modes fe, and alcoholism.

f we survey all the facts thus far known with regard to the ætiology of etes, we feel obliged to conclude just as in almost all other constitutional ases, that in regard to the development of diabetes we must distinguish genous and exogenous factors. The endogenous relate to the constitutional encies which the patient possesses, perhaps by inheritance; and the exoge- to harmful influences from without, such as trauma, infection, and intoxi- on. The severe cases of diabetes which seem to occur absolutely spontane- y in young persons, are mainly of an endogenous character; while in the ler forms of later years the above-mentioned exogenous influences often play important part, although even in them the endogenous factor can not be rely disregarded, for the same noxious influence does not produce, by any ns, the same effect upon all persons.

Diabetes occurs everywhere, but certain countries and districts seem to be icularly liable to it—for example, India, Ceylon, and Italy. In Germany, rtemberg and Thüringen are said to present the largest relative number of s. Jews are very liable to the disease. Most cases occur in patients between ty-five and fifty years of age. Next in liability to the disease come younger viduals, under thirty-five and over twenty years old. After the fiftieth year etes is not exceptional, but, in the other direction, children under ten are seldom attacked by it, although they are not absolutely exempt. With rd to sex, males are much more often attacked than females.

Clinical History.—With few exceptions, the symptoms of diabetes mellitus e on slowly and gradually. Sometimes the symptoms are merely general and finite, such as languor, emaciation, weakness, and deficient endurance. etimes we have mild nervous disturbances, including headache, mental de- sion, wakefulness, and neuralgia, and in still other cases gastro-intestinal ptoms, including nausea, eructations, and irregularity of the bowels. At last patient's attention is called to the altered character of the urine, and particu- y to its increased amount. He also notices that he is very thirsty, and that, pite of his enormous appetite, he is constantly growing weaker. Sometimes e different symptoms first arouse suspicion of the existence of diabetes; these be mentioned later. In order to make a diagnosis of diabetes mellitus, a wledge of the abnormal character of the urine is indispensable, so that it is er to begin our consideration of the symptoms of diabetes with a description iabetic urine.

1. Character of the Urine. Demonstration of Sugar.—Usually the first point that attracts attention is the increased amount of urine. There are often a hundred to a hundred and fifty ounces (three to five quarts, 3,000–5,000 cubic centimetres) excreted in twenty-four hours, and sometimes the amount equals or exceeds ten or twelve quarts (8,000–12,000 cubic centimetres). Under suitable treatment and with proper diet the amount may, of course, be much smaller. In not very exceptional cases, mostly of the milder varieties (*vide infra*), there is slight, if any, increase in the amount of urine (*diabetes decipiens*); and in severe cases with initial polyuria we may often observe that during intercurrent diseases, and likewise in the last days before the fatal termination of the disease, the daily amount of urine diminishes.

In color the urine is light yellow, corresponding to its amount. It often has something of a greenish hue, but a small quantity of it may seem almost as colorless as water. Ordinarily the urine is clear and without sediment; but after it has stood for some time it may become cloudy, usually as a result of the abundant development of fermentation spores.

The odor may be somewhat aromatic, suggesting acetone (*vide infra*). Its taste is said sometimes to be distinctly sweetish. The reaction is acid, and the acidity of the urine may increase on standing, because of the alcoholic and lactic-acid fermentation processes which the sugar undergoes.

The specific gravity is almost invariably greatly increased, as a result of the large amount of sugar. If a pale urine is found to have a specific gravity of more than 1025, we may feel almost certain that it contains sugar. Specimens often have a specific gravity of 1030–1045, and even higher. In exceptional instances the specific gravity may fall below 1020. A specific gravity below 1020 is exceptional, but one should never be kept from making an examination for sugar because of the low specific gravity of the specimen. We have ourselves demonstrated sugar by means of the fermentation test in urine which had a specific gravity of 1007.

The diagnosis requires that sugar be detected in the urine. The sugar found in both the blood and the urine of diabetic patients is grape-sugar (glucose, dextrose). The amount of sugar secreted in twenty-four hours often reaches half a pound to a pound (grammes 200–500). Of course the amount varies greatly according to the diet, mode of life, and treatment of the patient. The greatest amount ever known to be produced in twenty-four hours was more than two and one fifth pounds (grammes 1,000). The percentage of sugar in the urine varies between 0.5–1 per cent. at the lowest extreme, and 8–10 per cent. as the maximum; usually it is about 2–4 per cent. It is noteworthy that in the last weeks, or just before death, the sugar in the urine may become greatly diminished in amount, or may absolutely disappear.

The most important tests for sugar in the urine are: First, Trommer's test: To urine in a test-tube sufficient potassic or sodic hydrate is added (say one part to three or four of urine) to make the reaction strongly alkaline; then a solution of sulphate of copper (about one part of the salt to ten of water) is added, drop by drop. If the urine contain sugar, the hydrated cupric oxide, which is at first formed, is largely dissolved, and usually the fluid assumes a beautiful deep-blue color. We ought, properly, to go on adding the sulphate of copper until the hydrated cupric oxide ceases to be dissolved. The urine is then heated, whereupon the cupric oxide is reduced and a yellow, or reddish-yellow, precipitate of cuprous oxide, or hydrated cuprous oxide, is formed. The application of heat should not be continued long after the precipitation begins to take place, lest the test be obscured; the reduction will go on even without heat. If the urine contains more than 0.5–1 per cent., this test is perfectly reliable. If the reaction be a doubtful one—that is, if there be no precipitate of cuprous oxide, although the urine becomes yellow; or if cuprous oxide be finally precipitated when the urine cools—we should be cautious in making a diagnosis, as the urine may contain substances other than sugar capable of reducing the copper, such as uric acid, kreatinine, mucine, etc. Second, the bismuth test (Böttger's): Sodic hydrate or sodic carbonate is added to the urine, and then a small pinch of subnitrate of bismuth. Upon boiling, the urine, if it contains sugar, quickly assumes a perfectly black color, the oxide being reduced to the metallic state. It is much more convenient, and consequently universally customary at present, to make the bismuth test by adding to the urine about one tenth of its volume of the following solution (Nylander): two parts subnitrate of bismuth, four parts Rochelle salts, one hundred parts of an eight-per-cent. solution of sodic hydrate. After one or two minutes' boiling the fluid turns black. Errors with this test are most apt to occur if the patient has taken certain drugs, such as antipyrine, salicylic acid, or rhubarb. Third, the potassium test (Moore's): Potassic hydrate is added to the urine in the test-tube and the uppermost layer cautiously heated; if it contains sugar, the urine quickly assumes a deep-brown color, as a result of the action of the potassium on the sugar; and this upper dark-colored layer contrasts strongly with the clear urine below.

[Fehling's test is justly a favorite in this country, and has the advantage of being applicable to the quantitative as well as to the qualitative analysis. The difficulty of its not keeping well can be met by having separate bottles for the copper and tartrate solutions, and making the mixture at the time of using the test. The Fehling's-test pellets put up by chemists are convenient for the qualitative analysis, but, on the whole, they are inferior to the solution.]

If the above-described tests leave us still in doubt, there can be only a small amount of sugar, if any, present. We may, however, attain certainty by employing the fermentation test (which causes a decomposition of the sugar into alcohol and carbonic dioxide), or circumpolarization (deflection of the plane of polarization to the right by the grape-sugar). Certainly in no doubtful case should we omit to perform the fermentation test, for it is easy and gives absolutely unambiguous results. Further particulars in regard to these and other tests, and also in regard to the quantitative estimation of sugar, may be found in works on medical chemistry.

Diabetic urine sometimes contains other varieties of sugar in small amounts —namely, levulose, which deflects the plane of polarization toward the left, and inosite; these are, however, of no practical importance.

The amount of urea is usually somewhat increased (*vide infra*). In the main, this is doubtless connected with the increased ingestion of albumen, but in severe cases it may be that the carbohydrates are so imperfectly appropriated that the albumen of the body is broken up and consequently contributes to an increase in the excretion of nitrogen. Uric acid is usually excreted in approximately normal amounts. In exceptional cases it also seems to be excreted in excess. The amount of kreatinine is apt to be increased (Senator). The amount of phosphoric acid and sulphuric acid usually corresponds to the amount of urea, or, in other words, to the decomposition of albuminoids. Occasionally, according to Teissier, the amount of phosphates is surprisingly great, and it may either correspond with the amount of sugar simultaneously excreted, or replace the sugar in the urine. This subject has not yet been fully investigated. The amount of sodic chloride excreted depends, as in health, merely upon the amount ingested.

Hallervorden has discovered an important fact in regard to the excretion of ammonia. In many cases of diabetes, although not in all, it is much increased: forty-five to ninety grains (grammes 3–6), or even more, may be excreted in twenty-four hours. Despite this, diabetic urine has an acid reaction; and, as Stadelmann has shown, the basic elements are considerably out of proportion to the acids known to us. It is, therefore, evident that diabetic urine, since it contains a large amount of ammonium and yet has an acid reaction, must have among its constituents some unusual acid. The later investigations of Minkowsky and Külz have shown that the acid is mainly oxybutyric acid, or, more accurately, beta-oxybutyric, although sometimes still other acids are found in the urine, particularly volatile fatty acids and lactic acid (Rumpf). The oxybutyric acid is often excreted in large amounts, up to two or three ounces (grammes 50–100) daily. It is traced to the destruction of albumen, and usually does not appear in the urine until the patient is obliged, because of the imperfect appropriation of carbohydrates, to use the albumen of the body instead to satisfy the needs of the economy. The appearance of oxybutyric acid in the urine is, therefore, unfavorable from a prognostic point of view, and in particular it indicates that there is danger of diabetic coma (*vide infra*).

Oxybutyric acid is probably the substance from which are derived two other organic bodies which are not infrequently found in the urine of diabetes—viz., acetone and acet-acetic acid. These substances also arise from the destruction of albumen—that is, the body albumen—so that they are seen especially in severe cases with impaired nutrition (Van Noorden). Acetone is probably developed from acet-acetic acid. The latter is the cause of the reaction which was first demonstrated by Gerhardt, which consists in the appearance of a Burgundy-red color on the addition of chloride of iron, and which is not infrequently to be observed in diabetic urine. Acetone is shown by Legal's test: a few drops of fresh solution of nitro-prusside of sodium are mixed with the urine, and then sodic hydrate is added. If acetone is present a purple-red color appears, [and persists, even if acetic acid is added (excluding kreatinine).—V.].

The not infrequent occurrence of albumen in diabetic urine will be mentioned below, when considering the renal complications.

2. METABOLISM IN DIABETES. SOURCES OF THE SUGAR AND VARIATIONS IN ITS AMOUNT OCCASIONED BY EXTERNAL INFLUENCES.—Inasmuch as the presence of sugar in the urine is the most prominent symptom in diabetes, the question of its origin is all-important. One fact is indubitable—namely, that the secretion of sugar depends in large part upon the amount of potential sugar ingested—that is, upon the proportion of starches in the food. The amount of sugar excreted with the urine increases and diminishes with the amount of starchy food eaten. If a diabetic patient abstains totally for any length of time from such articles of food as contain starch, sugar will, in many instances, entirely disappear from the urine. In other words, the system of a diabetic patient is almost, if not quite, incapable of oxidizing sugar into carbonic-dioxide gas and water. Although, therefore, in a diabetic patient the absorption of oxygen from the atmosphere goes on as usual, yet there always remains a deficit in the excretion of carbonic-dioxide gas and water, corresponding to that portion of the ingested carbohydrates which is not oxidized.

The oxidation of sugar is not absolutely *nil* in diabetes. Külz has proved by numerous experiments that not all the starch is excreted in the form of sugar. In severe cases of diabetes the largest part of the ingested sugar is excreted as such, unoxidized, with the urine, but in mild cases only a small portion is thus excreted. It is, therefore, necessary to determine the degree of tolerance for carbohydrates in every individual case of diabetes; even in the same patient this tolerance is not the same at all times. Another very interesting fact was demonstrated by Külz. The diabetic patient has lost only the power of oxidizing that kind of sugar which turns the plane of polarization toward the right. The sugar which turns it toward the left (levulose), inuline, and also certain other carbohydrates, such as mannite and inosite, are broken up even in diabetic patients in very large part, if not completely, so that the amount of sugar excreted in the urine is but little, if any, increased by the ingestion of these substances.

Yet the carbohydrates are not the only source of the sugar excreted by diabetic patients. In many cases, although not in all, sugar persists in the urine even when the patient receives a dietary absolutely free from carbohydrates. In such instances, there can be no doubt that the glycogen, or the sugar which is formed out of it, is referable to the albumen of the food. In general, we may say that it is always a sign of a severe variety of diabetes to have a part of the sugar which is formed out of albumen, when carbohydrates are absolutely excluded from the diet, excreted unoxidized; while it is a proof that the variety is a milder one, to have the sugar vanish from the urine when the patient is put upon a diet which contains little, if any, carbohydrates.

Muscular exertion is one of the external influences which modify the excretion of sugar in diabetes. According to our present views, muscular activity consumes mainly non-nitrogenous substances, and, accordingly, we find in diabetes that an increase of muscular exertion, other things being equal, diminishes the amount of sugar excreted.

Emotional excitement is said to increase the amount of sugar excreted.

Intercurrent acute febrile diseases may cause a great diminution in the amount of sugar, but sometimes there is no essential change. Probably the altered diet of the patient plays an important part in this connection, although the modifications of metabolism occasioned by the high temperature or by the disease itself also exert some influence. Just before death the urine may become entirely free of sugar.

3. Constitutional Symptoms in Diabetes Mellitus.—In many of the milder cases there is for a long time little apparent disturbance of the general health. The patient is well nourished, and suffers little discomfort, except that he is rather easily fatigued, and somewhat inconvenienced by the polyuria and the polydipsia. In severer cases the system is deeply affected by the drain upon it. The patient becomes emaciated, weak, and easily exhausted, and at length there may be profound marasmus. Mentally, the patient is apt to be depressed and irritable. The intellectual powers are not impaired, but there is indisposition to mental effort. The temperature is normal or subnormal. Fever invariably indicates some complication.

4. Symptoms referable to the Digestive Organs.—We have already mentioned the excessive thirst experienced in diabetes. This may be a source of great discomfort, obliging the patient to drink at short intervals, even through the night. The interdependence of polyuria and polydipsia is not yet fully understood. The most natural view seems to be that the increased excretion of water by the kidneys is the primary factor, and the increased thirst secondary thereto. One cause of the polyuria is the excretion of sugar, in order to dissolve which a large amount of water is necessary, but certain nervous factors would also seem to be implicated. That they exert some influence is rendered probable by the fact that the amount of urine does not always correspond to the amount of sugar excreted. A very large amount of urine may be excreted containing little or no sugar; and, on the other hand, there are genuine cases of diabetes mellitus where the amount of urine is normal, and the patient feels no unusual thirst (*diabetes decipiens*). It has also been suggested that the sugar may irritate the nerves of the mouth and throat, and thus cause thirst. According to this idea, the polyuria would be partly the result of the excessive ingestion of liquids. The abnormally great appetite in diabetes seems to be due to defective assimilation of the food. Many patients are never able to eat enough. They have a longing, in many cases, for carbohydrates. Occasionally the hunger becomes ravenous, and is associated with headache and a general sense of weakness, all these symptoms

being alleviated when food is taken. This rule has rare exceptions in which the appetite is not unusually great, even though the case be severe.

The tongue is frequently dry; it is broad and thick, with an irregular and fissured surface, sometimes coated and sometimes red. The gums may be spongy, and may exhibit a tendency to bleed. A very characteristic condition is necrosis along the free borders of the gums, probably due to an enfeeblement of the resisting power of the tissues. This is particularly apt to lead to a gradual loosening of the incisors, so that at last they drop out. The teeth frequently decay rapidly. The saliva is invariably found to give an acid reaction. This is true of the isolated secretion of the parotid gland also, and is said to be due to the presence of lactic acid. It is only in exceptional instances that sugar can be demonstrated in the saliva. In severe cases thrush sometimes forms upon the soft palate.

Special gastric symptoms are usually absent. There may be a dilatation of the stomach, due to the great quantity of food ingested, but this has no practical importance. If disease of the stomach does develop (e. g., chronic gastritis) the complication is an unfavorable one. There is usually constipation, but sometimes there is a severe, though temporary, diarrhœa. The liver and spleen are seldom much affected; the liver rarely is somewhat enlarged. Jaundice is frequently observed, but it is always referable to some complication. As a rule, the secretion of the bile goes on as in health. After death, we may find atrophy of the pancreas (*vide infra*).

5. SYMPTOMS REFERABLE TO THE RESPIRATORY ORGANS.—In many cases the organs of respiration are unimpaired for a long while. It may be mentioned that often patients have a decidedly fruity odor to the breath (acetone odor). In the later stages of the disease pulmonary complications are very frequent. First in importance is tuberculosis of the lungs. This is particularly apt to occur in the severe forms of diabetes in youth, and it may pursue a rapid and fatal course. The diagnosis can be settled by the discovery of tubercle bacilli in the sputum. Next in point of frequency is pulmonary gangrene. Sometimes there is a diffuse gangrenous process, and sometimes there are isolated foci of necrosis, which become liquid, and have an acid reaction, but they often have comparatively little odor. The expectoration in these cases may be odorless. Croupous pneumonia may also occur. It often terminates unfavorably, and may, as we have ourselves observed, result in gangrene.

6. SYMPTOMS REFERABLE TO THE CIRCULATORY SYSTEM.—In many instances the circulatory apparatus presents no special lesions. The pulse is either of a normal rate or a trifle slow. It is usually soft, although exceptionally it may exhibit increased tension. There is often distinct evidence of cardiac weakness (Schmitz); the pulse is small, intermittent, sometimes very slow (50 or even 40 beats per minute), and sometimes accelerated (100 to 120 beats per minute). There are shortness of breath, faintness, nausea, and the like. Sometimes sudden and profound cardiac disturbance occurs, and this may occasion speedy death (*vide infra*, coma). Not infrequently, diabetes is combined with general arteriosclerosis (*vide infra*).

7. GENITO-URINARY SYMPTOMS.—Despite the great demands made upon them, the kidneys often maintain a normal condition. As we shall see when we come to the pathological anatomy of diabetes, the kidneys are often very large. Sometimes a chronic nephritis is developed as a complication, usually in the later stages of the disease. The urine contains albumen, and there are œdema and other symptoms of renal disorder. The cause of albuminuria in diabetes does not seem to be the same in every case; sometimes it is due to changes in the kidneys resulting from the persistent excretion of abnormal urinary constituents. In this connection we must think, not only of the sugar, but also of the other substances—such as acetone, diacetic acid, and oxybutyric acid—which, as they

traverse the kidneys, damage the renal epithelium. In other cases, diabetes and nephritis seem to be simultaneous and co-ordinate results of the same cause, such as arterio-sclerosis or alcoholism. Finally, it must not be forgotten that other complications of diabetes—e. g., pulmonary tuberculosis—may promote the development of nephritis. It is an interesting fact that in diabetes, glycosuria and albuminuria may to a certain extent alternate with each other. If the amount of albumen in the urine becomes considerable, the excretion of sugar usually undergoes marked diminution. Many physicians therefore consider the transition of diabetes into chronic renal disease (contracted kidney) as a comparatively favorable symptom.

Saccharine urine is apt, as it decomposes, to cause irritation of the skin (development of fungi). This is the explanation of the troublesome pruritus pudendi, which is especially marked in women. It may, indeed, be this symptom which first directs attention to the disease. Sometimes the external genitals are attacked by eczema or furunculosis. Men often suffer from balanitis, with inflammatory phimosis, or paraphimosis. A frequent and important symptom in men is impotence. This sometimes occurs very early in the disease, but it may afterward undergo improvement. The origin of it is probably referable to degenerative changes in the nervous system (*vide infra*). Some authorities state that diabetes is apt to occasion atrophy of the testicles.

8. Disturbances of the Organs of Special Sense.—An important and not infrequent result of diabetes is cataract. This may occasion almost total blindness. The cause of cataract in diabetes is not known. It was formerly supposed that the sugar in the blood absorbed water from the crystalline lens, and thus occasioned its opacity; but this has not been confirmed. Diabetic patients are subject to disturbances of accommodation. Retinitis and atrophy of the optic nerve may occur, but they are very rare. Purulent choroiditis, if it develops, is probably merely a chance complication.

None of the other special senses is peculiarly affected in diabetes.

9. Cutaneous Affections.—In most cases the skin is remarkably dry and rough. There may, however, be abundant perspiration. Several authorities claim to have found sugar in the perspiration, but this statement has not been confirmed by later investigators. Sometimes there is a troublesome pruritus. We often see a rapid loss of hair and a shedding of the nails. In many cases there is a great tendency to furunculosis. This may be the first symptom to suggest the existence of diabetes. In the later stages there are sometimes extensive carbuncles, and multiple phlegmonous abscesses in the subcutaneous cellular tissue, which may prove fatal. Bacteriological examinations of all these processes have not yet been made, so far as we know. Probably they are complications, like the frequent occurrence of pulmonary tuberculosis in diabetes. Occasionally we have seen a pemphigus-like eruption on the skin. This, in one case, developed into a localized gangrene. Gangrenous processes have been repeatedly observed, in particular necrosis of one or more toes, which may take the form of perforating ulcer (*mal perforant*), or rarely of an entire extremity. This gangrene often seems to be due to arterial sclerosis; in other cases its cause is obscure. The gangrene of the toes may be apparently the first symptom of the disease, and it may be very slight. It is not until all therapeutic and operative procedures prove unavailing that suspicion is aroused, leading to the discovery of diabetes. We have often seen cases of this sort, which usually turn out badly, in obese drinkers.

Œdema of the subcutaneous cellular tissue may occur independently of nephritis. It is then probably occasioned by the cardiac weakness.

10. Symptoms referable to the Nervous System.—It has already been mentioned that there is frequently in diabetes a moderate disturbance of the whole nervous system, as indicated by headache, physical and mental hebetude, and

capable of producing the nervous symptoms, are not true diabetic coma. Nor do the cases of sudden death reported by Frerichs deserve to be classed as diabetic coma, where death occurred with the symptoms of acute cardiac failure—namely, collapse, coolness of the extremities, small and rapid pulse, and unconsciousness. Besides, these patients never have the acetone odor, the exaggerated respiration, nor the ferric-chloride reaction, and usually the myocardium is found to be in an advanced state of degeneration.

In genuine diabetic coma, on the other hand, everything seems to indicate that the system has been poisoned by some noxious product of abnormal metabolism. Great effort has been made to discover what this product is, but with imperfect success. Kussmaul regarded acetone as the injurious substance, and therefore called diabetic coma "acetonæmia." Other investigators believe that acet-acetic acid (Jaksch) is the cause of the phenomenon in question, or at least regard the coma as a result of poisoning from some of the unusual acids present in diabetes (Stadelmann, *vide supra*). No one view has obtained general acceptance, nor has it been possible to produce diabetic coma in animals by the employment of acetone, acet-acetic acid, crotonic acid, or similar substances (Brieger and others). It is, nevertheless, extremely probable that these substances do have some close connection with "diabetic intoxication" (Frerichs). Far more probability attaches to the theory which was advocated first by Stadelmann and then by Minkowsky, according to which diabetic coma is the result of an "acid intoxication." There is no doubt that, as we have already stated, in diabetes there is a formation of abnormal acids, particularly oxybutyric acid. If these acids accumulate in the blood, they neutralize its alkalies, so that it loses its capacity of combining with the carbonic acid formed in the tissues, and carrying it away. Certainly the symptoms of diabetic coma bear a great resemblance to the phenomena which have been observed in animals when poisoned by acids—viz., stupor, dyspnœa, diminution of the amount of carbonic dioxide in the blood, and increase in the excretion of ammonium in the urine (*vide supra*). The blood of patients who die in diabetic coma contains large amounts of oxybutyric and sarcolactic acids.

11. CHANGES IN THE LYMPH-GLANDS.—There may be extensive and distinct swelling of the lymph-glands, particularly in severe cases of diabetes. The glands are hard and insensitive.

Pathological Anatomy and Histochemistry of Diabetes Mellitus.—If we exclude the organic diseases, such as pulmonary tuberculosis and nephritis, which are merely complications, the pathological changes in diabetes are trifling. Bernard's discovery, that an injury inflicted in a certain spot on the floor of the fourth ventricle produces glycosuria in animals, has directed the attention of investigators to the condition of the nervous system in this disease. In some instances, tumors, sclerosis, or similar troubles have been found in the medulla and cerebellum; but in these cases there was evidently a symptomatic glycosuria and not an idiopathic diabetes (*vide supra*). In idiopathic cases the central nervous system presents no striking macroscopic changes. By means of the microscope, Frerichs has found lesions of the medulla oblongata in frequent instances. The minute blood-vessels are widely dilated; there are small capillary hæmorrhages, some of a more recent and others of a more remote date; and occasionally there are microscopic foci of myelitis. The nervous elements proper, the nerve-fibers and ganglion cells, betray no alteration; and as the changes enumerated are not invariably present, their significance is dubious.

The stomach and intestines present no constant alterations of importance.

The liver has naturally been the object of repeated and careful examinations, because of its well-known part in the manufacture of glycogen. Yet this organ seldom presents any special abnormality. It is usually of the natural size, and it may contain either considerable or very little blood. The amount of glycogen in the hepatic cells can be demonstrated with iodine by a micro-chemical reaction. It appears that, other things being equal, there is less glycogen present than normal. It is usually found only in the cells situated upon the periphery of the lobules, and in small quantities. In an extremely advanced case of diabetes Ehrlich obtained, by means of a hollow needle, small amounts of the hepatic parenchyma for examination during life, and found that glycogen was almost completely absent. In other cases the liver has been examined as early as possible after death, and, with rare exceptions, has presented no trace of glycogen.

The spleen is usually of normal size. Occasionally it is atrophied, or, on the other hand, slightly enlarged. No other changes in it have been observed.

Not a few cases have presented a striking atrophy of the pancreas (Bouchardat), a fact which has acquired great significance from Minkowsky's discovery, already mentioned, that glycosuria may come on after extirpation of the pancreas. It is uncertain whether the cœliac plexus may also sometimes share in the atrophy of the pancreas. We must also state, however, that atrophy of the pancreas is not at all a constant lesion in diabetes, so that it is not proper to regard the disease of the pancreas as the special cause of diabetes in all cases. Lately in repeated instances the pancreas has been found perfectly normal, even upon the most careful microscopic examination.

The kidneys are usually enlarged, from functional hypertrophy. Ehrlich discovered in them a glycogenic degeneration of the loops of Henle. The epithelial cells in the loops are enlarged, and the protoplasm in these cells, although apparently homogeneous, is found, by the addition of a solution of iodine in mucilage, to contain glycogen, in flakes and clumps of varying size. How important this glycogenic degeneration of the kidneys may be has not yet been determined. Perhaps the glycogen represents sugar which has been absorbed by the cells. That chronic nephritis may complicate diabetes has already been mentioned.

No thorough investigation of the chemical composition of the blood in diabetes has yet been made. One fundamental and constant characteristic is the greatly increased proportion of sugar in the blood. There is usually somewhere between 0.2 and 0.45 per cent. of sugar, while in health the blood rarely contains over 0.1 per cent. The lymph, and such serous effusions as are found, contain sugar, but the saliva, perspiration, bile, gastric juice, and other secretions rarely furnish evidence of its existence.

Varieties, Course, and Prognosis of the Disease.—The study of a large number of cases of diabetes will show great variations in the course and duration of the disease. As already stated, we may in practice distinguish between mild and severe forms of diabetes. Following Seegen's suggestion, this division is usually based upon the relation between the sugar excreted and the character and amount of the food eaten. Those cases are regarded as belonging to the mild form in which the sugar disappears from the urine when the patient takes food containing no carbohydrates. Some patients with this milder form may ingest small amounts of the amylacea, particularly if they take a proper amount of physical exercise (*vide infra*), without bringing on glycosuria. In the severe form of diabetes, on the other hand, the excretion of sugar in the urine persists even on a purely meat diet, and if any carbohydrates are taken, there will be a corresponding and large increase in the amount of sugar in the urine, as soon as half an hour to an hour thereafter. Of almost greater practical importance are the clinical signs which sometimes indicate, at the first glance, that an individual suffers from the mild or the severe form, as the case may be. The severe cases usually attack youthful individuals; the patient loses flesh rapidly, his face becomes narrow, and is apt to assume a peculiar expression of quiet melancholy; he is languid and feeble, and the urine presents all the indications of diabetes in extreme degree. The milder cases, on the other hand, are more apt to occur in ter life, the patient has only moderate discomfort, and usually retains considable strength and energy; he is but little, if at all, emaciated, and even the anges in the urine are so slight that they are not recognized until the attention especially directed to them. It must be noted, however, that an apparently ild case of diabetes may become severe. Sometimes, also, the behavior of the se as to the excretion of sugar remains like that of the mild variety, and still, tal complications occur at last, such as coma, necrotic processes, and pulmonary berculosis.

In general, the clinical course of diabetes presents great variations. In a few stances the disease occupies only a few weeks, and it may almost be termed cute diabetes." Other cases last one or two years, and still others ten or enty years. The patient's condition may vary from time to time. We have peatedly seen the sugar disappear temporarily from the urine, and the patient parently completely recover; but sooner or later the disease breaks out again. ese cases, which are commoner in old people, have been called "intermittent abetes." The relapse is often brought about by emotional excitement or some ave error in diet. Again, the disease may become apparently stationary and e patient enjoy comparative comfort for years.

Different cases also differ in the relative severity of particular symptoms. us, the clinical picture presented by diabetes may be modified by the general nstitution of the patient—for instance, his corpulence or emaciation; by such mplications as diseases of the lungs, kidneys, or brain, or syphilis and gout; d by many other conditions. In practice, especially in practice among the tter classes, it is very important to have a knowledge of the milder forms of abetes, in which sugar is present in the urine only at times, and not in very rge amount. The more we are accustomed to examine every urine for sugar e oftener we find such cases, as to the significance of which our knowledge is ill very slight. The glycosuria of the corpulent is worthy of special consideran. It is, as we have said, particularly apt to occur in obese beer-drinkers, and is not uncommon in patients who have formerly suffered from true gout. The ndency to the formation of furuncles and cataract is noteworthy. We must also ntion the cases of diabetes in neurasthenics—that is, cases where there is gar in the urine, and where the chief complaint is of general nervous symptoms, essure in the head, anxiety, a dull feeling, incapacity for mental work, neuralpains, etc. In such cases glycosuria has not at all the serious significance that has in genuine and severe diabetes. Finally, we would point out an especial nical form of diabetes, in which glycosuria occurs in patients with severe genal arterio-sclerosis; usually the patients are men in middle or advanced life. t infrequently the condition seems referable to long-continued mental excitent or exertion. The amount of sugar in the urine is not large, and the sugar ually entirely disappears with appropriate diet; still, this form can not be unalifiedly regarded as mild, for we must bear in mind, also, the serious results of terio-sclerosis. There are particularly apt to be cardiac symptoms referable to terio-sclerosis of the coronary arteries. In all these "mild" varieties of diates there may be no polyuria or thirst, so that an error in diagnosis is easy less a careful examination of the urine is made.

We should also mention the interesting fact, of which Frerichs reports some riking examples, that diabetes mellitus in a few exceptional cases may graduly change into diabetes insipidus (see the following chapter). The reverse ange is also said to occur.

The usual termination of diabetes is death. We have already seen how great difference there may be in the length of time preceding the fatal termination, d in what various ways it may be brought about. The most frequent immediate causes of death are marasmus, diabetic coma, pulmonary consumption, nephriti furunculosis, or the development of carbuncles.

There is no doubt that complete recovery may occur; but this is rare, and possible only in the milder cases. It should also be borne in mind that, as w have said, apparent recovery does not exclude the possibility of a fresh outbrea of the disease.

Theoretical Discussion of the Nature of Diabetes.—We have endeavored to give an approximately complete summary of the facts pertaining to diabetes. We trust we shall be excused from detailing all the theories and hypotheses which have been devised to explain the peculiar phenomena of the disease, particularly the glycosuria. It is a better way simply to confess that the true nature of diabetes mellitus as yet remains very obscure. We shall confine ourselves to a few remarks upon the present state of the question.

The essential fact which demands explanation is the excess of sugar in the blood. The blood of a healthy man does not contain over 0.15 per cent. of sugar, while that of a diabetic has 0.22–0.44 per cent. The source of this sugar is probably the same as of the sugar normally contained in the blood. The largest part of the sugar probably comes from the carbohydrates ingested as food. These are, for the most part, converted in the primæ viæ, into sugar, which sugar thereupon enters the portal system. It may also be assumed that glycogen, so widely diffused throughout the system, is another source of sugar. The liver is the main seat of the manufacture of glycogen; but it is produced in other parts as well, and in particular in the muscles. The question next arises, From what is the glycogen formed? A part of it is manufactured from the carbohydrates contained in the food, but another part is certainly due to the ingested albuminoids. Thus, Von Mehring found that in fasting animals, who have no more glycogen stored up in the body, diabetes may be produced by giving phloridzine (*vide supra*). The sugar excreted can therefore come only from the destruction of albumen in the body. Again, the transformation of glycogen into sugar is not confined to the liver, but it may take place wherever glycogen is produced. How it takes place is unknown. It is usually assumed that there is some "saccharific ferment."

It would seem, therefore, that the sources of sugar are the same in diabetes as in health. We have next to seek for the reason of its excessive accumulation in the blood. Under normal circumstances, the sugar present in that fluid rapidly undergoes decomposition into other substances. In health there is no great excess of sugar in the blood, even upon a diet extremely rich in starch; and it is possible to eat large amounts of sugar without any glycosuria. That changes in the secretory action of the kidneys are not the cause of the excretion of the sugar, is evident from the fact that in diabetes insipidus no glycosuria occurs even if a large amount of sugar is ingested (Frerichs). We see, therefore, that diabetes can not be explained by assuming that there is an increased production of sugar, nor in fact is there such an increase, except as corresponds to the increased amount of ingesta. On the other hand, everything points toward the conclusion that in diabetes the ordinary processes effecting the decomposition and destruction of sugar are suspended. The sugar is excreted by the kidneys unaltered, for the reason that it is not destroyed. It is difficult to conjecture what the circumstances may be which thus interfere with the decomposition of the sugar. Perhaps it is some special nervous influence, or perhaps some ferment may be wanting in diabetes which in health promotes the metamorphosis of the sugar. This latter supposition is rendered the more probable by the above-mentioned experiments of Von Mehring and Minkowsky. Lépine also, having repeated these experiments, has reached the conclusion that the blood, under normal conditions, receives directly from the pancreas a "glycolytic" ferment which decomposes sugar. In a diabetic subject this glycolytic function of the blood is supposed to be much impaired, because the blood is deprived of this ferment, and as a consequence sugar is excreted unchanged; but this explanation has been disputed, so that really the question as to the true cause of diabetes remains absolutely unsettled.

Diagnosis.—For the diagnosis of diabetes mellitus it is indispensable that sugar should be demonstrated in the urine. We have also to decide whether the condition be a temporary or a permanent one, or, in other words, whether we have to deal with mere glycosuria (*vide supra*) or genuine diabetes mellitus. This point is to be determined by means of the symptoms and general course of the disease.

Diabetes often exists for some time unsuspected even by the physician. It may therefore be well to name over the symptoms which may be the first to attract the patient's attention, and which should therefore always suggest to a physician the possibility of the existence of diabetes. They are: 1, languor and debility; 2, furunculosis; 3, pruritus pudendi in women, balanitis in men; 4, cataract; 5, sciatica, especially if bilateral; 6, impotence. We should be in the habit of examining the urine for sugar in all cases when complaint is made of indefinite symptoms, which can not be explained without further evidence, especially if the patient is fat or nervous, and also if he is elderly and shows signs of arterio-sclerosis.

If symptoms similar to those just enumerated lead to an examination of the urine, and the result of this is ambiguous, it is advisable to have the patient partake of a meal rich in starchy elements, and to examine the urine thereafter, preferably by means of the fermentation test. If even then no sugar is found, diabetes does not exist.

Treatment.—Medical science does not possess the power to cure the disease, but it can greatly benefit the patient, both by alleviating his symptoms and by shielding him, at least for the time, from many of the secondary effects.

The first requisite is to institute a proper regimen. All the hygienic circumstances of the patient should be regulated. This is more important than any sort of medicinal treatment. We must consider that the increased amount of sugar in the blood is not without influence upon the tissues of the body, that as a consequence of the imperfect conversion of the carbohydrates there may be other injurious substances formed in the blood, and that the presence of sugar in the urine may lead to certain disagreeable symptoms (pruritus, balanitis); and consequently we must aim by our dietetic treatment to prevent any excessive accumulation of sugar in the blood, and at the same time to furnish the body with a substitute for the carbohydrates which can no longer be employed as food.

It would be erroneous to conclude that this last point is the only essential one, and that we accomplish our whole duty by reducing the amount of sugar contained in the urine to a minimum. The general condition of the patient should invariably be considered. There can be no doubt that a diabetic patient whose strength is well maintained is better off, even if there is considerable sugar in his urine, than one whose urine contains less sugar, but who yet is daily growing weaker. In managing the dietary of a diabetic patient, we must first determine what tolerance for carbohydrates there may be in each particular case. The principle of dietetic treatment of diabetes does not consist in the simple withdrawal of all carbohydrates, but rather in allowing each patient as much of such substances as his body is in a condition to appropriate. This amount varies greatly in different cases, and also in the same patient at different times. We must first determine whether the case belongs to the severe or the mild type, as above defined—that is, whether sugar entirely disappears from the urine if no carbohydrates whatever are allowed. If it does—that is, if we are dealing with the milder form of diabetes—we then proceed, after the urine has been freed from sugar, to give gradually increasing amounts of carbohydrates, at the same time examining the urine every day in order to see whether the starch ingested is completely oxidized in the blood. The more carbohydrates that can be allowed without the appearance of sugar in the urine, the more favorable the case. When we have in this manner measured the ability of the system to utilize carbohydrates, the patient is kept for a considerable time upon such food as his system can manage completely to appropriate. The experience of physicians has shown that this sort of limitation of the carbohydrates may have a permanent beneficial influence, inasmuch as after the patient has adhered for a considerable time to a diet which keeps the urine free from sugar, the tolerance of the system for carbohydrates increases. We may, therefore, try gradually to make some increase in the amount of carbohydrates, and we shall not infrequently find that after persistence in such a diet for a considerable time, the patient will bear more carbohydrates without the return of glycosuria. Thus, guided by constant examinations of the urine, we can easily prescribe the correct diet for the individual patient.

If we are dealing with the severe form of diabetes—that is, if sugar is still excreted in the urine, even when the diet consists exclusively of meat and fat—we may try the effect of withdrawing all carbohydrates completely for a considerable time. Many physicians (Cantani, Naunyn, and others) have observed that an absolutely strict diet of this sort, excluding all carbohydrates, may have a very favorable therapeutic effect, so that finally the patient will regain a certain tolerance for carbohydrates; but it is precisely in these severe cases that, in our opinion, it is improper to be guided too much by general rules. We should consider not only the character of the urine, but the condition of the individual in other respects—his subjective sensations, his weight, and his strength. Many of these severe cases undoubtedly feel better when they are allowed a moderate amount of carbohydrates than when they are on a purely nitrogenous and fatty diet, despite the somewhat greater amount of sugar excreted. Particularly when the appearance of acetone, acet-acetic acid, butyric acid, and the like in the urine, suggests a considerable destruction of the albuminoids of the body, and perhaps an impending diabetic coma, practical experience has shown that we should not insist too strictly upon a purely nitrogenous diet, but should try, by the cautious administration of carbohydrates, to improve the general condition and avert the danger of coma.

As we have already mentioned, it is decidedly advantageous to make up for the diminution in carbohydrates by giving other food which does not contain nitrogen. The most natural thing, certainly, is to allow an abundance of fat as a substitute for the carbohydrates; and practical experience is, as regards this matter, in entire unison with theory. Fat is well borne by most diabetics, and it may be allowed in the form of butter, cream, salad oil, and the like. It should be particularly recommended to emaciated patients, if they can digest it. Sometimes cod-liver oil may be prescribed for a time. Beside the fats, the use of alcohol as a preventive of waste might be considered; in small amounts brandy and claret may be allowed to some patients. We would earnestly advise against the use of larger amounts, for the toxic effect of alcohol might injure the vitality of the cells (*vide supra*).

With a view of aiding the physician in preparing a bill of fare for his individual cases, we append a list of the separate foods which are available for the nourishment of diabetic patients: 1. A diet which contains almost no carbohydrates would be as follows: On waking, tea or coffee with cream, but no sugar; for breakfast, eggs, ham, cold meat; at noon, broth, not thickened, fish with butter, meat with greens or salad; for supper, tea, eggs, cold meat, cheese; in

ddition the patient may drink water or a moderate amount of claret. The fol-
owing articles are almost absolutely devoid of carbohydrates: all sorts of meat,
am, smoked meat, tongue, all sorts of fish, crabs, caviare, oysters, sardines, eggs,
reen-colored vegetables, lettuce, endives, cress, asparagus, cauliflower, spinach,
ucumbers, red and white cabbage, string beans when young, sorrel. Of course,
f these are boiled, only butter can be added, or meat, but no cereal. Other arti-
les of this class are all sorts of cheese; such fatty foods as butter, olive-oil, and
ream; meat broths, artificial meat-peptone, meat extract, somatose, mineral
aters, and light claret. 2. Articles which contain a small amount of carbo-
ydrates, and which, therefore, may be allowed in moderate amounts if there is
certain degree of tolerance for carbohydrates (*vide supra*): rusks, rye bread,
ilk, cocoa, turnips, kohl rabi, radishes, nuts, sour fruits—such as apples, cur-
ants, and cherries. 3. Articles which contain a large amount of carbohydrates,
nd are therefore to be prohibited or allowed sparingly, according to individual
ircumstances: all sweet dishes, cake, honey, potatoes, oatmeal, rice, peas, beans,
entils, sago, sweet fruits, sweet wines, and beer.

The hardest thing for diabetics to do without is bread. Every physician who
as had much experience with diabetic patients can relate experiences of the cun-
ing of patients in satisfying their unconquerable longing for this forbidden
rticle by the employment of underhand means. Hence, many attempts have
een made to furnish special sorts of bread for diabetic patients, containing a
inimum proportion of carbohydrates. Most of these preparations, however,
he patients do not like. Chiefly to be recommended are gluten bread and aleu-
onat bread. Finally, it is well to employ saccharine to sweeten food and drink,
articularly tea and coffee, provided the saccharine does not produce dyspepsia
nd also proves agreeable to the patient.

[The dietetic treatment of diabetes is so important that it is desirable to go
ore into detail.

There is some discrepancy between the authorities on this point, a more strict
iet being laid down by some than by others. Ralfe's list is as rigid as any, and
s as follows:

To avoid: Milk (except very small quantities for cooking purposes). The liver
f all animals (as the liver of oysters and all mollusca is large, and abounds in
lycogen, these animals must be forbidden), so also the interior of crabs, lobsters,
tc. Bread, biscuits, rusks, toast, farinaceous vegetables, such as potatoes, Jeru-
alem artichokes, rice, oatmeal, corn-flour, sago, tapioca, arrowroot, etc. Saccha-
ine vegetables, turnips, carrots, parsnips, green peas, French beans, beet-root,
sparagus, tomatoes. Blanched vegetables of every sort, as celery, sea-kale,
ndive, radishes; also the stalks and white parts of such vegetables as cabbage,
ttuce, broccoli, etc. Fruits of all kinds. Jams, syrups, sugars. Certain condi-
ents, such as chutney and sweet pickles, cocoa, chocolate, liqueurs, sweet wines.

May take: Meat, fish, poultry, game, bacon, ham, eggs. Bread and biscuits
de with prepared gluten, bran, or almond-flour. Green vegetables, summer
bage, turnip-tops, spinach, broccoli-tops, water-cresses, mustard and cress,
r, sauerkraut, the green parts of lettuce, sorrel, mushrooms. Nuts of various
s (except chestnuts). Cheese.

lint's list is more lenient, allowing oysters and a much larger choice of vege-
s, such as asparagus, string beans, artichokes, cauliflower, tomatoes, etc.
urnal of the American Medical Association," July 12, 1884; also "Pepper's
m of Medicine," vol. ii, page 221).

nkin's treatment by skim milk exclusively is highly approved by Tyson.

enish and similar wines, moderate quantities of spirits, or a malt liquor in
the sugar has been entirely converted into carbonic acid and alcohol (Bass's
instance), are permitted.

In the opinion of the writer, it is always well to begin treatment with a very stringent dietary, which may be relaxed gradually as circumstances dictate. The severity of the case is to be regarded rather than the name of the disease. The gluten and other diabetic flours are unreliable; and I agree with Flint that, if bread be allowed, it is better to give the crust of a French roll, the ingredients of which are known. Saccharine may be used to sweeten tea and coffee, jellies, ice cream, and the like.]

Certain other general directions are important. The patient should take sufficient exercise. Külz has determined by means of accurate experiments that, other things being equal, the assimilation of sugar is increased by muscular activity, with a consequent diminution in the excretion of sugar. Practical experience also shows that regular exercise is extremely beneficial. A proper discretion should be employed, however; nothing would be more injudicious than to force a feeble patient to exhausting efforts; but if the patient be vigorous and well nourished, he should be strongly urged to take a walking-trip in the mountains, or to try horseback riding and the like. Methodical massage of the muscles sometimes has a good effect.

Proper care of the skin is indispensable. Baths, cold sponging, and douching may be employed. Strict attention should also be given to the teeth, lest they become carious. Thorough ventilation should be maintained, both day and night.

Of internal remedies, opium should be named first. One good effect of this drug is that it lessens the annoying thirst. It sometimes also causes decided diminution in the amount of urine and sugar excreted. It is further indicated when there is general restlessness or sleeplessness. It is often well borne by diabetic patients, even in large doses. A patient sometimes can take four to eight grains (gramme 0.25–0.5), or even more, of opium in twenty-four hours without any bad effects. We have repeatedly administered to patients with a severe form of diabetes, for weeks at a time, from sixty to one hundred drops of laudanum, divided into three doses daily, with apparently the best results. It should be especially noted that the bowels were not at all disturbed by the drug. It is deserving of mention that the alkaloids of opium, such as morphine and codeine, possess much less value than opium itself.

[The inhalation of oxygen is worthy of mention as a remedial measure.

Purdy seems to show that diabetes attains its highest mortality in those States which combine relatively high altitudes with low temperature. A residence in the Gulf States or Southern California may therefore be curative or prolong life.]

The alkalies, and still more the alkaline mineral waters, enjoy a reputation second only to that of opium. Hundreds of patients visit Carlsbad, Neuenahr, and Vichy every year, to return much benefited. Of course it must not be forgotten that it is not merely the waters which produce these beneficial changes. Other factors are also important, in particular the strict diet, exercise, fresh air, and freedom from the cares of the household and business. Why the alkalies should act favorably we do not know. Perhaps it is because they neutralize the abnormal acids in the organism. Griesinger, and later Külz, as well as others, have made careful comparisons of the amounts of sugar excreted under like circumstances, with and without the ingestion of bicarbonate of soda or of Carlsbad water and similar substances, and for the most part have not been able to perceive any benefit from these remedies. Practical experience, however, shows the value of these alkaline waters; and their use is to be recommended, provided the patient is comparatively vigorous and fit for travel, although his expectations should not be raised to too high a pitch.

From a theoretical point of view, there is interest in the fact that certain remedies which are antagonistic to fermentation have been shown by Ebstein and

Müller to diminish the excretion of sugar in many cases of diabetes. Chief among these are carbolic acid and salicylate of sodium. Carbolic acid is given in the amount of ten to twenty grains (gramme 0.5–1.5) per diem. The amount of salicylate of sodium is one to two and a half drachms (grammes 5–10) daily. There is no doubt that these drugs possess the property ascribed to them; but they are not advantageous to the patient, inasmuch as the general condition is seldom improved by their use. On the contrary, very unpleasant effects are sometimes observed.

We need not enumerate all the remedies which have been recommended in diabetes. We have already mentioned those that possess any extended reputation. We may therefore merely refer to certain drugs which have been lately introduced.

Cantani has suggested the employment of lactic acid in the amount of one to two and a half drachms (grammes 5–10) per diem, dissolved in half a pint of water. This drug may perhaps serve as a physiological substitute for sugar, as glycerine is supposed to do (*vide supra*), but it has no specific virtues. Certain salts of ammonia, such as the carbonate and acetate, are said to diminish the excretion of sugar, and have therefore long been employed in diabetes. Good results can not, however, be claimed for them, nor for iodoform (3–7 grains, gramme 0.2–0.4, daily), nor for the lately much-lauded jambul. The trials of pancreatic juice, also, have led to no definite results.

It is evident, in brief, that the best mode of treating diabetes, at least accordng to our present knowledge, is by regulating the diet, and that it is well, in addition, to recommend the employment for a time of the above-mentioned minral waters, with opium and other internal remedies to combat special symptoms. The treatment of such complications as phthisis or cutaneous eruptions need not e described here.

In diabetic coma, camphor or ether, subcutaneously, should be employed, ogether with lukewarm baths and douching. In consideration of the possibility hat diabetic coma is connected with an acid intoxication (*vide supra*), we should lways try the administration of large doses of carbonate of soda, either by the mouth or in an enema, or by the subcutaneous or intravenous injection of a three-o five-per-cent. solution. The results thus far, it must be confessed, are not very avorable; still, sometimes this mode of treatment has been decidedly beneficial. A more important matter seems to be, as Naunyn pointed out, the administraion of two to five drachms (grammes 10–20) of the bicarbonate of soda, daily, or a long time, to any diabetic patient whose urine gives a positive ferric chloide reaction (*vide supra*), thus justifying suspicion that an acid intoxication is eginning.

CHAPTER X

DIABETES INSIPIDUS

Definition and Ætiology.—In the preceding chapter a distinction was drawn between diabetes mellitus and the symptomatic condition termed glycosuria. There is a similar distinction to be made between diabetes insipidus and polyuria. Polyuria is an increase in the volume of urine, and mainly in the amount of water excreted by the kidneys. It is a symptom which may be produced in many ifferent ways. In the first place, it is a natural consequence of the ingestion of arge amounts of water, or of the absorption of serous effusions; it also occurs in ertain diseases of the nervous system, especially of the medulla and cerebellum;

it is occasionally seen, as we have had opportunity to observe, in chronic hydrocephalus, and is a not very infrequent phenomenon in hysteria. Large amounts of urine are also secreted in certain renal diseases (interstitial nephritis and amyloid degeneration), and often during convalescence from acute diseases, such as typhoid fever, or after the ingestion of certain drugs, called diuretics.

Diabetes insipidus, on the other hand, is a disease which may develop idiopathically in persons otherwise perfectly healthy. Its ætiology is unknown. It occasionally seems to be excited by emotional disturbance, concussion or other injury of the brain, or some previous acute disease, such as typhoid or typhus fever, malarial poisoning, and cerebro-spinal meningitis. The disease sometimes appears in the syphilitic, and it may, therefore, in many instances, be due to syphilis. Patients frequently state that their symptoms began immediately after drinking a very large amount of some fluid, as on a very hot day or after a long march. Finally, there are cases in which the disease appears to be hereditary, or to attack members of the same family. A distinction should be made between genuine diabetes insipidus and those cases in which the primary symptom is not polyuria, but an abnormal thirst, polydipsia, so that the polyuria is merely a result of drinking so much water. In many of these cases the polydipsia is undoubtedly a hysterical symptom (see the chapter on hysteria).

The true nature of diabetes insipidus is entirely unknown to us. The view which seems most probable of any is, that some nervous disturbance is its direct cause. In support of this, we have the appearance of a "symptomatic diabetes insipidus" in connection with organic disease of the brain (*vide supra*), and the fact that polyuria may be artificially excited by injury to a definite spot in the floor of the fourth ventricle or by section of the vagus nerve. Diabetes insipidus presents a most striking analogy with diabetes mellitus. This is shown both by the similarity in ætiology and symptomatology, and still more by the fact that occasionally one disease merges into the other.

Diabetes insipidus is a disease of very infrequent occurrence, and is decidedly less often seen than diabetes mellitus. Most patients are youthful or middle-aged. Genuine diabetes insipidus has also been observed in children, perhaps in connection with hereditary syphilis. The male sex is somewhat more liable to the disease than the female.

Clinical History.—Diabetes insipidus may be developed gradually or with considerable abruptness; the latter case is especially frequent when there is some definite cause, such as the ingestion of a large amount of liquid, or traumatism.

The essential and characteristic symptom is an increase in the volume of urine. This is usually very considerable. Often eight or ten quarts (8,000 to 10,000 cubic centimetres) are excreted in twenty-four hours, and cases have even been reported where the amount reached the almost incredible volume of thirty to forty quarts (litres). If a healthy person and a sufferer from diabetes insipidus are each given the same amount of water in food and drink, the sick man will excrete more urine than the healthy. In color the urine is very pale, and som times almost like water. The specific gravity is very low, being usually 1004 1002, or even 1001. The reaction is slightly acid, sometimes almost neutral.

The percentage of solid constituents in the urine is, of course, trifling. total amount of solids, however, corresponds perfectly with the ingesta, or inc is even somewhat above normal. The amount of urea, in particular, seems t increased, but it has also been stated that other constituents of the urine been excreted in abnormally large amounts—namely, phosphoric acid, sulp acid, lime, and kreatinine. Inosite has been found in the urine by Straus other observers, so that it has even been proposed to give diabetes insipid name of "diabetes inositus," in distinction from diabetes mellitus. Inc not invariably present, however, in the urine of diabetes insipidus. In cases of true diabetes insipidus, albuminuria is extremely exceptional.

Another important symptom is the excessive thirst. To make up for the loss of water by way of the kidneys the patient is obliged to drink great quantities of liquid, and, indeed, it is always found that the amount of water ingested, in the way of drink and solid food, somewhat exceeds the total volume of urine excreted. Despite this, the tongue is usually dry, as is also the skin, the insensible perspiration being considerably below the normal amount. The furunculosis seen in diabetes mellitus is exceptional in diabetes insipidus. The same is true of pruritus, balanitis, etc. Occasionally profuse salivation has been associated with the disease.

Symptoms referable to the various internal organs are few. Cataract has been occasionally observed, but it is much less frequent than in diabetes mellitus. The same may be said of pulmonary tuberculosis. In most cases the appetite is not excessive. The bowels are regular or slightly constipated. There is seldom much gastro-intestinal disturbance, unless from some chance complication. The sexual functions are usually unimpaired.

The general health is considerably affected in cases of any severity. The patient becomes emaciated, languid, and feeble, and has no inclination to mental or physical exertion. Sleep is often disturbed, the mind depressed. The temperature is normal, or even a trifle below normal, probably as a result of the large amount of cold water drunk.

Diabetes insipidus is a very chronic disease. If there is no serious complication it may last for decades, yet there are cases that run a more rapid and unfavorable course. Sometimes there are considerable vicissitudes in the condition of the patient, in part dependent upon external circumstances and in part apparently spontaneous. In case some intercurrent acute disease develops there may be, during its continuance, a considerable diminution in the amount of urine excreted.

The termination is usually unfavorable. Recovery is extremely rare. In the more fortunate cases the condition finally becomes stationary, and the patient attains to advanced years. Sometimes, however, death occurs more prematurely, being usually hastened by phthisis or some similar complication.

Weil has lately contributed to our knowledge of this disease the results of an accurate study of its hereditary and apparently congenital variety. Weil narrates the history of a family in which marked polyuria and corresponding polydipsia appeared in numerous members for several generations. These persons all enjoyed excellent health, with this exception; and most of them attained old age. We hardly need to emphasize the fact that this form of the disease is radically different from the ordinary acquired variety. Perhaps its true cause is an abnormal congenital permeability of the glomeruli, but we have no certain information in regard to it.

Post-mortem Appearances.—Such lesions as have been found in diabetes insipidus are usually the result of fortuitous complications, such as tuberculosis, carcinoma, and pneumonia. There are but very few changes referable directly to the disease itself: among these are enlargement of the kidneys and dilatation of the ureters. In rare instances a possible cause for the symptoms has been found in some lesion of the central nervous system, but these were cases of symptomatic polyuria and not of genuine diabetes insipidus. Instances of this sort are seen in connection with tumors or inflammatory changes in the medulla or cerebellum, and exostoses at the base of the skull.

Diagnosis.—The characteristic urinary phenomena usually render the diagnosis of diabetes insipidus an easy matter. It is of course necessary to exclude such diseases as might occasion symptomatic polyuria (*vide supra*); but this is seldom difficult if we make a careful physical examination and duly consider all the attendant symptoms. The differential diagnosis between diabetes insipidus and diabetes mellitus can almost invariably be made by means of the urinometer. If the specific gravity is below normal, it is scarcely necessary to test for sugar, although in doubtful cases this is the sole criterion.* [The early stage of contracted kidney has repeatedly been mistaken for diabetes insipidus.—V.]

Treatment.—No special injunctions with regard to diet are required. It would of course be a mistake to forbid the patient to assuage his excessive thirst; but we may possibly lessen the amount of water drunk by prescribing bits of ice, or lemonade and other acid drinks. Opium sometimes lessens both the thirst and the amount of urine excreted. Rather large doses should be prescribed, say fifteen to twenty drops of laudanum, several times a day. It is also important that the skin should be well cared for by means of baths and friction, and every effort should be made to promote the general vigor of the patient. He should have nourishing food and good air.

There have been a great number of internal remedies recommended as specifics, but, in general, we can expect no great results from them. Most employed are valerian, of which one to one and a half drachms (grammes 5–10) are given daily in powder, or infusion; and ergotine, of which one and a half to eight grains (gramme 0.1–0.5) are given several times a day. Other remedies which are said to have been sometimes tried with benefit are carbolic acid, atropine, and the bromides; also, galvanization of the cervical cord.

Occasionally we may find an apparent cause for the disease and endeavor to remove it. If there is a suspicion of syphilis, mercurial inunctions should be tried, perhaps in combination with iodide of potassium or sodium. Sometimes, as we can confirm by our own experience, they have an excellent effect. Of course, where there is symptomatic polyuria, the primary disease, such as hysteria, demands treatment. [Da Costa and others report very good results as following the administration of ergot.]

CHAPTER XI

GOUT

(Podagra)

Ætiology.—Thomas Sydenham was the first to write a careful clinical description of gout. He himself suffered from the disease for about forty years, and he has given a detailed description of his own case in the treatise which he published in 1683, under the title "Tractatus de podagra et hydrope." It was, however, Wollaston who, in 1797, threw the first light upon the peculiar anomaly of metabolism which exists in this disease. He demonstrated that the gouty deposits found in the joints and other parts of the body are mainly uric acid. From his time an all-important point with regard to the nature of the disease has been the relation between the symptoms of gout and disturbances in the manufacture and excretion of uric acid. In 1848 Garrod showed that in gout the blood contains an excess of uric acid, and that the excretion of uric acid by the kidneys is diminished. He was thus in a position to frame a theory consistent with all the clinical facts. Numerous investigations have been undertaken since his day; but we still remain with regard to gout in a position analogous to that which we hold toward diabetes. We are, it is true, in the possession of a considerable number of well-established facts relating to it, but we do not know why the normal chemical processes are disturbed, and we are unable to explain the connection between the various phenomena observed.

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* [Compare, however, the author's statements on page 752.—Trans.]

Clinical experience has taught us certain remote causes of gout, first of which comes heredity. About fifty per cent. of all cases occur in patients whose families have already suffered from the disease, or have given other indications of the uric-acid diathesis—such as renal calculi; and it is sometimes possible to trace this transmitted taint through many generations. It is decidedly more apt to pass down through the male members of the family than through the female.

Next in importance to hereditary influences is the mode of life. From time immemorial this has been regarded as an exciting cause of the disease. It has been a matter of universal belief that over-feeding, and especially the ingestion of too large a quantity of albuminoids, is strongly provocative of the disease. The same opinion has been held with regard to persistent over-indulgence in alcoholic beverages. Seneca relates that at the time of the decay of the Roman Empire women practiced such excesses that they were as subject to gout as the men, and an old verse runs: "Wine is the father of gout, feasting is its mother, and Venus is the midwife." That this old view of gout as a rich man's disease has much truth in it can not be denied. The not infrequent cases of genuine gout which we observe every year in Erlangen attack mainly great beer drinkers, who for years have imbibed three or four litres of beer a day; but, on the other hand, it would be going too far to refer all cases of genuine gout to an improper mode of life, and excessive indulgence in alcohol. Sometimes genuine gouty arthritis is seen in individuals who have always lived moderately, or who have even been in needy circumstances.

Besides alcohol, there is another well-known poison which is capable, by its chronic action, of producing that change in metabolism which forms the foundation of gout; we refer to lead. We are absolutely certain that persons who have much to do with lead, such as compositors and house-painters, suffer with comparative frequency from genuine gout, with deposits of uric acid in the joints. We have repeatedly seen cases of this sort in Leipsic and Erlangen. In a surprising number of patients lead-poisoning is associated with contracted kidney. We have repeatedly observed that the combination of chronic alcoholism and chronic lead-poisoning is especially provocative of gout.

With regard to all other alleged ætiological factors, confirmatory evidence is lacking. Possibly, however, when the foundation for the disease is already laid, an attack may be excited by certain determining causes—namely, trauma, taking cold, errors in diet, or mental emotion.

The geographical distribution of gout is remarkably unequal. The disease is more frequent in England than in Germany, although in the latter country certain regions appear to be more affected by it than others. In Leipsic gout seemed to us to be rather rare, but in Bavaria it is more common.

Gout is rarely seen in childhood and youth. It is a disease of middle or advanced life. Men are much more often attacked than women.

Clinical History.—Gout may produce symptoms in many different organs; but its effect upon the joints is so characteristic that the arthritic disturbance has long been termed "normal or regular gout," in contradistinction from "atypical, internal gout." This distinction is of course an artificial one, for the various phenomena of gout present the most manifold gradations and transitions. It will, however, be advantageous, in attempting to gain a practical insight into the various symptoms of the disease, if we first discuss the so-called "typical attack of gout," subsequently appending a description of the other manifestations of the disease. Furthermore, the regular attack of gout is, in at least a majority of cases (*vide infra*), the first and earliest symptom of the disease.

1. The typical attack of gout (primary articular gout) is sometimes abrupt; but it is often preceded for a longer or shorter period by certain premonitory symptoms, the meaning of which, though not evident to one who is about to suffer from his first attack, is sufficiently clear to more experienced patients, particularly as each individual case is apt to present a marked similarity in the prodromata of the separate paroxysms. These premonitory symptoms vary in different individuals. Sometimes they consist of dyspeptic disturbances; sometimes of a feeling of languor and mental depression; very often of dragging, muscular pains or cramps in the calves of the legs; or again of slight feverishness, with chilliness, sense of heat, and perspiration. These prodromes occur a longer or shorter time before the true attack of gout. On the other hand, it must be confessed that a patient may feel unusually well just before an attack.

The attack is noticeably apt to begin in the night-time, or very early in the morning. The patient is awakened by a sudden and very violent pain in the metatarso-phalangeal joint of one of the great toes ("podagra"). The joint becomes swollen, the skin over it red, hot, and tense, the veins in the neighborhood distended. At the same time there is chilliness and moderate fever (101°-102°; 38.5°-39° C.). This condition persists till morning. Then the pain is almost sure to abate, the fever to remit at the same time that sweating begins, and the patient to feel tolerably well during the day. The joint, however, remains swollen, with inflammation and œdema. The next night the pain begins again, and there is a fresh fever; and these alternations are repeated, as a rule, for three to seven days. Usually there are not many other symptoms present; the appetite is impaired, but ordinarily there are no marked gastric disturbances, such as eructations and vomiting, or intestinal symptoms. There may be a slight bronchitis. Even when the attack is more persistent than this, the pain is usually much less severe after the first two or three nights. After that time it gradually abates; and it is commonly said that an attack is brief in proportion to the violence of its first symptoms. When the pain ceases, swelling soon disappears, the skin undergoes a slight desquamation and resumes its normal appearance, the general health of the patient rapidly improves, and he is often found to be better after an attack than he was before.

For theoretical purposes (*vide infra*) it would be very advantageous to possess a more accurate knowledge of the condition of the urine, and in particular of the excretion of uric acid during the attack and at other times. As yet, however, there have been but few careful investigations made, and these have led to somewhat contradictory conclusions. Garrod has made a very important observation; it is, that the amount of uric acid excreted diminishes several days before the commencement of an attack, and remains diminished during the attack. Subsequently to the attack the excretion of uric acid is said to be above normal. In contrast with the excretion of uric acid in the urine, the amount of uric acid in the blood is increased during the attack. This is an important fact which was discovered by Garrod, and which has been confirmed by others, including Salomon, Klemperer, and others.

If there has been one attack of gout, there are almost sure to be others. They come sooner or later, at regular or irregular intervals, and separated by weeks, months, or even years. The attacks recur at long intervals in mild cases, more frequently and at gradually diminishing intervals in the severe. Spring and autumn are regarded as the time when attacks of gout are most apt to occur. In these subsequent attacks the great toe still remains the part most constantly and severely affected; but other joints may also suffer—for example, the wrist, the small joints of the fingers (see Fig. 82), the knee, or the shoulder. In all these joints there appears an acute and very painful inflammation, with considerable swelling of the affected parts. Sometimes traumatism apparently determines the

alization of the gouty disturbance. In each attack, the trouble is usually con-
ed to a single joint. It is only in exceptional or advanced cases that several
nts are simultaneously invaded.

The longer the disease has lasted, the less typical are the separate attacks.
ere may now be less suffering at the time of an attack; but the articular

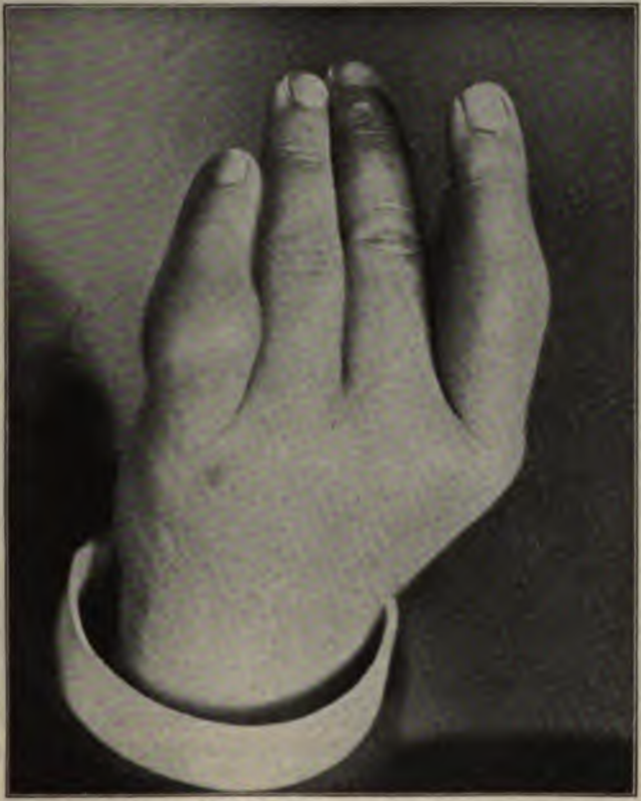

FIG. 82.—Acute attack of gout in the joints of the fingers. (Personal observation.)

inges are more persistent. The joints gradually become deformed. There are
nptoms referable to other parts of the body; and the gout gradually passes into
second chronic stage. Occasionally the disease is irregular and atypical from
incipiency; and the first manifestations may not be arthritic, but referable
the kidneys (*vide infra*) or other organs.

2. **Chronic Gout and the Gouty Diseases of Various Organs.**—As a rule, the
st striking results of chronic gout are the changes in the joints. There is a
dual development of marked deformity, particularly in the small joints of the
gers, the joints of the great toe, and less often the other joints. The fingers
e Fig. 83) are deflected toward the ulnar side, at the metacarpo-phalangeal
nts, as in arthritis deformans, and at the same time the fingers, as a whole, are
ckened, particularly at the joints. Almost always the small finger-joints early
dergo firm anchylosis. The great toes are often bent out of shape. Not infre-
ntly there are formed around some of the joints which are often attacked,
ticularly the metatarso-phalangeal joint of the great toe, soft fluctuating
sses, which are very painful. If these burst spontaneously, or if they are in-

cised, they are found to contain little, if any, pus, but much detritus, and numerous crystals of uric-acid salts, mainly the urates of sodium and calcium. When these nodes break open they may develop into tedious ulcers; similar gouty nodes (arthritic tophi) may develop in other portions of the skin, particularly in the lobe of the ear, and sometimes also in the tendons and muscles.

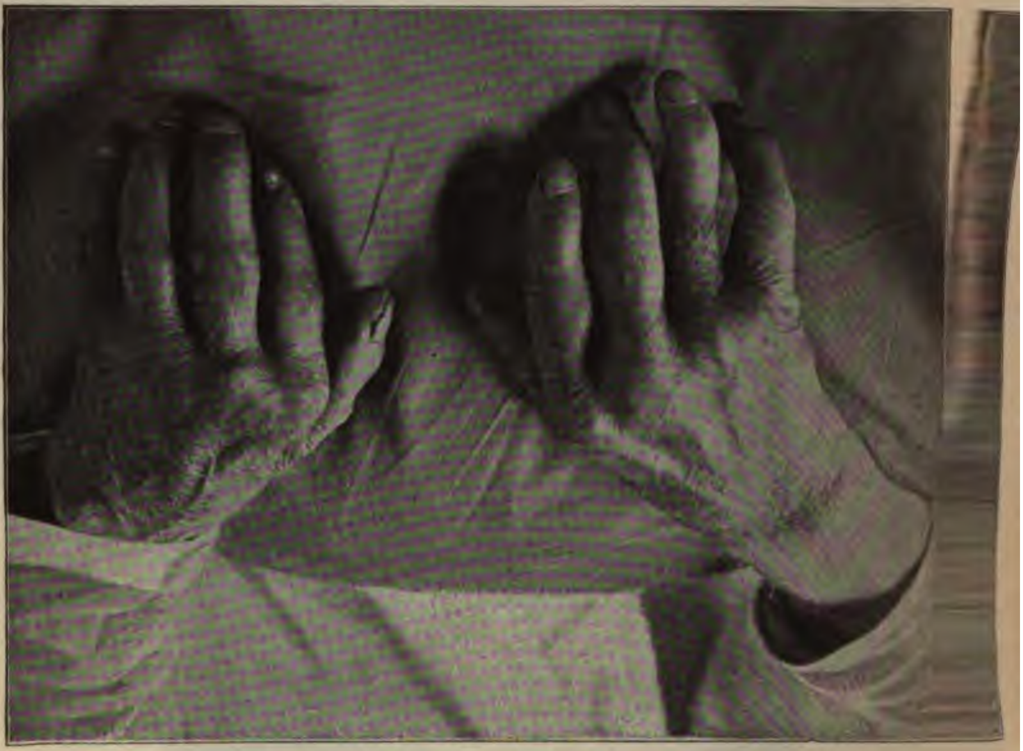

FIG. 83.—Changes in the joints of the fingers in genuine chronic gout. (Personal observation.)

Gout may affect the mucous membranes. A gouty dyspepsia is very frequent. Its symptoms are more or less severe. The gouty are also subject to intestinal catarrh of varying severity, and to bronchitis and conjunctivitis, as well as to catarrh of the urinary organs. Ebstein regards "gouty gonorrhœa" as essentially a catarrh of the excretory ducts of the prostate gland. It is not an easy matter to explain why these various forms of catarrh should occur in gout. Some of them may be complications. In other cases they are doubtless the result of passive congestion, due to cardiac failure (*vide infra*); but in still other instances it must be confessed that they are apparently due to the toxic influence of the accumulated uric acid or other noxious products of abnormal metabolism.

[My experience, though far more limited than that of Draper, nevertheless leads me to believe, as he does, that the irregular forms of gout are by no means uncommon in women as an inheritance from a previous generation. The manifestations in some of these cases are so ill-defined that a diagnosis may be difficult without a knowledge of the family history; in other cases, more or less deformity of the smaller joints, or slight recurrent swellings of those parts, throw much light on the condition underlying the symptoms.]

Inflammation of serous membranes—for example, of the pleura—also occurs;

and there may be pneumonia. The skin not infrequently suffers from acute or chronic eczema, which sometimes appears to be referable directly to the gout. Keratitis, iritis, and other inflammatory disturbances of the eye are also said to be caused directly by gout. Cirrhosis of the liver has been repeatedly found, and is probably to be referred chiefly, in our opinion, to chronic alcoholism.

By far the most important of all these gouty manifestations are referable to the kidneys and to the circulatory system. The disorder of the latter is sometimes symptomatic of the renal trouble, but in other instances it occurs independently of it. There may be severe gouty arthritis for years without any lesion of the kidneys; but this is exceptional. In severe cases of gout it is the rule that, sooner or later, symptoms of renal disorder present themselves. The so-called "gouty kidney" is a form of chronic interstitial nephritis. However important this complication, its symptoms do not need to be discussed here, as they are precisely similar to those seen in ordinary cases of contracted kidney (*vide* page 628 *et seq.*). The distinctive symptom of this disturbance is albuminuria; and the gradually developing secondary hypertrophy of the left ventricle is the pivot on which turns the whole future course of the disease. So long as the heart remains capable of performing its functions, the condition of the patient will probably remain endurable if not actually comfortable. Finally, however, compensation is sure to become impaired; and then appear œdema, dyspnœa, debility, and emaciation—in short, all the familiar symptoms of cardiac failure. A speedy end may be brought about by uræmia, cerebral embolism, or cerebral hæmorrhage; but in other cases the patient suffers for years, both from the heart disease and from fresh arthritic attacks.

The cardiac hypertrophy just mentioned is the result of the contracted kidney. Other disturbances of the circulatory system appear to be referable directly to the gout itself. Among these are chronic endocarditis or myocarditis, and perhaps certain "functional" symptoms, such as palpitation and angina. An important symptom is chronic endarteritis or arterio-sclerosis. This is often seen in gouty subjects, and in many instances seems to be connected with the gout. Sometimes, also, there are gouty lesions of the veins, such as varicosities or thrombosis. Of course, these changes in the blood-vessels may, in their turn, give rise to various disorders.

In a few very rare instances gout seems to attack the brain and spinal cord. Usually, however, the nervous disturbances seen in gout are, as already stated, symptoms of uræmia, or of circulatory disturbance, arterio-sclerosis of the cerebral arteries, and the like. The patient may also have certain functional nervous troubles, such as neuralgia or migraine. The direct cause of these is seldom evident.

Complications of Gout.—Apart from chance complications there are several morbid conditions which undoubtedly may stand in close relation to gout. Among these is, first, the formation of renal calculi, or gravel (*q. v.*). This has often been observed in gouty persons. Another not very infrequent combination is gout and diabetes mellitus; ordinarily, attacks of gout appear first, and later glycosuria develops. Often gout and diabetes seem to alternate. The connection between the two affections seems to us to be that both diseases result from the same cause (chronic alcoholism, less often heredity), so that they develop together, or one after the other. Equally obvious is the explanation of the frequent association of gout with obesity, arterio-sclerosis, etc.

Anatomical Changes in Gout. Theory of its Nature.—The essential anatomical lesion in gout consists in an abundant deposit of crystalline urates in the tissues. This is most evident in the affected joints, the cartilaginous surfaces of which are often completely covered with white, chalk-like material. In severe cases the same appearance may also be presented by the articular ligaments, tendons, and bursæ, while there are at the same time numerous concretions of urates here and there beneath the skin. These deposits are composed mainly of acid sodic urate, with traces of calcic urate, calcic phosphate, and sodic chloride. However certain it is that these abnormal deposits of uric acid should be regarded as the pathognomonic sign of gout, we are absolutely without knowledge as to their origin and intrinsic importance. One question in particular remains unsolved—whether the deposits of uric acid excite the inflammatory symptoms, or whether, conversely, the deposits of uric acid are secondary and are formed in tissues already attacked by the disease. Ebstein believes that the process of the precipitation of uric acid is invariably preceded by a tissue necrosis. The uric acid while still in solution acts as a chemical irritant upon the cartilage here and there, and thus produces necrosis, whereupon the urates are crystallized out and deposited; but this view has not escaped contradiction, and possibly the primary damage in gout is due not to the uric acid itself, but to some other noxious substance.

The nephritis of gout corresponds in its pathological appearances to that of the true contracted kidney, with one exception; the organs usually present deposits of uric acid or urates, in stripes, both in the lumen of the urinary tubules and also in the epithelial cells and the interstitial tissue. In the connective tissue it is probable that some lesion precedes the deposit.

The lesions presented by the heart, blood-vessels, and remaining parts of the body are not especially characteristic of the disease. The blood of gouty subjects, as Garrod demonstrated, contains an excess of uric acid.

With regard to the true nature of gout our knowledge is still more limited and unreliable than in the case of diabetes. The earlier view was that in gout an excessive formation of uric acid took place because of the incomplete oxidation of uric acid into urea. This view has been losing ground, because of late it is assumed that uric acid is not at all an end-product of the ordinary metamorphosis of albumen, but that it is the specific product of the metamorphosis of nucleine, which is the special form of albumen contained in the cell nuclei; thus uric acid resembles the alloxur bases—that is, xanthine, hypoxanthine, etc. Upon the ingestion of ordinary albumen the uric acid is not increased, but it is increased by food rich in nucleine—for example, liver or sweetbread. The formation of uric acid seems, however, to be particularly favored by the destruction of the nucleines already organized in the body, and especially the nucleated white corpuscles, but whether there is in gout an actual increase in the amount of uric acid, or whether merely a normal amount is formed and its excretion through the kidneys is hindered, we are entirely unable to say. Many authors have assumed that the blood is less alkaline than normal, and consequently uric acid is deposited in the tissues; but the most reliable investigations do not indicate any marked change in the alkalinity of the blood. In brief, the true riddle of gout, the special rôle which uric acid plays in this disease, is as yet unsolved.

Diagnosis.—It is usually easy to recognize an acute attack of gout. The sudden onset of the pain and its localization in one of the great toes are very characteristic symptoms, and render it easy to distinguish between it and other acute affections of the joints. When an acute attack of gout affects other joints, we have a sudden development of marked swelling and tenderness in one single place, and a comparatively rapid abatement of the inflammation, while in acute articular rheumatism the disease attacks several joints, and moves quickly from one to another; so that usually the two diseases can be readily distinguished. The diagnosis is more difficult in advanced stages of the disease, in which the symptoms are more obscure, but in such cases we can usually obtain, by careful questioning, an account of the previous occurrence of typical gouty paroxysms and the existence of certain ætiological factors, such as heredity, mode of life, or chronic poisoning from alcohol or lead, which are of considerable diagnostic importance. In chronic articular gout the main question is its diagnosis from ordinary arthritis deformans. The external appearance of the diseased joints is not of itself decisive, although the gouty arthritis leads to greater and more diffuse enlargement, especially in the fingers, and much oftener occasions complete anchylosis in individual joints than does arthritis deformans. The most important means of diagnosis, however, in every case, are a careful inquiry into the previous history with regard to typical attacks of gout, and a consideration of the characteristic gouty changes—such as tophi breaking open. We should also bear in mind, with regard to differential diagnosis, the chronic arthritis of gonorrhœa.

If there is chronic nephritis of gouty origin, its source can be recognized only from its association with other indubitable symptoms of gout, particularly typical gouty paroxysms, unless possibly the knowledge of certain ætiological factors, such as a history of gout in the family or of chronic lead-poisoning, should put us upon the right track. Ebstein has reported cases of "primary renal gout" where there was no arthritis during the whole course of the disease. Such cases can seldom be correctly diagnosticated during life.

Brief mention should here be made of an experiment performed by Garrod, which may be employed to demonstrate the existence of uric acid in the blood, for diagnostic purposes. A drachm or two of blood-serum, or of the serum obtained from a fly-blister, is put into a shallow watch-glass, and six or eight drops of a thirty-per-cent. solution of acetic acid are added to it. A linen thread is then laid in the fluid, and the whole allowed to remain at a moderate temperature for about a day. If the proportion of uric acid in the fluid is sufficiently large, crystals of that acid will now be found on the thread and may be recognized by their shape and chemical reaction. This "thread test" of Garrod's is not extensively used, because it does not succeed unless there is a comparatively large amount of uric acid in the blood, and, furthermore, uric acid may exist in the blood in health, or in other diseases than gout.

Prognosis.—However favorable the prognosis may be with regard to the single gouty attack, yet a permanent release from the disease is rarely to be hoped for. Only such patients as, from the first appearance of gout, pursue most carefully all the requisite prophylactic and dietetic measures, can expect that future attacks will be rare and comparatively mild, and that severe lesions of the internal organs will not occur. So long as the kidneys and other viscera are intact, there is no immediate danger to life, and the patient may attain advanced years despite his gout. The gradual and chronic alterations in the joints may, however, impede locomotion as well as all other movements of the body. Except for this, the general condition of the patient in the intervals between the attacks is often one of tolerable comfort. Indeed, it is frequently the case that the patient will feel at his very best directly after a severe paroxysm, while rudimentary or atypical attacks are often regarded as an ill omen. Really, however, there is no serious danger until chronic nephritis has developed. The prognosis then becomes as unfavorable as in the other forms of contracted kidney (*q. v.*), and involves the same possibilities.

Treatment.—All authorities agree that the first essential in treating gout is a proper regimen. Unless the patient has energy enough to yield the most implicit obedience to the injunctions regarding diet and mode of life in general, from the first appearance of his disease, no great benefit can be obtained from treatment.

Various authorities have, of late, prescribed special diets for the gouty. The various and almost contradictory theories as to the nature of gout have led to extremely diverse dietetic injunctions. If we cling to simple practical experience, the general conclusion for centuries has been that we should enjoin upon gouty subjects moderation in everything, most of all in alcoholic beverages, and in meat.

We should advise, therefore, that the total amount of meat eaten should be limited, and that so-called "white" meat (veal, mutton, fowl, fish) should, on the whole, be preferred to "red" meat (beef and game). The cellular organs (liver, kidneys, sweetbread, brain) are now usually forbidden, out of regard to the above-mentioned views as to the formation of uric acid. Milk and eggs may be allowed, fat and carbohydrates are to be given freely, or to be limited, according to the general nutrition of the patient. Gouty subjects who are obese should have less of such articles of diet, while emaciated patients should be encouraged to partake of them. Green vegetables that are easily digested, such as spinach, salad, kohl rabi, cauliflower, Brussels sprouts, and asparagus, have long been regarded as especially suitable. The most appropriate beverages are water, milk, tea, and coffee, also the ordinary mineral waters, Seltzer, Giesshübler, and lemonade. Fruits, especially when cooked, are usually permissible and to be recommended. Such alcoholic beverages as beer and wine are to be allowed in very small quantities, if at all. In general, we should not be schematic and theoretical in our dietetic prescriptions, but should consider the individual. The quality of the articles of food eaten is, on the whole, usually less important than the total quantity.

[Many gouty persons, especially those suffering from the irregular form of the disease and acid dyspepsia, are more comfortable and seem to do better on a diet which is largely nitrogenous, the starches and sugars being greatly limited in amount. The diet, in fact, should be similar to that laid down for diabetes mellitus, though not so strictly carried out. In chronic and irregular cases it is often desirable to prescribe a stimulant, for a time at least; brandy, whisky, or gin, well diluted with water, is perhaps the best form.]

By thus limiting the amount of food taken, we shall avoid any excessive formation of uric acid, and perhaps other harmful substances; but a direct means of promoting metabolism is muscular exercise. If a corpulent patient is still vigorous and is not threatened by any serious internal disease, he should be urged to take a large amount of exercise in mountain-climbing, gymnastics, gardening, or similar pursuits. This same indication of accelerating tissue-metamorphosis is fulfilled also by bathing. In early stages cool baths and sponging are useful, as are also baths containing common salt, or perhaps even sea-bathing cautiously employed. In more advanced cases, particularly if the joints present permanent lesions, the warmer baths are more desirable, such as are found in Wiesbaden, Baden-Baden, Teplitz, Ems, and Aix.

The internal administration of alkalies is an efficient adjuvant to the dietetic and hygienic prescriptions above enumerated. Although there may be doubt about the correctness of the ordinary theoretical basis for this prescription—viz., that alkalies prevent the deposit of uric acid in the tissues—yet simple practical experience justifies the employment of suitable mineral waters. These waters stimulate the conversion of the albuminoids, increase the activity of the kidneys, benefit gastric catarrh, and overcome constipation, and in all these ways combine to produce a favorable effect upon the patient's health. Still another factor is the judicious diet and mode of life at such health-resorts. The waters of Carlsbad and Vichy have gained the greatest repute in gout, although the waters of Ems and Neuenahr have an analogous composition, and doubtless they would produce similar effects. The same is true of Kronenquelle and Fachinger-water. Most of these waters can be taken very well in the patient's own home. The salts of lithium especially promote the solution of uric acid, and the waters which contain lithium have been strongly recommended. The natural springs of this sort, such as the Crown Spring in Obersalzbrunn, and those of Assmannshausen and Salzchlirf, contain comparatively insignificant amounts of lithium; and it might be a better way to use the artificial lithium waters, such as are made by Struve or

Ewich. Another way is to prescribe carbonate of lithium, in powders containing two or three grains each (gramme 0.10–0.20), one powder two or three times a day, in a glass of Seltzer or Biliner water.

As to the treatment of the acute attack, it has ceased to be customary to employ any potent remedies. The patient must, of course, keep his bed. The affected joint should be wrapped up in cotton wool, the whole leg elevated, and a proper diet strictly enjoined. Free movement of the bowels should be maintained by means of enemata. If there is considerable gastric disturbance, bicarbonate of sodium, magnesia, or some bitter may be prescribed. The most certain remedy for severe pain is a subcutaneous injection of morphine. Less efficient are narcotics locally applied, and warm compresses. Whether any internal remedies are calculated to abbreviate the attack is doubtful. Formerly colchicum (twenty or thirty drops of vinum colchici seminis three or four times a day) was the favorite medicine, but it seems to be going out of use. Salicylic acid and salicylate of sodium may be administered in the same way as in acute articular rheumatism, and sometimes, although not always, they are followed by improvement. Antipyrine, salipyrine, salol, and similar remedies sometimes succeed in allaying the pain in the joints. Some physicians recommend subcutaneous injections of antipyrine in the neighborhood of the joint, but we have no personal experience on this point. On the basis of dubious theoretical assumptions, a number of drugs have been recommended recently which possess the power of dissolving large amounts of uric acid. Prominent among these are piperazine, of which about eight grains (gramme 0.5) are given daily in soda-water; and lysidine, fifteen to seventy-five minims (cubic centimetres 1–5) of a fifty-per-cent. solution daily. Many physicians report favorable results with these remedies; others, on the contrary, have found no virtue in them.

[One reason why colchicum has fallen into relative disuse is that the relief obtained from it is often so prompt and complete that patients are tempted into continued indulgence in a faulty manner of life. It is also supposed by some that the drug interferes with the effort of nature to eliminate the poison, which becomes generally diffused, and sets up changes in the vessels and internal organs. Precisely how colchicum acts we do not know, but that it does act, and sometimes with marvelous success, there can be no question.]

The chronic affections of the joints in gout are treated as are other varieties of chronic arthritis. The most efficient remedies are cautious massage and baths, including hot sulphur baths and mud-and-sulphur baths. The internal administration of the alkalies, lithium, and similar drugs, to combat the general gouty diathesis, should be associated with these external remedies. Some physicians report that iodide of potassium favors the absorption of the gouty deposits.

The treatment of the nephritis and other complications need not be discussed at length. The gout itself should always be the main object of our therapeutic efforts, and beyond this we may be guided by general principles.

CHAPTER XII

OBESITY

(Corpulence. Polysarcia adiposa)

Definition and Ætiology.—The amount of adipose tissue in the body is subject to considerable variation, and it is not possible to state absolutely what should be considered as normal and what as abnormal. For practical purposes we may draw the line where the increased size grows burdensome to the individual. After a certain point, any further addition to the amount of fatty tissue is almost sure to work serious injury, and is therefore to be regarded as an actual disease, and not merely an inconvenience. It should be said, however, that in such cases the symptoms of obesity are very frequently confounded with those springing from other disorders—these latter possessing, indeed, the same ætiology as obesity, but distinct from it.

The most frequent and important cause of obesity is the habitual ingestion of too large an amount of food. By "too large" is meant an amount sufficient to occasion a continual increase of the adipose tissue of the body, when this is already fairly well developed. It is a matter of indifference whether the excess is composed mainly of albuminoids, carbohydrates, or fats; for in either case, if the quantity be sufficient, an increase of adipose tissue may take place. We shall soon see, however, that the excess is usually in fat and carbohydrates. Inasmuch as this overfeeding is almost certain to be habitual, the excess at any one time need not be at all large. We often hear a corpulent person express his surprise that he grows heavier every day, although he "does not eat any more than others who are lean." The explanation is easy if we consider that a daily increase of five grammes of fat (one and one fourth drachm) suffices to increase the weight in ten years—for example, between the thirty-fifth and forty-fifth years—forty pounds avoirdupois. In reality the daily increase is sometimes greater than this.

The basis for a detailed consideration of the causes which lead to the deposit of fat will be gained by a consideration of the physiological laws relating to nutrition discovered by Voit, Pettenkofer, and their pupils. It has been shown that both the albuminoids and the carbohydrates of the food may be a source of fat, formed within the economy, and also that the fat contained in the food may be to a large extent directly deposited in the fat-cells of the body. One product of the decomposition of albuminoid substances is invariably fat. This usually undergoes further oxidation, but it is sometimes retained unaltered in the system. It would even seem that the albuminoids give rise to much more of the fatty tissues of the body than are produced from the carbohydrates ingested, although there is no doubt that these latter may also yield fat. Carbohydrates do promote obesity, but less because they are a direct source of fat than because they are easily decomposed, and so shield both the ingested fat and that which is formed out of the albuminoids from oxidation. In this indirect way they do greatly favor a tendency to corpulence.

We thus perceive that various diets may, each one of them, permit of an increase of adipose tissue. In actual life, of course, the most frequent conditions are such as result from the customs and habits of the population in general. The diet is almost invariably a "mixed" one—that is, it contains albumen, fat, and carbohydrates—and in most instances the obesity is due to an excessive amount of all three elements, or at least of the fat and carbohydrates. A person may, however, become corpulent who eats very little fat, provided he consumes a large quantity of albuminoids and carbohydrates; or if he eats very little starchy food, provided he consumes a large amount of meat and fat. Perhaps these facts may be made clearer by giving a concrete example. Voit tells us that a vigorous man who requires daily 118 grammes of albumen and 259 grammes of fat to maintain a physiological equilibrium as regards fat and albumen will, other things being equal, begin to store up fat if there is any further increase in the amount of fat in his diet. The same result will also take place if, instead of the rations previously stated, he ingests more than 118 grammes of albumen and 600 grammes of starch, or more than 664 grammes of albumen alone, or, finally, more than 118 grammes of albumen, 100 grammes of fat, and 368 grammes of starch.[*] It is obvious that this last diet, which closely resembles the average diet of an adult[†] in good circumstances, whose weight is neither increasing nor diminishing, is the one most likely to be exceeded; whereupon there must take place a deposition within the system of the superfluous fat.

Among the various kinds of food is one group which deserves mention, as being an important factor in many cases of obesity; we refer to alcoholic beverages. There can be no doubt that intemperance in this regard plays a prominent part in many instances. Sufficient illustration is furnished by brewers, hotel-keepers, and the inhabitants of countries like Bavaria, where beer-drinking is prevalent. In this particular it is evident that beer works more harm than does wine or strong liquor; for beer contains, in addition to the alcohol, an appreciable amount of starchy material, making the sum total from the beer drunk during the entire day a considerable one. Many persons who would be extremely indignant if called "tipplers," habitually take five or six glasses of beer a day, equivalent to 150 grammes of starch, or, in other words, to one half the total amount of starch required by the system. Even this quantity is frequently exceeded. Of course, the three or four per cent. of alcohol which the beer contains also promotes the deposition of fat. Alcohol is readily oxidizable, and it thus shields to a considerable extent the fat already present in the body; and it also, in all probability, works such injury to the tissues as to diminish their power of effecting decomposition. We must also remember that most beer drinkers take little exercise. The long sitting at the beer table, and the mental and physical sluggishness which immoderate indulgence in beer always causes, and finally the increasing corpulence itself, explain why most beer drinkers dislike protracted exercise.

We have thus seen that in by far the larger number of cases obesity is mainly due to the ingestion of too much food. No weight need be attached to the usual statement of corpulent persons, that they eat no more than others. Few of them have any idea how much nourishment they do consume. Others, having already become corpulent, eat less, to be sure, than they used to, but nevertheless an amount sufficient to maintain the acquired weight.

Other factors may no doubt exert an influence upon the increase of adipose tissue by diminishing the consumption of fat in the system. The most important factor of this class is physical inactivity. Muscular contractions lead to the decomposition of a large amount of fat. This explains why people of sedentary habits, and those who sleep long and exercise little, are more apt to become corpulent than are manual laborers. Again, there are certain diseases which seem to promote corpulence. In anæmia there is sometimes a striking tendency to obesity, due in part to the diminished supply of oxygen and in part to diminished muscular activity. This same inactivity is probably the main cause of corpulence in paralysis (hemiplegia). It may be, however, that disturbances of the nervous system may directly affect metamorphosis. Thus, idiots and other subjects of congenital defects of the brain are liable to obesity. Disturbances of the circulatory system seem to favor the production of corpulence by impairing oxidation. This is seen in many young persons with cardiac disease, although it is not easy to exclude in this case the influence of still other factors, such as a sedentary life.

Finally, some cases of obesity seem to result from a constitutional and inborn predisposition. Young children sometimes suffer from obesity; and the condition seems in many cases to be hereditary. Many races and nations exhibit an especial tendency to corpulence—for example, the Jews. Age and sex have some importance in this regard: extreme obesity is seldom seen previously to the thirtieth year; and women appear to be somewhat more subject to the disease than men. The importance of a "tendency" to obesity should not be overrated. Upon careful investigation, we shall almost invariably find in the habits of the individual, as regards food and exercise, a satisfactory explanation of his obesity. Still, it may be possible that upon accurate examinations into metabolism it would be found that the power of fat-combustion is subnormal in many persons, analogous to the impaired combustion of carbohydrates in diabetics. This diminution of the oxidizing function might be due to congenital or acquired anomalies. If our supposition prove correct, it might explain the not infrequent combination of obesity and diabetes (*q. v.*), or obesity and gout. Strictly speaking, the condition can not be regarded as a disease unless the habits as to diet and exercise fail to account for it.

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[*] This statement is founded upon an important discovery of Rubner, that, as far as the storing up of fat is concerned, the measure of value for different foods is the amount of heat given off by them when they undergo oxidation into carbonic-dioxide gas and water. Measured in this way, 100 grm. fat = 211 grm. albumen = 232 grm. starch = 234 grm. cane-sugar = 256 grm. grape-sugar.

[†] Probably the amount of fat contained in the food is often less than the above, and the amounts of albumen and starch somewhat larger. Voit estimates the diet of a well-to-do person at 127 grm. albumen, 89 grm. fat, and 362 grm. starch; and that for a vigorous laborer at 118 grm. albumen, 56 grm. fat, and 500 grm. starch. Of course, these figures are merely approximate.

Pathology.—After corpulence has passed a certain point the condition is evident at the first glance. The subcutaneous cellular tissue is one of the chief places in which the fat is deposited. Consequently, the panniculus adiposus soon attains considerable thickness. The countenance grows more round and plump; beneath the chin is formed a second prominence known as the "double chin"; the chest appears broadened; the waist enlarges; and, particularly in women, the breasts are changed to great shapeless masses, over which the skin is so tightly stretched as to present lineæ albicantes. The abdominal walls are greatly altered. The belly projects more and more until it becomes actually pendulous, and its lower surface touches the interior surface of the thighs. Intertrigo is apt to occur in the groins, below the breasts, and between the buttocks. The skin everywhere has a fatty feel, due to the increased secretion of the sebaceous glands. This hyperplasia of the fatty tissue in the panniculus adiposus is associated with a deposit of fat in many parts of the interior of the body, including the mesentery, mediastinum, pericardium, and the capsules of the kidneys. Some of these will be mentioned again further on.

Of course, the circumference and weight of the body become greatly increased. As an approximate measure it may be stated that for men of middle height a weight exceeding ninety kilogrammes (200 pounds), and for women seventy-five kilogrammes (165 pounds), may be regarded as abnormal.* This increase in bulk is the first cause of subjective symptoms. An obese person has to exert a greater effort in making any motion than do other people, and as a necessary consequence he gets easily tired, and seeks as far as possible to avoid exertion. The increased demand upon the muscles explains the familiar fact that corpulent persons perspire readily.

The graver symptoms of obesity, and properly the first pathological phenomena of the condition, relate to the respiration and the circulation. The patient begins to complain of shortness of breath, and is subject to marked dyspnœa upon running or climbing stairs. There may be, at the same time, cardiac disturbance, indicated by a rapid pulse, palpitation, intermission of the pulse, or other slight irregularities in cardiac action. All these symptoms grow gradually worse; and to them are added symptoms of cardiac failure and consequent passive congestion. There is a tendency to bronchitis and other catarrhal troubles. The appetite and digestion are affected, and œdema appears.

A careful analysis of all these symptoms shows that many causes combine to produce them, all having a common tendency to impede respiration, and, still more, circulation. One source of disturbance is the increased deposit of fat upon the framework of the body. It is probable that the excess of adipose tissue covering the thorax exerts a direct influence in obstructing the respiratory motion of the thoracic walls, and renders the respiration more superficial. In this way the return of venous blood to the heart and the pulmonary circulation are both impeded, because the negative pressure in the chest is less than normal. Likewise the diminished amount of bodily exercise affects the circulation unfavorably. Brauner has shown how numerous are the arrangements connected with the fasciæ for promoting the venous currents, by means of the negative pressure resulting from the movements of the body. Whether the fat deposited around the heart directly obstructs the cardiac movements is somewhat doubtful, though this view is held by many. The fatty infiltration of the myocardium is of more importance: the fat is deposited in the intermuscular connective tissue. Still, it is doubtful whether this condition, which is not so very frequent, after all, occurs as a primary change, or whether it is not rather a result of previous atrophy of the myocardium (compare the chapter on fatty heart).

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* A weight of even 150 kilogrammes (330 pounds) has been repeatedly observed.

There is no doubt that, in almost all cases where the corpulence actually induces grave disturbance, the cardiac symptoms are of prime importance. These are due, as has just been indicated, in part to the increased amount of adipose tissue, and in still greater part to complications, most of which are excited by the same causes as is the obesity itself. The abundant adipose tissue may obstruct the circulation in the smaller blood-vessels and capillaries inclosed within it. Furthermore, the excessive development of fatty tissue probably leads to the growth of new blood-vessels, and, as a consequence, to an increase in the total volume of the blood. This is one way in which the demands made upon the heart are rendered greater than normal, and explains why the corpulent frequently exhibit cardiac hypertrophy. Other influences are also at work to produce this same result: they are, in the first place, the same factor which occasions the obesity itself, namely, the ingestion of increased amounts of food and drink (see the chapter on cardiac hypertrophy), and, secondly, certain other lesions which are frequently associated with obesity and are referable to the same causes as it is. Chief among this latter class is general arterio-sclerosis. If this involve the coronary arteries, it may in turn occasion still further damage—for example, degenerative myocarditis (compare page 341). Chronic interstitial nephritis is another not infrequent complication. This is referable in part to the same causes as is the obesity. Gout and diabetes are less frequent.

We thus see that obesity is often merely one of many injurious results occasioned by an improper mode of life. It is, in a certain sense, the first danger-signal, warning the patient and his physician to avoid the graver disturbances which threaten. This is a point of great practical importance; for, when once we have a combination of obesity with cardiac hypertrophy, fatty infiltration of the heart, arterio-sclerosis, or interstitial nephritis, the various causes and effects act and react upon one another in a way most perilous to health, and even to life. There is no need of describing the grave disturbances which invariably develop at the close of the scene. They are the result of chronic cardiac insufficiency, and have been fully described under cardiac disease.

In every case of obesity the physician should examine the heart, lungs, vascular system, and kidneys, particularly if there be already subjective disturbances. The examination of the heart may present considerable difficulties, because the results of palpation and percussion are so much obscured by the thick cushion of fat which covers the thorax. We can, however, have recourse to auscultation, and can feel the pulse. The pulse may be either rapid, slow, or irregular. We need not mention any minute particulars as to the examination. It may be stated, however, that a hepatic enlargement is often found, but it is much less often the result of fatty infiltration than of simple hypertrophy or passive congestion.

We have thus seen that corpulence may sometimes be associated with grave and dangerous lesions; but, on the other hand, it should be stated that this unfortunate condition by no means invariably exists. Not infrequently the corpulence remains moderate, in which case it is not really dangerous, however inconvenient. This is true of a proportion of those cases which are due to the ingestion of a large amount of nourishment, associated with defective oxidation, and where there are no other unfavorable influences at work. The obesity of hard drinkers is almost always a more or less dangerous condition, while that seen in many elderly persons and in women is often comparatively free from peril. These latter individuals are, to be sure, discommoded by their great weight, they can accomplish less than they used to, they are easily put out of breath, and have a certain tendency to catarrhal inflammations and rheumatic difficulties; but they escape the severer lesions above enumerated. Even these apparently harmless conditions should not be disregarded by the physician, as he can never be absolutely certain that grave complications may not be developed eventually.

Treatment of Obesity.—To cause the disappearance of the accumulated fat, it is necessary to promote its oxidation in the system and to prevent the ingestion of new supplies of fat. To accomplish this purpose we possess only two means—first, a limitation of the ingestion of such kinds of food as may lead to the formation of fat in the system, and, secondly, stimulation of muscular activity and other factors which occasion the destruction of the fat already present. All the various methods of treating obesity, without exception, aim either to diminish the supply or to increase the destruction of fat.

The methods vary greatly. It must be borne in mind that the diminution of the adipose tissue must not involve injury to the body itself. The treatment should not weaken the constitution, but should invigorate the patient, or at least be innocuous.

It is of prime importance, in every method of treatment, that the total quantity of ingested food should be diminished. It is a mistake to forbid the patient some particular kind of food—for instance, the carbohydrates or the fats—with the idea that they alone do harm, or to allow him unlimited quantities of other kinds of food, in the belief that they are harmless. Any person can eat albuminoids, fat, and starch at every meal, and yet not grow fat; while, on the other hand, too much of any one of these may lead to an increase of adipose tissue. The amount of food which a person can take without increasing the amount of adipose tissue varies with the individual. It depends upon the amount of material already present in the body, and upon the various demands made upon the system, as well as other factors. This renders it difficult to draw up a universal dietary for the obese. We can best judge of the value of any course of treatment by its results, and these are best measured by the weight and the subjective condition of the individual patient.

Of the various elements of food, the albuminoids should be diminished least of any, because it would be sure to work injury to the system if they were supplied in too small an amount. Of course, the albuminoids must not be eaten to such an extent that the fat into which they decompose remains intact in the body. An increase in the amount of nitrogenous tissue is, however, desirable, because this promotes the vigor of the muscles and the heart, and so leads to the oxidation of larger quantities of the non-nitrogenous tissues.

The amount of fat and starch must be much more limited. The fats and starches are more potent in increasing adipose tissue and in shielding from oxidation the fat already stored up in the body than is nitrogenous food. It would not be at all advisable to forbid the use of either one of these two constituents of a mixed diet, allowing the other alone to be eaten. A varied diet is extremely desirable, even for one who is corpulent; and we should exclude neither fat nor starch wholly from the dietary, but we should merely limit the amounts to be taken. As already indicated, the amount of albuminoid food remaining unchanged, a person can eat double the quantity of starchy food that he can of fat, without increasing his adipose tissue. It is therefore irrational to allow the corpulent fat in larger proportions than starchy foods. The diet which Ebstein has recently proposed for the treatment of obesity does prove successful, but the explanation of its success lies in the comparatively small amounts of meat and fat ingested. Precisely the same results would be attained if a corresponding amount of starch were substituted for all or a portion of the fat; and in practice it is desirable, at least in most cases, to allow the patient both starches and fats. Of course, the likings and experience of the individual should be considered in each separate case. The Banting treatment, introduced in 1864, enjoyed for a time a great reputation. Its inventor applied it first of all to his own case, and with success. It rests upon a rational basis, inasmuch as the albuminoids are allowed in abundance, and the ingestion of fat and starch is limited. It lays too much stress, however, upon the exclusion of fat as compared with starch. Of great practical importance in many cases is the strict prohibition of beer drinking. Many cases of obesity are due merely to the excessive amount of carbohydrates contained in the unreasonable quantity of beer drunk daily. If corpulent beer drinkers of this sort change their mode of life in this single particular, and drink no beer whatever for from three to six months, they are almost sure to lose a great deal of weight.

The physician who bears in mind the principles just expounded can lay down his own rules for the diet of his patient. As already stated, it is impossible to give figures which will apply to every case. If we take as a basis the average diet for an adult—that is, about 125 grammes of albumen, 80 grammes of fat, and 350 grammes of starch—we might say that most cases of obesity would be sure to undergo improvement upon a diet containing 125 grammes albumen (or possibly even more than this), 40 grammes fat, and 150 grammes starch. The amounts of fat and starch could be even more diminished, but it is usually best not to be too precipitate. A gradual diminution of two or three pounds a week, extending over a long period without interruption, is to be preferred to the rapid treatment common at many health-resorts. Of course, the loss of fat is greater at the commencement of treatment than later on, when the amount of adipose tissue has already approached more nearly to normal, and the diet must undergo a gradual and corresponding change. It is of particular importance to increase the amount of non-nitrogenous foods in the later stages of treatment, lest the albuminoid tissues of the body become wasted.

The following dietary may be taken as an illustration of what would be suitable for a patient in the beginning of treatment: For breakfast, a cup of coffee with milk, and about 75 grammes of bread. At noon, a plate of soup, 150 to 175 grammes of lean meat or fish, lettuce, green vegetables, and about 25 grammes of bread. For dessert, about 75 grammes of boiled rice, or some simple pudding, or 100 grammes of fruit. To quench the thirst, water, or half a pint of light wine. In the afternoon, a cup of coffee, and with it not more than 20 to 30 grammes of bread. For supper, two eggs, or 100 to 120 grammes of meat, with 30 grammes of bread, a little fruit, lettuce, half a pint of wine, or one or two cups of tea, not much sweetened. Butter should be entirely proscribed at first; later on it may be used in small amounts.

Some approach to this bill of fare must be enforced not merely for a few weeks, but for months. It is absolutely necessary that the patient should be weighed ery two or three weeks. If the weight diminishes slowly and gradually, with ut any subjective disturbance, we have the best proof that the diet is a suitab ne. If the weight does not diminish, then the amount of ingesta must underg urther reduction. If more food can be taken without the weight increasin gain, a larger amount may be unhesitatingly permitted, and indeed may eve be advisable if the patient be languid. The increase should at first, howeve be mainly in the amount of albuminous food, the amount of starches and fa not being much increased. The "cure" can not be regarded as complete un the weight has been brought down to that of the average individual of t given age and sex. This goal having been reached, greater freedom in diet permissible.

The object of the treatment just suggested is exclusively the limitation of production of fat. We may also promote the destruction of the fat already sto up in the system. A chief means to this end is muscular exercise, which undou edly increases the oxidation of the adipose tissues. Carried out in a proper ma ner, it is therefore a most valuable adjuvant to treatment. Oertel has recent pointed out that muscular exertion does good in still another way—namely, b promoting cardiac activity and inducing deep respiratory efforts. Thus the hear is strengthened and circulation promoted. Mountain-climbing is one of the bes modes in which to take the desired exercise. We need hardly say that the increased muscular activity makes it possible for the patient to take an increased amount of food without injury. In dealing with cases of obesity in which the condition is due less to overfeeding than to lack of muscular exercise, it might be decidedly advisable to lay the most stress upon the increase in muscular activity; for certainly it is more advantageous for the organism to get rid of its excess of fat by means of lively metabolism, while nutrition is well maintained, than to be more or less starved by an excessive limitation of diet. We should not rest satisfied with a single mountain tour, but should insist upon a persistent and considerable employment of the muscles in gymnastics, walking, and the like.

Baths also promote oxidation, but they are far less potent than is muscular exercise. Cold baths, brine baths, or baths containing carbonic-acid gas, may be employed. One way in which they do good is by stimulating the nervous system. Oertel regards it of great importance to diminish the amount of water in the sys tem, a point which has until very lately received little attention. The diminutio in the amount of fluids may ameliorate any circulatory disturbance (*vide supr* and relieve venous congestion, and it undoubtedly has some value in the tre ment of obesity. Oertel has shown that a simple diminution in the amoun fluids ingested, when there is no other change in the diet or mode of life, effect a diminution of the adipose tissues. This result is probably due main the diminished strain upon the heart and the consequent increase of oxida but the chief cause of the rapid loss of weight, which we actually see in th pulent as a result of the deprivation of fluid, is to be found not in the loss but in the marked loss of water contained in the body. "Desiccation" ma ther be promoted by stimulating the perspiration by bodily exercise or by baths. This withdrawal of liquid from the system, however, is advisable cases where there is already incipient cardiac failure.

It is evident that numerous excellent methods are at our disposal for th ment of obesity; but their application to any particular case should be th of a careful consideration of the special circumstances presented. A ve tial point is that the injunctions of the physician should not merely be be carried out; and it is precisely here that the treatment of many cas shipwreck. We may be baffled by the patient's lack of energy and pe or by the importunate demands which his profession or social position him. Indeed, it is sometimes absolutely impossible to prosecute the tr e, in which case bathing- and health-resorts are to be urgently recommended. re alone can the patient muster up the resolution necessary for carrying out lesired changes in his mode of life. The incontestable success of treatment at sbad, Marienbad, Kissingen, Tarasp, and similar resorts is doubtless only to a small extent the result of their specific medicinal influence, but it is mainly to a strict observance of the above-described diet and regimen. The internal of mineral waters is not entirely without a beneficial effect. Their laxative ities diminish the absorption of food from the intestinal canal. It should be , however, that the patient is at the same time exposed to the danger of a e of his nitrogenous tissues. This is why patients frequently complain of the litating effect of these mineral springs; to avoid which, it would be well to ease the amount of albuminoids in the diet. We must also consider the need uarding against too great a supply of fluid, with regard to what was said e as to the occasional service of "desiccation" of the body.

CHAPTER XIII

SCROFULA

)efinition and Symptoms of what is called Scrofula.—We desire to present, ıe close of this section, a brief description of scrofula, but merely from a prac- l standpoint. From a scientific point of view scrofula is not to be regarded as special variety of disease. The term is applied to a group of symptoms seen t frequently in childhood, the essential features of which consist in the ap- ance of chronic enlargements of the lymph-glands, and in certain diseases of skin and mucous membranes. The simultaneous appearance of these various ıomena does really produce a somewhat characteristic picture, which can fre- ıtly be recognized at the first glance.

Most scrofulous children appear pale, with a flabby skin and soft muscles. panniculus adiposus may nevertheless be tolerably well developed. Not equently the face is puffy, with prominent lips. This is called the "torpid tus." In other cases the child has small features and a remarkably delicate te skin, which but partly conceals the superficial veins, and is readily suf- d with blushes. To these the name "erethitic habitus" is applied. Enlarged ph-glands are to be felt in the throat, at the angles of the lower jaw, and in back of the neck, and occasionally in other parts of the body. These glands remain indolent for a long while, or they may suppurate and break exter- y. Chronic cutaneous eruptions are often seen in various places. The most mon of these is a scaly or impetiginous eczema, affecting the face, scalp, or emities. More severe affections are lupus ("*lupus scrophulosorum*"), pru- , and lichen scrophulosorum.

)f the mucous membranes, the conjunctiva and the lining membrane of the rils are most frequently affected. Conjunctivitis in various forms is a char- ristic symptom of scrofula; as are also blepharitis ciliaris, keratitis, and nic rhinitis, which last often terminates in a pronounced ozæna (*q. v.*). onic diseases of the ear are also frequent, such as otitis media, with perfora- of the tympanum, and occasionally caries of the mastoid cells and its unfor- ate results.

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The object of the treatment just suggested is exclusively the limitation of the production of fat. We may also promote the destruction of the fat already stored up in the system. A chief means to this end is muscular exercise, which undoubtedly increases the oxidation of the adipose tissues. Carried out in a proper manner, it is therefore a most valuable adjuvant to treatment. Oertel has recently pointed out that muscular exertion does good in still another way—namely, by promoting cardiac activity and inducing deep respiratory efforts. Thus the heart is strengthened and circulation promoted. Mountain-climbing is one of the best modes in which to take the desired exercise. We need hardly say that the increased muscular activity makes it possible for the patient to take an increased amount of food without injury. In dealing with cases of obesity in which the condition is due less to overfeeding than to lack of muscular exercise, it might be decidedly advisable to lay the most stress upon the increase in muscular activity; for certainly it is more advantageous for the organism to get rid of its excess of fat by means of lively metabolism, while nutrition is well maintained, than to be more or less starved by an excessive limitation of diet. We should not rest satisfied with a single mountain tour, but should insist upon a persistent and considerable employment of the muscles in gymnastics, walking, and the like.

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It is evident that numerous excellent methods are at our disposal for the treatment of obesity; but their application to any particular case should be the result of a careful consideration of the special circumstances presented. A very essential point is that the injunctions of the physician should not merely be made, but be carried out; and it is precisely here that the treatment of many cases suffers shipwreck. We may be baffled by the patient's lack of energy and persistence, or by the importunate demands which his profession or social position make upon him. Indeed, it is sometimes absolutely impossible to prosecute the treatment at which case bathing- and health-resorts are to be urgently recommended. ne can the patient muster up the resolution necessary for carrying out d changes in his mode of life. The incontestable success of treatment at , Marienbad, Kissingen, Tarasp, and similar resorts is doubtless only to a ll extent the result of their specific medicinal influence, but it is mainly strict observance of the above-described diet and regimen. The internal ineral waters is not entirely without a beneficial effect. Their laxative diminish the absorption of food from the intestinal canal. It should be ever, that the patient is at the same time exposed to the danger of a his nitrogenous tissues. This is why patients frequently complain of the ng effect of these mineral springs; to avoid which, it would be well to the amount of albuminoids in the diet. We must also consider the need ing against too great a supply of fluid, with regard to what was said to the occasional service of " desiccation " of the body.

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e deeper-lying tissues, the bones and joints are most apt to suffer. The s located here are almost exclusively " fungous "—namely, fungous nd periostitis, white swelling, and caries. Formerly there was frequent use of such terms as "scrofulous inflammation of the knee-joint," or "scrofulous caries of the ribs."

If we inquire into the nature of this strange group of symptoms thus briefly enumerated, we shall find that by far the greater number of cases of well-marked scrofula are examples of tuberculosis. Tubercle bacilli have been demonstrated in connection with most of the fungous or "scrofulous" diseases of the bones and joints. Ozæna is often a tubercular disease of the nose, lupus is a tuberculosis of the skin, and many forms of otorrhœa are really tuberculosis of the ear. The ætiology of "scrofula" is therefore in the main identical with that of tuberculosis (*q. v.*), and this explains why the old physicians habitually insisted upon the intimate relationship between the two diseases. It was formerly thought that scrofula often terminated in tuberculosis—that is, a scrofulous child is apt to suffer eventually from tuberculosis of the lungs, intestine, or brain. To-day we know that most scrofulous children do not become, but that they are already, tuberculous.

It must, however, be borne in mind that in practice many diseases are termed scrofulous which have nothing to do with tuberculosis. Many cases of perfectly innocent eczema of the face and scalp lead to swelling of the glands in the throat, and are therefore termed scrofulous eczema. These cases are probably most of them secondary, and the result of external irritation and the like. Again, many enlarged glands in the neck are the result of pharyngeal trouble, as after scarlet fever, and are equally devoid of a tubercular taint. Pseudo-leukæmic lymphomata may also occur in children; and it should be borne in mind that hereditary or acquired syphilis may produce in children lesions closely resembling those of scrofula.

It is therefore the duty of the physician in every case of "scrofula" to analyze the ætiology and symptoms carefully, in order to determine with what he has to deal. "Scrofula" should be regarded merely as a short way of naming a certain group of symptoms. It is convenient to retain the term as being less likely to startle the friends of the child than would the true name of the disease.

Treatment.—In the treatment of scrofula we have first to attack the various local diseases, and, second, to invigorate the general health. We can not here enter into all the details of local treatment, but we must refer the reader to the special descriptions already given of the various local affections. We may, however, briefly mention a few facts with regard to the treatment of scrofulous swelling of the lymph-glands. Painting the overlying skin with tincture of iodine is a very common practice, but it seldom does much good. We have obtained more satisfactory results from iodoform ointment, or from the repeated inunction of sapo viridis. For particulars as to the opening of abscesses, or the extirpation of glands, we must refer to works on surgery.

Second, in the general treatment of scrofula every possible means is to be employed to invigorate the system. Abundant nourishment and fresh air are essential. The child may be taken either to the country, the mountains, or the seashore. Cod-liver oil is regarded by some as a specific in scrofula; but its undoubted value really lies in the fact that it is an easily digested fat. Some children can take a considerably larger dose than others without its disturbing the stomach. Usually we prescribe two or three tablespoonfuls per diem. Salt baths enjoy a great reputation as a remedy in scrofula. If circumstances permit, the best way is to visit some place where there are brine baths, such as Kösen, Sulza, Salzungen, Arnstadt, Kreuznach, Münster am Stein, Rehme, Reichenhall, Ischl, or Colberg.

Treatment at these resorts is preferable to the use of artificial baths at home, because it is under more favorable hygienic surroundings.

The chief internal remedies are iron, iodine, and arsenic. A favorite prescription is syrup of the iodide of iron. Arsenic may have a slight specific influence upon the tuberculous adenitis and bone diseases, as also upon lupus, but beyond this we can not expect any great benefit from internal remedies.

DISEASES OF THE NERVOUS SYSTEM

I.—THE DISEASES OF THE PERIPHERAL NERVES

SECTION I

DISEASES OF THE SENSORY NERVES

CHAPTER I

GENERAL REMARKS UPON THE DISTURBANCES OF SENSIBILITY

THE disturbances of sensibility, like all other functions of the nerves, are manifested in two directions. Under pathological conditions we observe either an abnormal diminution or a complete absence of sensibility—anæsthesia—or a morbid increase—hyperæsthesia. While, in anæsthesia, the ordinary or even the strongest irritations which excite the sensory nerves produce only a weak and insignificant sensation, or even no corresponding sensation at all, in hyperæsthesia very severe and painful sensations are caused by weak irritations. The "symptoms of sensory irritation" are to be distinguished from hyperæsthesia, although they are often present along with it. By this term we mean sensations which cause internal irritation, not from without, but from certain abnormal morbid conditions in the nerve itself. In the region of cutaneous sensibility, with which we shall chiefly concern ourselves in what follows, these symptoms of sensory irritation show themselves partly as actual pain and partly as the so-called paræsthesiæ—that is, abnormal sensations in the skin, which are termed "formication" (the crawling of ants), "prickling," "numbness," "a furry feeling," "hot and cold feelings," etc.

The Different Varieties of Cutaneous Sensibility and the Methods of testing them.—As is known from physiology, the irritation of the sensory cutaneous nerves produces in us a number of sensations, differing in quality according to the manner in which the irritation acts. If, therefore, we would obtain an accurate estimate of the condition of the patient's cutaneous sensibility, we must make a special test of all the different forms of sensation; for we often see that the disturbances of sensibility do not involve all the forms mentioned alike, but that one kind of irritation is followed by perfectly normal sensation, while there is more or less complete anæsthesia for another kind. We term such partial anæsthesias of the skin, which are manifest toward only one or more forms of irritation, "dissociated" or "partial anæsthesias." The study of these partial anæsthesias is the more interesting, since, according to recent physiological theories (Blix, Goldscheider, etc.), the different qualities of cutaneous sensibility are transmitted to the consciousness by special nerve-fibers, so that there are probably in the skin different terminal nervous organs for the perception of sensations of pressure, cold, heat, etc. Such a condition recalls the well-known specific energy of the different fibers of the optic nerve to colors, assumed by many physiologists.

The separate varieties of cutaneous sensibility, and the chief practical methods of testing them, are as follows:

1. SENSIBILITY TO CONTACT.—The examination of the sensibility of the skin to simple contact is usually performed by repeatedly touching or stroking the part of the skin to be tested with the finger, a fine brush, or some other blunt object (not of metal, in order to exclude sensations of cold) while the patient's eyes are shut, and asking the patient to say whether he has felt the touch or not. When it is necessary to call the patient's attention to the investigation, it is always best to do so afresh by asking "Now?" and then either really to touch the skin or else to ask the question when only pretending to touch it. In this way we are safest from the error, which otherwise may readily arise, from a lack of attention or of practice on the part of the patient. We may often make the patient close his eyes and then count the number of times we touch the skin, or we touch the skin alike in different parts of the body (alternating on the two legs, etc.), and then ask him to tell merely the place where he is touched (*vide infra*). The degree of correctness of his answer of course shows the condition of the sense of contact. The comparison of the sensibility in symmetrical parts of the body is of importance, especially in unilateral disturbances.

In a good many cases the patients feel the slightest touch, but nevertheless they say that the touch on the affected part is "more indistinct," "duller," in short, "different" from that on the normal part of the skin.

2. SENSE OF LOCALITY (*Localization of Sensation*).—Under normal conditions, as we know, not only do we feel the touch of an object, but we can tell with a good deal of accuracy the place on our skin which was touched. This power we term the ability to localize our sensation. In nervous patients we often see that, while cutaneous sensibility is still present (we refer not only to tactile sensibility but also to the other forms), it is localized more poorly and with less accuracy than is the case under normal conditions.

In the simple test of the sense of contact we may also examine, at least roughly, the power of localization if we ask the patient also to state where the touch is felt, or if we ask him to designate with his hand as accurately as possible the part of the skin touched. A more accurate method was proposed by E. H. Weber. It consists in determining the smallest distance which must separate two simultaneous cutaneous irritants from each other, in order that they may be perceived as two locally distinct sensations. Weber has found that this distance differs very much in different parts of the body, and from this he has divided the whole surface of the skin into so-called tactile circles. As data for the examination of patients, some of the figures obtained by Weber in healthy individuals may here be given: The smallest distance at which the two points of a pair of compasses* applied at the same time to the skin may plainly be distinguished from each other is 11 to 15 millimetres on the cheeks, 6 millimetres at the tip of the nose, 22 millimetres on the forehead, 1.2 millimetre at the tip of the tongue, 4 to 5 millimetres at the back of the tongue and on the lips, 34 millimetres on the neck, 77 millimetres on the upper arm, 40 millimetres on the forearm, 31 millimetres on the backs of the hands, 11 to 16 millimetres on the backs of the fingers, 2 to 3 millimetres at the tips of the fingers, 55 to 77 millimetres on the back, 45 millimetres on the chest, 77 millimetres on the thigh, 40 millimetres on the leg, 40 millimetres on the instep; but these figures show certain variations in different individuals, so that they are to be regarded as only average values.

Testing the sense of locality by Weber's method has no great practical importance; it also takes very much time and demands intelligence, patience, and good will on the part of the patient. The influence of practice is manifested in a very remarkable way, since the perceptible difference becomes considerably less if the examinations are often repeated. On the other hand, a single examination, as in testing any form of sensibility, must not be too long protracted, for otherwise the patient may easily become fatigued, and the data obtained will be entirely contradictory. If we test the sense of locality by bringing down the two points not at the same time but one after the other, and vary it by touching the same place twice, or a different place each time, we obtain from the outset, as we have repeatedly proved, smaller numbers than if we touch the skin with the two points of the compasses at the same time. We also obtain somewhat different values for the fineness of the sense of locality if we test the so-called sensations of motion (Leube)—that is, the distinction between a simple circumscribed touch of the skin, and a very short line drawn with a stick on the skin. In this way we can also determine whether the patient can distinguish accurately the direction of transverse and longitudinal lines.

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* There are special "tactile compasses" with blunt ivory points and graduated quadrants.

We may also mention here two peculiar anomalies of sensibility, *polyæsthesia* (G. Fischer) and *allochiria* (Obersteiner). Polyæsthesia is a symptom shown by certain patients (especially patients with tabes), who, when the skin is touched with only one point of the compasses, have a sensation as if they felt two or even more points. The cause of this remarkable anomaly of sensation is not yet adequately explained. In allochiria a stimulation of the skin (touch, pain) is felt not on the part touched, but on the corresponding point on the other half of the body. This remarkable symptom has been observed a few times in cerebral hemiplegia, tabes, etc.

3. SENSE OF PRESSURE.—The healthy man distinguishes with ease whether an object merely touches the skin or exerts pressure upon it. In the perception of pressure we must consider not merely the sensibility of the skin, but probably even more the sensibility of the deep parts below the skin (fasciæ, muscles, periosteum). If a fold of the skin be raised, differences of pressure are felt much less plainly than when pressure is at the same time exerted on the deeper parts. Since E. H. Weber's investigations we know that we estimate the difference in the intensity of our sensations of pressure, not according to the absolute, but according to the relative increase in the pressure. If, for example, a part of the skin has a weight of nineteen grammes on it, and we perceive the first manifest increase in our sensation of pressure when a weight of one gramme is added to it; when the skin has a weight of one hundred and ninety grammes on it, we first perceive the increase of pressure not when one gramme is added, but when we add ten grammes. If this law, on more accurate testing, has not proved so simple as it would seem according to the results of Weber's first investigations, still it is generally a fact that under normal conditions an increase of pressure of about one twentieth to one thirtieth of the original pressure may be plainly perceived in the different parts of the body.

Different methods and instruments, such as Eulenburg's "baræsthesiometer," have been devised for testing accurately the sense of pressure in patients, but they have entered but little into practice on account of their elaborate character. We usually content ourselves with testing the sense of pressure by applying different weights, or coins. We must mention here that the part of the body to be tested must be fully supported, that we must also exclude sensations of temperature at the same time by putting some non-conductor beneath the weights, and that we must apply the separate weights to the same place on the skin at equal intervals of time, which must not be too long after one another. For the ordinary needs of practice it is wholly sufficient to test the pressure sense simply with the finger or any blunt object. By pressing with the finger we can vary the pressure, make it weaker or stronger, and thus learn whether the patient is able correctly to state all these differences.

We should never fail to test the pressure sense in patients with nervous disease, since anæsthesia of the pressure sense, or at least a diminution, is by no means rare. We find quite often, especially in spinal diseases (tabes, myelitis, compression), that the patient feels a light touch on the skin, but that he can not distinguish a marked pressure at all, or only obscurely. On the other hand, the sense of pressure is sometimes well preserved when the other forms of cutaneous sensibility are much impaired (as in syringomyelia).

4. Sense of Temperature.—As we have already said (p. 789), we have lately been led by physiological investigations (Goldscheider, etc.) to believe more and more strongly that sensations of heat and cold are to be regarded as due to two wholly distinct functions of the cutaneous nerves. These functions are probably performed by special nerve terminations and nerve-fibers. If we test the sensibility of the skin for heat and cold with a pointed metal rod, heated or cooled, we can easily be convinced that the hot rod excites a sensation of warmth only on definite parts of the skin, and that the cold rod excites a sensation of cold only on other definite points ("warm points," "cold points"), but on the intervening points these rods excite no sensation of temperature. From repeated personal observation we can state that pathological symptoms agree completely with this theory of the existence of special nerve conduction for sensations of heat and cold, for we see that changes in the temperature sense in the skin by no means affect the heat sense and the cold sense equally. It is therefore always necessary to test both forms of the temperature sense separately. We often find that while one form of temperature sense is in normal activity, the other is much altered—a pronounced partial anæsthesia to heat or cold. The affected sense may be wholly lost or merely blunted, so that hot water is felt merely as tepid, or ice as cool. If the temperature sense be lost, the application of a hot or cold object will cause only a sensation of touch, and not of temperature. Extreme temperatures, especially extreme heat, excite, as is well known, a sensation of pain. This may, of course, remain normal; but if there is also analgesia (*vide infra*), it disappears.

If anæsthesia to cold be present, patients often speak of having a distinct sensation of warmth when the skin is touched with a bit of ice. This symptom, which we discovered and called "perverted temperature sense;" may perhaps be explained by supposing that the heat nerves are excited by the severe irritation from cold. The contrary symptom, that heat excites a distinct sensation of cold, is very much less common.

Besides the sensations of temperature we usually test also the patient's capacity to distinguish differences of temperature. Within the moderate degrees of temperature, 80° to 100° (25°–35° C.), differences of a degree Fahrenheit (0.5° C.) are easily distinguishable under normal conditions, and even half a degree (0.2° C.) can be distinguished on the face and fingers, but only about two degrees (1° C.) on the back.

Variations in the temperature sense are very common. We often find them (especially in spinal disease) in those cases where the simple sensibility to touch is fully retained. The test of the sensibility to heat and cold should therefore never be omitted in testing the sensibility. It can be done with sufficient accuracy for all practical diagnostic purposes simply by touching the skin with large test-tubes filled with ice or hot water. The various thermæsthesiometers that have been devised are too complicated for practical purposes. For a hasty test it is sometimes quite serviceable to try whether the patient can distinguish warm breathing upon a given portion of the skin from cool blowing upon it.

5. Sensation of Pain.—The sensibility to pain, also, can not be excited in all parts of the skin by circumscribed irritation, such as a pin-prick. We find "pain points" and parts insensible to pain. From this fact and for other reasons, which can not be detailed here, von Frey has concluded that there are special

ain nerves, whose terminal apparatus is to be found in the free intra-epithelial erve endings. It is a fact that the sensibility of the skin for touch and that or pain are not always parallel under pathological conditions. We sometimes see hat a patient does not feel a simple touch on the skin, when sticking a needle nto it is immediately painful; while, on the other hand, we still more frequently nd that a patient feels quite a light touch on the skin, but that the most marked ritation, such as pinching or pricking it, does not excite the slightest pain, but is elt only as a simple touch, or at most as a slight pressure. This latter con- ition of sensibility, the loss of cutaneous sensibility to pain with retained tactile ensibility, is termed analgesia. Both in peripheral and in central nervous dis- ases analgesia is a symptom that may be quite frequently observed.

The test of sensibility to pain may be best performed by pricking with the oint of a pin, and also by pinching a fold of the skin with considerable force. he simple test, whether a patient is able to distinguish between the head and the oint of a pin, is much employed. We thus obtain information at the same time f the sensibility to pressure or touch; for a prick with the point of a pin nor- ally excites a slight pain, while the application of the pin-head causes a sensa- on of simple touch or slight pressure. In testing the sensibility to pain we must e careful to prick deep enough (penetrating the epidermis), but at first we must nploy only brief pin-pricks. Only when these are not felt as painful should we se long-continued pin-pricks. Very often by summation of the irritation (*vide fra*) a lively sensation of pain is produced. If there be marked analgesia we an of course stick a pin deep into the skin or push it through a fold of skin with- ut causing pain.

It is well known that various kinds of irritation may produce pain: severe ressure, extreme temperature, strong electrical currents, etc., as well as wounds f the skin (for pin-pricks are to be regarded as such). It is always some irrita- on which acts on the deeper layers of the skin.

6. Electro-cutaneous Sensibility.—The test of cutaneous sensibility by eans of the electric current has been proposed by various observers. The dvantage of it is that in this way the intensity of the irritation can be very asily and accurately graded and expressed in numbers, by the position of the oil in using the faradic current, or by the galvanometer in using the con- tant current. The faradic current is usually sufficient in testing the sensibility, nd we designate it by the position of the coil when the first sensation is felt nd the position when the first pain is felt. In general, the differences of the farado-cutaneous sensibility are not very marked. Pathological deviations are given by comparison with normal portions of the skin (testing, if possible, symmetrical parts) or with healthy people. The galvanic current is also used to test sensibility, noting the occurrence of a burning sensation at the cathode. For practical purposes, the test of electro-cutaneous sensibility is unnecessary, since its results are the same as in testing the sensibility to touch and to pain.

7. Delay of Sensation (so-called Delayed Conduction), Summation of Irritation and After-sensations.—Quite frequently in diseases of the spinal cord, especially in compression paralysis and tabes (*q. v.*), and not infrequently in peripheral nerve disease (neuritis), we see a marked delay in sensation after the action of an irritant. This symptom affects chiefly the sensibility to pain. If in such a case we stick a pin into the sole of a patient's foot, several seconds elapse, even ten or twenty, it is said, before the pain is felt. As was first observed by Naunyn and E. Remak in tabes, and as has often been confirmed since, after sticking a pin into the foot there is in such cases first a simple sensation of slight pressure, and some seconds later the peculiar sensation of pain, so that the patient at once responds to the prick with "Now," and a little later with "Ow!" as an expression of pain.

On analyzing this symptom somewhat more closely we will have to distinguish between single brief and continuous irritations (pin-pricks). If we make, for example, a single short prick in the skin, it may happen that the pain thus caused appears comparatively late. This symptom depends perhaps upon a "delay of conduction," although it is still undetermined, of course, whether this delay takes place actually in the peripheral-nerve fiber or in the interpolated ganglion cells. The conditions in permanent painful excitation (continued pin-prick) are different. In these cases we notice very often that immediately after the insertion of the pin the patient feels nothing or only a simple touch. If the prick be continued, however, there is felt a lively sensation of pain, often only after a few seconds. This phenomenon is apparently due, not to a delay of conduction, but to a summation of the irritant action, which thus gradually attains sufficient intensity finally to break through the existing resistance and to reach the consciousness in full strength. At the same time with the sensation of pain there is usually a reflex twitching, and the same conditions are therefore to be considered in testing the cutaneous reflexes (*vide infra*).

In this connection, it is well to mention briefly the after-sensations, which we have noted in the most striking fashion, especially in cases of tabes. After a single short prick these after-sensations appear as a burning feeling in the skin, which lasts a strikingly long time, or else the first sensation soon ceases after the prick, and then, in the same spot on the skin, new sudden sensations of pain are felt several times, just as if the patient had been pricked anew.

8. THE SENSIBILITY OF THE MUSCLES AND JOINTS.—A number of sensations are classed together under the names of "muscular sense" or "muscular sensibility." They are not all wholly of the same value, and, under pathological conditions, they must be tested separately.

Ordinarily we call our power to be informed of the position of any of our limbs without the help of our eyes, and of the extent of any motion made by them, the "muscular sense." If we put a healthy person's arm into a given position when his eyes are closed, and tell him to put the other arm into the same position, he can do it with considerable accuracy. If we make passive movements in any of the joints of the extremities, a healthy person whose eyes are closed can easily and correctly state the form and direction of these movements. In nervous patients, however, this power may be diminished or lost, and we often speak of "disturbances of the muscular sense"; but we must note that the judgment as to the position of the limbs and the passive movements executed with them is not chiefly, much less exclusively, due to the sensibility of the muscles. In all probability it depends much more upon the sensibility of the articular surfaces, the ligaments and tendons, and in part even of the skin, all of which tissues are relaxed or made tense in the different movements. It would therefore be more correct to speak of sense of position (the sensation of position of the limbs) and sense of motion (sensation of passive movements) instead of the "muscular sense." The sensibility to movement is tested simply by making the patient close his eyes, by taking an arm or a leg firmly in both hands, and then by making passive movements up and down, right and left, at first extensive and then gradually less. The patient should state the direction of the movement made. We may also describe with the extremities different letters or figures in the air, which the patient should recognize with his eyes closed. If the test is to be absolutely accurate, passive movements must be tested in all the joints individually (shoulder, elbow, hip, knee, etc.), but as a rule we can judge correctly if we test the passive movements for the whole extremity at the shoulder or hip. By control experiments on healthy persons we can readily convince ourselves how uncommonly sharp and accurate the sensation of passive motion is under normal conditions.

The sensation of the position of the limbs is tested by putting one extremity passively into a given position and then asking the patient to put the other corresponding extremity into the same position, as nearly as possible. It is still better to have the patient make some definite movement, especially pointing to or seizing some object, at first with the eyes open; then to let him close his eyes and repeat the movement as well as he can.

The sensation of the strength of voluntary muscular contraction, the so-called "sense of power," is quite different from the sensations just described. Besides the peripheral sensations in the muscles and tendons we perhaps have to do with special sensations of innervation. In raising weights we can distinguish with considerable accuracy the lighter from the heavier, when the pressure on the skin is excluded as far as possible. In such cases, also, we do not deal with the absolute, but with the relative differences in weight; we can usually tell quite plainly when one fortieth of the original weight has been added or taken away. The sense of power, then, is somewhat finer than the sense of pressure. In order to exclude the latter in the test we have the patient lift the weight, suspended in a towel, with his hand or foot, but in the lower extremities it is scarcely possible to exclude entirely the co-existing sensations of pressure.

We must mention, in conclusion, that muscular contraction is in itself accompanied by a sensation, as may be proved, for example, in irritating the muscles by faradism—electro-muscular sensibility. In certain forms of spasm the muscular contraction becomes so strong that it causes a decided pain, which is probably due to irritation of the sensory muscular nerves discovered by C. Sachs.

Tests of the sense of power and of the electro-muscular sensibility have thus far been but little employed in nervous diseases. They also have no great practical significance, but the test of the sensations of position and motion are often of great importance. Marked disturbances in these functions are often found in advanced cases of tabes and sometimes in other spinal diseases and in cerebral (especially cortical) paralyses. Complete loss of "muscular sense" is also found quite frequently in severe hysterical affections, usually associated with other hysterical anæsthesias and hysterical paralysis.

9. THE SPECIAL SENSIBILITY TO TOUCH (STEREOGNOSTIC SENSE, ACTIVE TOUCH).—The highest and most valuable function of the skin as a sensory organ is the judgment we can often make of the size, form, hardness, and consistency of objects solely by feeling of them—in short, the recognition of them. This function, the special sense of touch, is apparently a complicated estimate of all the separate sensations derived from the different cutaneous nerves, to which are always added at the same time a large number of sensations of movement. For "touch" and "feeling" always imply a movement of the parts which touch. We see, therefore, that a patient whose hand is completely paralyzed can recognize objects either with difficulty or not at all by the touch, although the cutaneous sensibility is retained. If there be actual anæsthesia of the skin it is, of course, also impossible to recognize objects by the sense of touch, although mobility is retained. There are, however, also cases where the hand can be moved well enough and where the sensibility of the skin and muscles for the different qualities of sensation is retained, but nevertheless the recognition of objects by the sense of touch is markedly impaired. In such cases there is probably always some cerebral (cortical) disturbance, which may be regarded as analogous to cortical blindness and cortical deafness—that is, the normal mental estimate, the "understanding" of the various cutaneous sensations received. The author has seen such conditions, especially in cerebral infantile paralysis; the intelligent child feels every touch on the skin perfectly well, but, when his eyes are closed, he can not recognize by feeling of them with the affected hand the most familiar objects (key, knife, ring, money, etc.). At any rate it is very important, especially in cerebral diseases,

not to neglect this mode of investigating the special "sense of touch." If we choose for our tests simple stereometric figures (sphere, cube, cone, octahedron), we may also speak of a "stereognostic sense."

CHAPTER II

THE SENSORY CONDUCTION TRACTS AND ANÆSTHESIA OF THE SKIN

1. **Course of the Sensory Conduction Tracts.**—In every tract of the conducting path, which runs from the terminal apparatus of the sensory cutaneous nerves to the centers for the perception of sensation in the cerebral cortex, we may have, under pathological conditions, a break in the conduction, and, as a result of it, a complete or partial anæsthesia of the corresponding part of the skin. We speak of a peripheral, spinal, or cerebral anæsthesia, according to the place where this break in the conduction occurs. The anatomical course of the sensory fibers (Fig. 84) is, however, not yet known as accurately as we could desire; the course of the sensory tract is also apparently far more complicated than that of the chief motor tract (*vide infra*, Section II). This is dependent partly upon the different qualities of sensation, and partly on the fact that the sensory tracts enter into many combinations with motor cells for the excitation of reflex movements or for the purpose of co-ordination of motion.

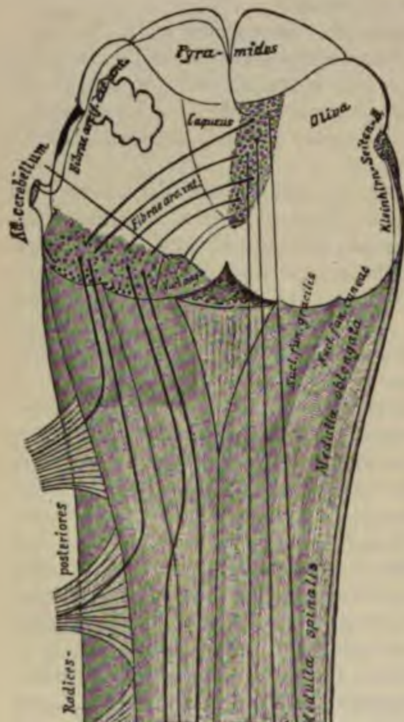
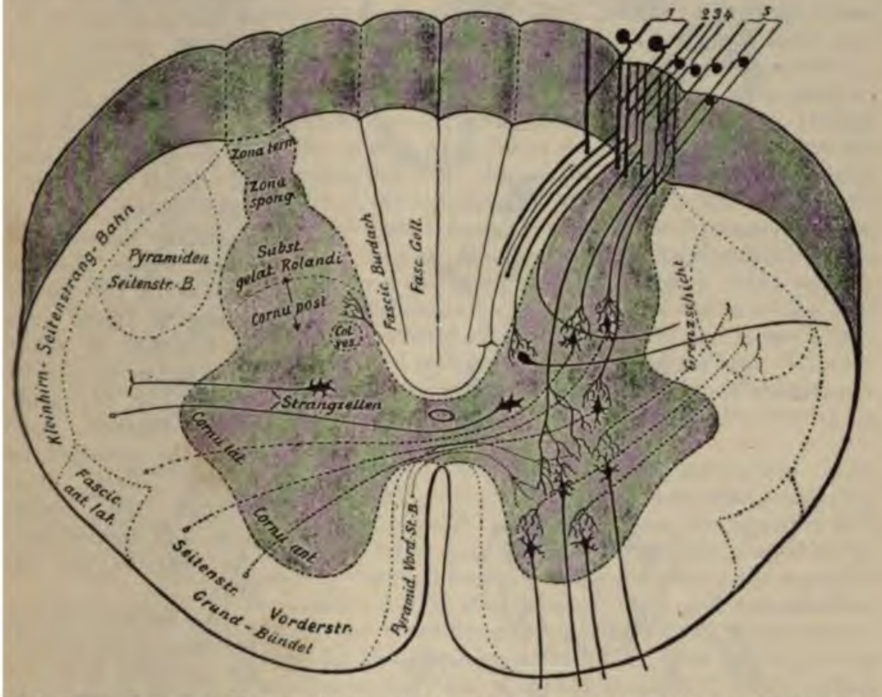


FIG. 84. (From EDINGER.) — Diagram of the course of the posterior sensory root-fibers in the spinal cord and medulla oblongata. *Vorder Seitenstr.* = Antero-lateral columns. *Kleinhirn. Seiten. B.* = Lateral cerebellar tract.

We must regard the sensory nerve-fiber, like every fiber in the nervous system, as the direct process of a nerve-cell (ganglion-cell). This sensory fiber receives the external impressions made upon its peripheral terminal organs in the skin. We now know definitely that all peripheral sensory nerve-fibers come from the cells of the spinal ganglia (intervertebral ganglia). All, or at least most of these cells, have a short process which soon divides into two offshoots from the cell: one long one, going to the surface of the body (the peripheral sensory nerve), and a shorter one, which enters the posterior root of the spinal cord and is intended for the further conduction of sensory impressions. Almost all the fibers of the posterior roots accordingly come from the spinal ganglia; during fœtal development ıey grow from their cells of origin into the spinal cord. The spinal ganlion cell with its processes forms the first (peripheral) sensory neurone. The osterior root-fibers, entering the spinal cord with their branches (collaterals, *ide infra*), take very different courses, apparently according to their different ınctions (see Figs. 84 and 85). A part of them (Fig. 85, 4) go to the anterior



ıg. 85.—(From EDINGER.) Diagram of a transverse section of the spinal cord with especial regard to the course of the posterior root-fibers: 1. Fibers in the posterior column. 2. Fibers to the cells of Clarke's column, whose processes enter the lateral cerebellar tract. 3 Direct fibers in the "lateral limiting layer" of the lateral column (not mentioned in the text). 4. Fibers to the motor cells of the anterior horn (reflex fibers). 5. Fibers in the posterior horn splitting up about its cells. *Bahn*, tract; *Grenzschicht*, limiting layer; *Grund-Bündel*, ground bundle; *Kleinhirn*, cerebellum; *Pyramiden*, pyramid; *Seitenstrang*, lateral column; *Vorderstrang*, anterior column; *Strangzellen*, cells to columns.

ɔrns and break up about their motor ganglion cells; these are apparently tracts ır the reflex movements. Another part go to the cells of Clarke's columns, from hich arise fibers for the lateral cerebellar tract (see Fig. 84 and Fig. 85 in the ght half of the figure); these fibers seem to serve for the maintenance of the ɔdily equilibrium and perhaps for the co-ordination of movements. The special ·nsory fibers, however, divide into two main groups (Figs. 84–86); a part of ıem, chiefly the median root-fibers, enter directly into the posterior column of ıe same side. Soon after their entrance the fibers divide again into a short escending and a long ascending branch. The short descending branches serve, ; I suspect, for reflex processes, while the ascending fibers in the posterior columns form the special continuation of the centripetal tract. That portion of the osterior columns, into which these root-fibers enter, is called the "posterior rootɔne," or "root entrance zone"; in the lumbar cord it lies in the middle part of the osterior columns, in the dorsal and cervical cord in the lateral parts (columns of Burdach). Since new root-fibers are constantly entering the cord as we go upard, the fibers coming from the lower portions, and especially from the lumbar ord, are gradually pressed toward the median line by the new fibers that enter.

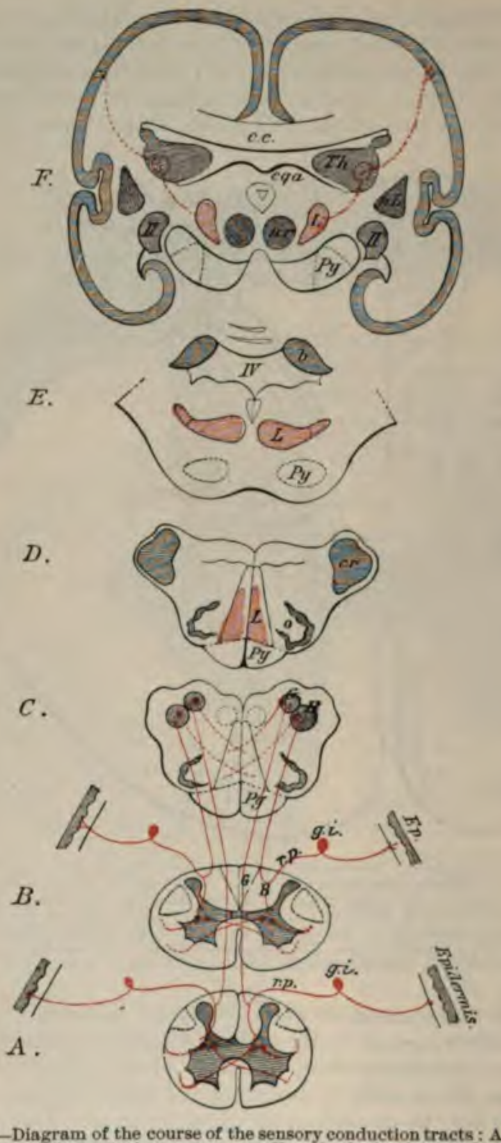


Fig. 86.—Diagram of the course of the sensory conduction tracts: A. Entrance of the posterior sensory root-fibers in the lumbar cord; *gi*, intervertebral ganglion; *rp*, posterior root. A part of the fibers that enter end in the posterior horns, from whose cells new fibers arise and enter the lateral columns, partly crossed, partly uncrossed. Another part of the fibers from the posterior roots pass upward in the posterior columns and form in B the so-called columns of Goll (*G*). B. Cervical cord. Lettering as in A. *B*. Columns of Burdach. C. Medulla oblongata. Region of the decussation of the lemniscus. The fibers from the posterior columns end in the nuclei of Goll's and Burdach's columns (*G* and *B*). New fibers arise from the cells of these nuclei, which form the decussation of the lemniscus. *Py*. Motor pyramids. D. *L*, sensory lemniscus; *ol*, olivary body; *cr*, corpus restiforme. E. Section through the pons. *L*, lemniscus; *b*, superior cerebellar peduncle; IV, fourth ventricle. F. Frontal section through the region of the corpora quadrigemina; *L*, lemniscus; *nr*, red nucleus; *Py*, pyramidal tract in the crusta of the crus cerebri; *nL*, lenticular nucleus; *Th*, optic thalamus (interruption of the sensory tract? Beginning of a third sensory neurone?); *cqa*, anterior corpora quadrigemina; II, corpus geniculatum externum and optic nerve; *cc*, corpus callosum.

Thus it happens that the fibers from the lumbar and the lower dorsal cords are in quite a median position in the cervical cord, in the region of the so-called columns of Goll. All these fibers end in the nuclei of Goll's and Burdach's columns in the beginning of the medulla oblongata. From the cells of these nuclei come the secondary sensory neurones, whose fibers decussate in the medulla oblongata (fibræ arcuatæ internæ, or sensory decussation in the lemniscus, Fig. 86), and in their further course form the so-called lemniscus, which passes through the medulla, pons, and tegmentum of the crus to the cerebrum.

A second (lateral) portion of the fibers of the posterior roots enter at once the gray matter of the posterior horn. These fibers also divide into an ascending and a descending branch. A number of especially fine fibers remain in the periphery of the posterior horn (*zona terminalis*, Fig. 85, also called Lissauer's zone). The fine collaterals of these fibers probably enter the posterior horn through the zona spongiosa, and enter into similar relations with the ganglion-cells as the other coarser fibers of this portion of the posterior root-fibers, which enter the posterior horns directly through the substantia gelatinosa, and after a short course split up in so-called terminal arborization about the ganglion-cells there. With these cells begin the second sensory neurones for this part of the sensory tract; the processes (fibers) of these cells at once enter the lateral column of the opposite side through the anterior commissure, decussate therefore in the anterior commissure of the cord, ascend in the anterior and middle portions of the antero-lateral columns (anterior and internal to the motor pyramidal lateral tract), reach the medulla oblongata also in the lemniscus, and unite here with the above-described fibers from the cells of Goll's and Burdach's nuclei. The diagram on the opposite page (Fig. 86, the red portion, *L*, signifying the lemniscus) gives an idea of the position of the lemniscus—that is, of the chief sensory tract—farther upward in the pons and tegmentum. Higher still, in the internal capsule, the sensory tract lies in the posterior limb behind the motor pyramidal tract (*vide infra*, Fig. 95). There is still considerable obscurity as to its further course. A part of the lemniscus fibers probably end in the optic thalamus, from whose cells, to all appearances, a third sensory neurone arises, sending its fibers to the cerebral cortex. Another part of the lemniscus fibers, however, pass directly to the cerebral cortex without interruption. The sensory fibers from the posterior columns probably end in the cerebral cortex in the central convolutions, especially in the posterior central convolution and in the neighboring portion of the parietal lobe, and perhaps also of the frontal lobe. It is extremely interesting physiologically that the central terminal region of the sensory fibers in part quite coincides in its localization with the motor centers (*vide infra*).

It would be extremely valuable if we knew the centripetal conduction tracts to the cerebrum for the different kinds of cutaneous and muscular sensibility (see the preceding chapter). Unfortunately, our knowledge in this regard is still extremely defective and uncertain. The hypothesis, which seems relatively the most certain, is that the sensory fibers entering directly into the gray posterior horns serve chiefly for the conduction of sensations of pain and temperature. This is supported especially by the observations on syringomyelia (*vide infra*). It has also long been claimed by the physiologists (Schiff) that section of the posterior gray matter causes analgesia. The posterior root-fibers, which ascend in the posterior columns, seem to serve especially for the conduction of muscular sensations and the co-ordination of motion (*vide infra*). Many ways seem to be open for the conduction of simple sensations of contact: it is striking how rarely total anæsthesia of the skin occurs in organic diseases of the spinal cord, and it almost never occurs in organic diseases of the brain.

2. **The General Causes of Cutaneous Anæsthesia.**—Regarding the separate causes of anæsthesia, we see, in the first place, peripheral anæsthesia under conditions where the terminal organs of the sensory cutaneous nerves have lost their direct irritability. After chilling the skin, after the local action of ether and similar substances, after the corrosive action of acids and alkalies, carbolic acid, etc., as well as after the use of certain narcotics, such as cocaine, we see an anæsthesia of the skin, which is due to injury of the terminal sensory organs. We may probably put the frequent anæsthesia of washerwomen in this class, for their hands and forearms are exposed all day to the action of cold, lye, etc. The anæsthesias which develop in circulatory disturbances of the skin also have the same peripheral origin, especially the "spastic anæmia" which sometimes comes in the hands and is due to a spasm of the small arteries.

We distinguish the peripheral anæsthesia of conduction, which may be produced by all forms of lesion of the nerve-trunks, from the peripheral anæsthesias in the strict sense of the term. Traumatic influences, compression from new growths, and inflammation and degeneration of the peripheral nerves, as in neuritis, are the most frequent causes of this form of anæsthesia, which is often limited to the region of distribution of one or more individual nerves. The circumscribed anæsthesias which often come on after acute diseases (typhoid fever, diphtheria), and in some chronic diseases (diabetes), are usually peripheral (neuritic) anæsthesias.

Spinal anæsthesia is very often seen in the different diseases of the spinal cord, most frequently in tabes, because this, as we shall see later, is a direct disease of the peripheral sensory neurone; but spinal anæsthesia is not infrequent in diffuse acute and chronic inflammation of the cord and in compression and new growths, and especially in syringomyelia. As a rule, it is bilateral para-anæsthesia, but in unilateral lesion of the cord it may also be unilateral, the anæsthesia being found on the half of the body opposite to the affected half of the cord, owing to the decussation of the sensory fibers in the gray matter (*vide supra*).

Cerebral anæsthesia is seen especially in hæmorrhages, foci of softening, and tumors, which affect any part of the lemniscus tract or the posterior portion of the internal capsule. Such anæsthesias affect the half of the body opposite to the lesion, and are called hemianæsthesia. As a rule, experience shows that cerebral anæsthesia of organic origin is seldom very intense. Hysterical anæsthesias, however, may attain an extreme degree; they are due to a disturbance of the perception of sensory impressions by the consciousness.

3. **The Symptoms of Cutaneous Anæsthesia.**—In many cases the patient himself notices the existence of anæsthesia. He finds that in certain parts of the body he no longer feels the pressure of his clothing or the bed-clothes in the usual way. Anæsthesia of the hands is soonest noticed, because this affects the patient's occupation in diverse ways, so that, for example, he is apt to let fine objects, such as needles, drop from his hands. In other cases, of course, the anæsthesia is first found on a physical examination. It is worthy of note that hysterical anæsthesia especially, even if it be very marked and extensive, may often be wholly overlooked by the patients themselves.

Anæsthesia is very often combined with abnormal subjective sensations, paræsthesiæ, in the affected portions of the skin. The patient has a feeling there of "numbness," or a "furry feeling," or complains of prickling or formication. The anæsthetic parts may even be the seat of very decided pain (*anæsthesia dolorosa*), if there is abnormal irritation of the sensory nerves from the break in the conduction toward the center. The most diverse forms of anomalies of motility and of the reflexes, and vaso-motor disturbances, may of course be present in addition to the anæsthesia. Special mention must be made of the so-called trophic disturbances (inflammation, ulceration, bed-sores, etc.) which are often seen in anæsthetic regions. Whether in these cases we actually have to do in part with direct injury of the tissues from the failure of normal "tropho-neurotic" influences is at present wholly undetermined. The majority of the ordinary so-called "trophic" disturbances are without doubt due to external injuries (slight wounds with consequent purulent infection). When there is anæsthesia, or especially analgesia, of the patient's skin, these injuries do not receive sufficient attention, and therefore they are neither avoided nor thoroughly treated (compare especially the chapter on syringomyelia). Greater external injuries—burns, bed-sores, etc. —are often not noticed at all when on anæsthetic parts, and therefore they may attain an extraordinary size and become very bad.

Voluntary motion, if we may draw conclusions from existing clinical observations, is not disturbed by anæsthesia in itself, of however great a degree, as long as the motions can be controlled by the eyes, but finer movements are often considerably impaired by cutaneous anæsthesia; thus patients with diminished sensibility in the fingers can usually no longer sew, because they lose the needle every minute. With the eyes shut, however, the motions of the anæsthetic parts become very uncertain, both in anæsthesia of the skin and of the deeper parts, the muscles and joints; since then the patient loses, to a great degree, his power of judging of the extent and of the precise direction of his movements. Very extensive anæsthesia of the skin, associated at the same time with anæsthesia of the organs of special sense, is sometimes not without influence upon consciousness. We have had under observation a very remarkable case of total anæsthesia of the whole body associated with unilateral blindness and deafness. If we entirely excluded this patient from all external impressions of sense by closing his still serviceable eye and ear, we could at once in this way put him into a deep sleep! It must, of course, be said in this connection that such extensive anæsthesias have thus far been observed almost exclusively in hysteria (*vide infra*), and that therefore special psychical factors have perhaps been of influence in the phenomenon just mentioned.

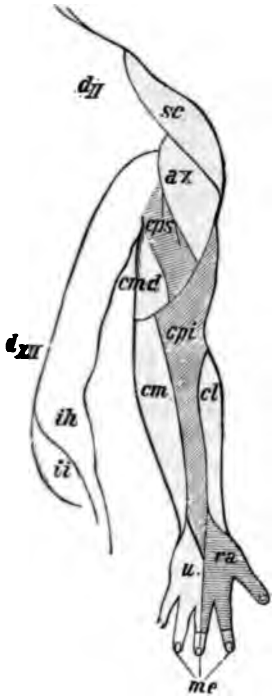


FIG. 87.

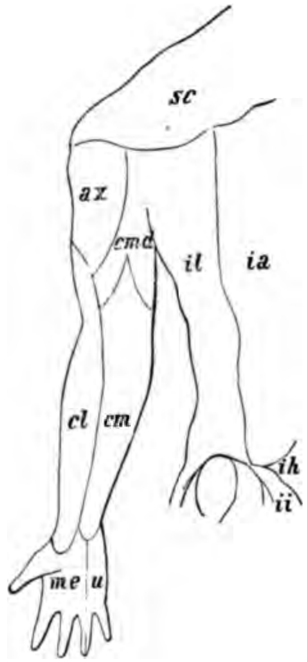


FIG. 88.

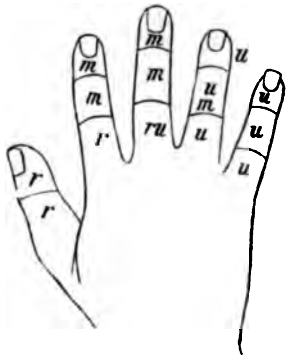


FIG. 89.

FIGS. 87 and 88.*—Distribution of the sensory cutaneous nerves in the trunk and upper extremities: FIG. 87. Posterior aspect. FIG. 88. Anterior aspect. The shaded portion in Fig. 87 designates the territory supplied by the radial nerve. (From HENLE.) *sc*. Supraclavicular nerves (from the cervical plexus). *ax*. Cutaneous branch of the axillary nerve. *cps* and *cpi*. Superior and inferior posterior cutaneous nerves from the radial (*ra*). *cmd*, *cm*, and *cl*. Median cutaneous, median, and lateral nerves. *me*. Median nerve. *u*. Ulnar nerve. *d*II. Second dorsal nerve. *d*XII. Twelfth dorsal nerve. *ih*. Ileo-hypogastric nerve. *ii*. Ilio-inguinal nerve. *il*. Lateral perforating branches, and *ia*, anterior perforating branches of the intercostal nerves.

FIG. 89.—Detailed distribution of the nerves to the dorsal surface of the fingers. (From KRAUSE.) *r*. Radial nerve. *m*. Median nerve. *u*. Ulnar nerve.

We can not here go into details of the different forms and varieties in the course of anæsthesia, since they will be spoken of under the different diseases which lie at the basis of the anæsthesia. The regions of distribution of the peripheral sensory nerves of the skin are given diagrammatically in the accompanying cuts (Figs. 87 to 91). Experience shows, however, that complete section of a peripheral sensory (or mixed) nerve almost never causes total anæsthesia in the corresponding cutaneous region, probably because there are many anastomoses of the terminal branches of the sensory nerves.

[* Henle's nomenclature of the peripheral nerves has been adopted.—TRANS.]

Anæsthesia in the region of the trigeminus demands a brief special description.

4. **Anæsthesia of the trigeminus** is observed in tumors, syphilitic new growths, chronic inflammations, and analogous processes at the base of the skull, which compress the trunk, the Gasserian ganglion, or one of the three branches of the trigeminus, or directly involve the nerves. Traumatic lesions of the trigeminus also are not very uncommon. The distribution of the anæsthesia, according as the affection involves the whole trigeminus, or only one branch of it, may be seen in Figs. 92 and 93, but individual variations occur. In total anæsthesia of the trigeminus, the conjunctiva and cornea, the mucous membrane of the nose, the cavity of the mouth, the tongue up to the foramen cæcum, and the soft palate are anæsthetic on the affected side. Hence we often find ulcers on the tongue and the mucous membrane, which come from being bitten. The "neuro-paralytic ophthalmia," not infrequently seen in anæsthesia of the trigeminus is of special interest, and it has been much studied by physicians and physiologists. This is an ulcerative keratitis, almost always beginning in the lower segment of the cornea, and sometimes passing over into a purulent inflammation of the whole eyeball. This affection is regarded in many quarters as an immediate result of the disturbance of special "trophic" functions, but, after careful experiments, it seems certain that external traumatic influences always occasion the first trouble, and render the intrusion of inflammatory agents possible. As a result of the anæsthesia of the eye the patient does not notice the inflammatory symptoms, which therefore often attain an extremely high degree. It is still uncertain, but not wholly improbable, that we must also assume a specially diminished power of resistance on the part of the tissues and a limitation of all compensating functions as a result of the nervous lesion.

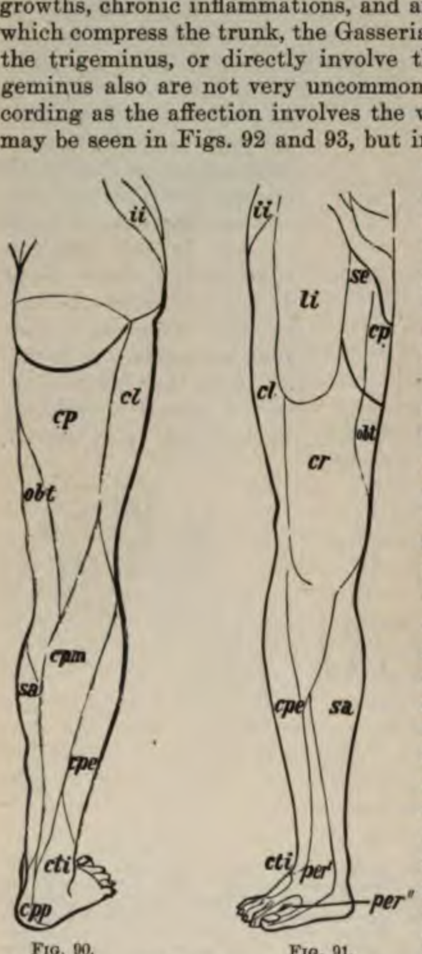


Fig. 90. Fig. 91.

Figs. 90 and 91.—Distribution of the sensory cutaneous nerves to the lower extremities. Fig. 90. Posterior aspect. Fig. 91. Anterior aspect. (From Henle.) *ii.* Ilio-inguinal nerve. *li.* Lumbo-inguinal nerve. *se.* External spermatic. *cp.* Posterior cutaneous. *cl.* Lateral cutaneous. *cr.* Crural. *obt.* Obturator. *sa.* Saphenous. *cpe.* Peroneal communicating nerve. *cti.* Tibial communicating nerve. *per'.* Superficial branch of the peroneal nerve. *per''.* Deep peroneal nerve. *cpm.* Posterior median cutaneous nerve. *cpp.* Cutaneous plantar nerve.

The skin of the face is often somewhat bloated and reddened in anæsthesia of the trigeminus, and it is sometimes warmer, but often colder, than normal. The reflexes are lost in peripheral anæsthesia, and the lachrymal secretion ceases or at least is persistently diminished. The taste is almost always decidedly diminished on the anterior two thirds of the tongue on the affected side, the territory supplied by the lingual nerve. According to the investigations of Krause on patients in whom the Gasserian ganglion has been extirpated (*vide infra*), the trigeminus contains fibers which conduct sensations of taste for sweet, sour, and salt from the tip of the tongue and the anterior two thirds of its lateral border;

idual variations seem to occur. The secretion of saliva as a rule suffers
tible change, but we sometimes see a diminished secretion of mucus in
cavity on the affected side.

es the peripheral anæsthesias just described, other anæsthesias occur,
in syringomyelia, and more rarely in other diseases of the medulla
, which are due to a lesion in the region of the trigeminal nucleus (nu-
sthesia). In those cases a remarkable condition is manifest—namely,
iseases which extend from below upward, the anæsthesia first appears
per part of the forehead next to the hairy scalp (and usually on the lat-
on before the median), and that the sensory disturbance extends in a
wnward and
to the eye-
hen to the
rtion of the
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ortion of the
finally to the
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The frontal
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efore, seems
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ich (see the
al description in the section on diseases of the medulla oblongata).

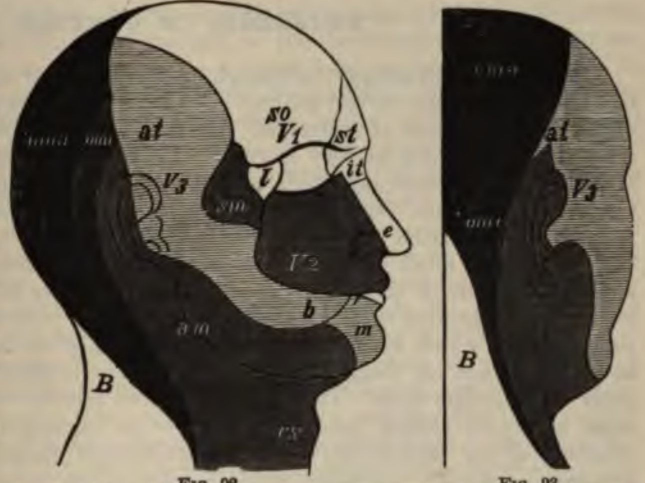

FIG. 92. FIG. 93.

FIGS. 92 and 93.—Distribution of the sensory cutaneous nerves in the head: *oma* and *omi*. Occipitalis major and minor. *am*. Auricularis magnus. *cs*. Superficial cervical. V_1. V_2. V_3. First, second, and third branches of the fifth (V). *so*. Supraorbital. *st*. Supratrochlear. *it*. Infratrochlear. *e*. Ethmoidal. *l*. Lachrymal. *sm*. Subcutaneus malæ, or zygomatic. *at*. Auriculo-temporal. *b*. Buccinator. *m*. Mental. *B*. Posterior branches of the third cervical.

rbances of sensibility in the mucous membrane of the mouth are usually
in the posterior portion of the mucous membrane of the cheeks, then in
palate, and finally in the region of the soft palate and the tonsils. The
embrane of the oral cavity, therefore, derives its sensory fibers from the
tion of the trigeminus nuclei (Schlesinger).

atment of Cutaneous Anæsthesia.—Since anæsthesia in most cases is
mptom, treatment, of course, must always first be directed against the
g disease. Therefore we will mention here only those measures which
used symptomatically against anæsthesia.

hief remedy is the electric current. We treat the anæsthetic part of the
the faradic current, using ordinary electrodes, or, better still, the wire
with the galvanic current, stroking the skin slowly back and forth with
de for three or four minutes. Sometimes we can see a result imme-
fter the sitting. Hysterical anæsthesia may often be removed in this
very short time.

es electricity we prescribe embrocations, such as spirit of camphor,
id, spirit of thyme, etc., and also baths, and cold or hot local douches,
with rubbing the skin. The action of internal remedies, such as strych-
xtremely doubtful.

It is very important to protect the anæsthetic part against external injuries. In anæsthesia of the trigeminus, particularly, we guard the eye as far as possible from the development of a neuro-paralytic keratitis, by a carefully applied occlusive bandage, and treat even the slightest inflammation with the greatest care. We can thus almost certainly avoid bad results.

CHAPTER III

NEURALGIA IN GENERAL

Concept and Ætiology.—Although every pain is, of course, excited by abnormal irritation of the nerves, still we are justified in giving a certain special variety of pain the name of neuralgia. By that name we understand pain due to a disease of a definite sensory nerve (or perhaps sometimes of its central continuation). The name neuralgia has therefore both a symptomatical and an anatomical significance. Symptomatically most neuralgias are characterized by the following peculiarities: 1, the pain is felt exactly in the course or in the distribution of one or more special nerve-trunks or nerve-branches; 2, it is usually of very considerable intensity; and, 3, as a rule, it is not present continuously, but it shows manifest variations. Many neuralgias come on in pronounced paroxysms of pain, which are interrupted by wholly painless intervals. Anatomically, neuralgia is not one single disease. Every severe affection of a sensory nerve leads to neuralgic pain. The trouble is often merely one symptom of another more extensive morbid process (symptomatic neuralgia). In other cases the affection of the sensory nerve is an independent disease by itself (idiopathic, primary neuralgia). In many idiopathic neuralgias we may with great probability assume an inflammatory affection of the nerve or the nerve-sheath, as in sciatica, but in other cases, and indeed in most typical neuralgias, as in severe trigeminal neuralgia, this theory is improbable. In these cases the special nature of the "neuralgic disease" is still entirely obscure. At times, in separate attacks and without any known cause, extremely active irritative processes occur in the special nerve territory, causing the most intense pain. Trousseau has compared these paroxysms of sensory irritation to the motor discharges of the epileptic attack, and hence has spoken of "epileptiform neuralgia."

In regard to the general occurrence of neuralgia, age has thus far an influence that most cases occur in middle life, but neuralgia also occurs in older people and much less frequently in children. Sex exerts an influence so far that certain forms, such as sciatica and brachial neuralgia, are more common in men. On the other hand, certain phases of the female sexual life, such as puberty, pregnancy, childbed, and the climacteric, favor the disposition to neuralgia. The general neuropathic predisposition, which is hereditary in the majority of cases, is of great significance. Neuralgia often appears in people who suffer from other neuroses, or in whose families other nervous diseases, such as the psychoses, epilepsy, hysteria, or neurasthenia, have repeatedly occurred. The physical constitution also seems to be of influence. We often see neuralgia in anæmic people, or in those whose constitution has been impaired by physical and mental strain, by imprudent living, or by mental excitement.

We may mention, as exciting or at least as favoring causes of neuralgia: 1. Cold, the action of draughts, wind, or wet—the so-called "rheumatic neuralgias." It is not perfectly clear how the cold acts in such cases. We usually assume that by its action slight anatomical (inflammatory?) changes arise directly or in a reflex manner in the nerves themselves. 2. Mechanical and traumatic action. Among these are, first, wounds and contusions which directly involve the nerves. Thus, for example, extremely severe neuralgia sometimes arises from the pressure of foreign bodies, such as splinters of wood or of bones in wounds, on the branch of a nerve. We may also mention here the very severe neuralgia sometimes met with after amputations. These cases are probably due less to the so-called amputation neuromata than to the pulling and stretching of the nerve-trunks in the amputation cicatrix, giving rise to the pain. To this class, too, belong many diseases in the neighborhood of nerves. Diseases of the bones or periosteum, by pressure and mechanical irritation, and often perhaps by the accompanying inflammation, may lead to neuralgia in those nerves which run through bony canals or grooves. Lastly, tumors, aneurisms, herniæ, or the gravid uterus, may lead to neuralgia by pressure on the neighboring nerves; but we must call to mind that every pressure on a nerve does not lead to neuralgia in the same way, so that we must assume a special consequent change in the nerve in "neuralgia from compression." 3. The relation which certain infections and poisons have to the development of neuralgia is very important. In the first place, it is not impossible that many of the apparently "idiopathic" neuralgias are to be referred to infectious causes—an assumption which may be made, for instance, in intercostal neuralgia associated with an eruption of zoster (*q. v.*), or in many acute trigeminal neuralgias. Many neuralgias, however, also have a definite relation to other infectious diseases. We may mention in particular the malarial neuralgias, which are directly dependent upon the malarial infection, often come on at regular intervals, and are cured by specific treatment—namely, quinine. Neuralgia is often seen during the course, or as a result, of typhoid fever, small-pox, and similar acute infectious diseases, and in the secondary stage of syphilis. Among toxic substances we may mention especially alcohol, lead, arsenic, mercury, and perhaps nicotine, as those which have a relation to the development of neuralgia. Yet in these cases it is usually not neuralgia but pain of some other kind—the arthralgia of lead poisoning, the polyneuritic pains of alcoholic subjects, etc. 4. Symptomatic neuralgia is also found in many constitutional diseases, in gout, and very often in diabetes mellitus. It is supposed that in such cases abnormal chemical influences act on the sensory nerves. 5. Finally, there was formerly much said about "reflex neuralgia," where, for example, disease of the sexual organs was thought to cause neuralgia in distant nerves. Such observations, however, can not endure strict criticism. Most of these cases are hysterical.

[Although the reflex origin of neuralgia and other nervous disturbances is very doubtful, it is well known that pain due to disease of a given organ may be referred to another part of the body. A familiar example is the reference of the pain from hip disease to the knee. This reference of pain to distant parts is probably dependent upon the anatomical distribution of the nerves. Head has made an exhaustive study of the distribution of the visceral nerves in the spinal cord, and has found that, in disease of the internal organs, the pain is apt to be referred to certain areas of the skin whose sensory nerves go to the corresponding spinal segments. The internal organs are represented in the spinal cord as follows: Heart, 1–3 D;* lungs, 1–5 D; stomach, 6–9 D (cardiac end, 6–7 D; pylorus, 9 D); intestines, 9–12 D; rectum, 2–4 S; liver and gall-bladder, 7–10 D; kidney and ureter, 10–12 D; bladder, mucous membrane, and neck, 1–4 S; over-distention of bladder, 11 D–1 L; prostate, 10–12 D; 1–3 S; 5 L; epididymis, 11 D–1 L; testicle and ovary, 10 D; appendages, 11 D–1 L; uterus, in contraction, 10 D–1 L; os uteri, 1–4 S. For the cutaneous areas corresponding to these segments of the cord see the colored plate (page 924). Disease of the internal organs may also cause referred pains in the head, perhaps through the connections of the vagus nerve. Disease of the lungs causes referred pain in the fronto-nasal, mid-orbital, and fronto-temporal areas; of the heart in the mid-orbital, fronto-temporal, and temporal areas; of the stomach and intestines in the temporal, vertical, and parietal areas; of the liver in the vertical and occipital areas; and of the ovaries and testicles in the occipital area. Diseases of the uterus, Fallopian tubes, and bladder do not give rise to referred pain in the head. Affections in the head itself also give rise to referred pains in the head. Refraction errors cause pain in the mid-orbital region; disease of the cornea or anterior chamber of the eye, pain in the fronto-nasal area; of the iris, in the temporal area; farther back in the eye, in the temporal, fronto-temporal, and maxillary areas; of the membrana tympani and middle ear, in the hyoid area; elevation of tension in the middle ear, in the vertical and parietal areas; disease of the tongue, in the mental, hyoid, and superior laryngeal areas. Dana had previously represented the regions of referred pain in visceral disturbances in much the same way.]

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* D, dorsal segment; L, lumbar; S, sacral.

General Symptomatology of Neuralgia.—The neuralgic paroxysm begins either quite suddenly, or, more frequently, after certain prodromata, such as cold feelings, prickling, slight painful sensations, etc., have preceded it for some time. The pain during the attack is usually of the greatest severity, and is described as either burning and boring, or as shooting and tearing like lightning. There are frequently short temporary remissions of the pain. The location of the pain usually corresponds precisely to the distribution of the affected nerve, so that the patient can often point out quite definitely the anatomical course of the nerve. At the height of the attack there is often an "irradiation" or shooting of the pain into the territory of neighboring nerves. External irritants, such as cold air, mental excitement, and especially movements of the affected part, often produce an increase of the pain.

On physical examination, we notice, first, certain disturbances of sensibility. The skin in the neuralgic part often shows more or less anæsthesia, which is most apparent in the intervals between the separate attacks and immediately after them. Much more frequently, however, both during the attack and during the time when the patient is free from pain, there is hyperæsthesia of the skin and the parts beneath. There are certain definite points which are often very sensitive and tender even to light pressure. These are called painful points (*points douloureux*). They were first fully described for the different forms of neuralgia by Valleix, in 1841, although in much too schematic a way, but they have considerable diagnostic importance, since they may often be found, not only during the attack itself, but also during the free intervals, although then they are less marked. The painful points always correspond to certain places in the course of the trunk or the larger branches of the affected nerve, and are found especially where, in marked and deep pressure on the nerve, we can press on some firm part beneath. They are probably always due to an abnormal sensitiveness to pressure in the affected nerve itself. In many cases of neuralgia they may of course be wholly wanting.

Motor symptoms as well as sensory are not infrequent in neuralgia. Co-existing symptoms of paralysis must always be regarded as a complication caused by some coarse lesion of the motor nerves; hence in pure idiopathic neuralgia they are entirely absent. The co-existing symptoms of motor irritation, which are often seen, are usually directly dependent upon the neuralgia, however, and hence are to be regarded chiefly as reflex contractions, due to the great irritation of the sensory nerves.

Vaso-motor symptoms are often seen in neuralgia. In the face especially, in trigeminal neuralgia, we often see a marked pallor or a decided reddening of the skin and conjunctiva. Abnormal secretions, of tears or sweat, may also be seen during the attack or at the end of it. We do not know whether all these symptoms arise from direct or reflex nervous irritation. Trophic disturbances are noticed in various ways. During the attack we see eruptions of urticaria, or still more frequently of herpes vesicles, along the course of the affected nerves, as in herpes zoster. In severe and protracted neuralgias permanent changes in the tissues have been repeatedly seen in the parts supplied by the affected nerves. Among these are a falling out or a whitening of the hair, or more rarely an abnormally great growth of hair, thickening or atrophy of the skin, staining or pigmentation of the skin, atrophy of the deeper parts, etc. Lastly we may mention that during the neuralgic attack we sometimes find a diminished frequency of the pulse.

The general nutrition of the body often does not suffer at all in neuralgia, but in many cases, especially when sleeping and eating are constantly disturbed by the attacks of pain, the disease gradually has a noticeable action on the whole constitution. The patient becomes pale and emaciated, and often the persistent and distressing pain is not without influence on his mental condition. He becomes irritable and inclined to melancholy. Patients have, in repeated instances, even committed suicide as a result of severe and incurable neuralgia.

The whole course of neuralgia shows the greatest diversity. As has been said repeatedly, the onset of the disease in separate attacks is the chief characteristic feature. The precise pathogenesis of these attacks is, of course, entirely unknown to us. These attacks usually come on every day or several times a day, or sometimes at longer regular or irregular intervals. They may last only a few minutes or several hours. During the period between the attacks many patients feel quite well, but some have still a certain sensitiveness of the skin. The disease as a whole sometimes lasts only a few days or weeks, but sometimes it persists with manifold variations for years and years, and, in a word, is not capable of improvement; although, on the other hand, there are recoveries after the disease has lasted for years. In many cases, of course, the disease depends upon the presence of some definite anatomical cause, such as a tumor, a disease of the bone, or an aneurism.

Many details will be spoken of in the following chapter.

General Treatment of Neuralgia.—Prophylaxis of neuralgia is possible in this way, that certain constitutional anomalies, such as anæmia or a general nervous predisposition, favor its appearance, as we have seen; and in attacking these conditions, we may recognize a factor which can prevent to a certain degree any subsequent development of neuralgia. It is still more important, in people who have already suffered from neuralgia, to prevent the return of the affection if we can. For this object we must first consider the strengthening of the whole body, in order to make it better resist the action of any causes of disease. The measures to be chiefly employed for this purpose are proper food, good air, baths, sea-bathing, cold bathing, gymnastics, etc. Of course, we must also particularly guard the part of the body that has once been attacked from any irritation, such as cold, mechanical and toxic irritants, or over-exertion.

In treating neuralgia itself we must always look first with great care for some causal factor, which may be accessible to treatment. This fulfillment of the causal indication is often possible in neuralgias which are due to mechanical causes. The extirpation of tumors, the excision of cicatrices, the removal of foreign bodies, the treatment of inflammatory new growths, of syphilitic affections, of aneurisms, etc., is in many cases attended by brilliant success, but, of course, in many other cases the underlying disease is unfortunately not amenable to successful treatment. We should also carry out a causal treatment in the neuralgias which are to be referred to general anæmia, to a general neuropathic constitution, to hysteria, etc. In such cases we must always ascribe great value to the general treatment, such as diet, manner of life, psychical treatment, baths, iron, and nervines, as well as to the special treatment directed against the neuralgia; and the same holds true, of course, in the neuralgias occurring in diabetes, gout, and syphilis. We must always bear particularly in mind the possibility of syphilitic neuralgia, since in such cases iodide of potassium may often have a marked curative action. We may also fulfill the causal indication in the malarial neuralgias. If neuralgia comes on at approximately regular intervals in persons who come from a malarial district, and who perhaps have already suffered from other malarial affections, the exhibition of quinine in large doses, twenty to forty-five grains (grammes 1.5–3.0) at once, will usually speedily cut short the attack. In obstinate cases in which quinine does no good, we should try arsenic, in the form of Fowler's solution. In many apparently toxic neuralgias, too, from lead, mercury, or alcohol, our first endeavor in treatment should be to remove the cause of the disease.

In all cases where the causal treatment can not be carried out, or where it alone is not sufficient, we must consider those numerous remedies and methods of treatment which correspond to the *indicatio morbi* and to the symptomatic indications. Starting with the hypothesis of an inflammatory condition of the nerve, we have often tried to exert a favorable influence on the disease by local derivatives, mustard plasters, irritating embrocations, such as spirit of mustard, veratrine ointment (two and a half per cent.), or tincture of iodine, or by blisters, or even by the cautery. The remedies first mentioned are used only in mild cases. Vesicatories, placed along the course of the affected nerve, or behind the ear in facial neuralgia, sometimes act very well in fresh cases, especially in fresh "rheumatic" cases of sciatica. We resort to the hot iron chiefly in old and very severe cases, in which, indeed, especially in sciatica, some very favorable results have been thus obtained.

The local electrical treatment of neuralgia is sometimes of undoubted benefit. We many times secure an improvement in the symptoms, which is of course temporary, even in those cases where the special cause of the disease is not influenced by the electricity, although in idiopathic neuralgias in fresh, and sometimes even in old cases, we can often obtain a complete cure. There are no general rules with regard to the methods to be employed, since different specialists have their own favorite methods. The following forms of application are most in use and are most to be recommended: 1. Stabile action of the anode of a constant current on the affected nerve-trunk over as great an extent as possible, especially on any painful points. We must entirely avoid any great variations in the current or interruptions of it. We gradually increase the intensity of the current up to medium strength. The sittings should last three to six minutes, and sometimes even longer, and they must be repeated daily. 2. In neuralgia of the larger nerves we should use a stabile descending (sometimes ascending) constant current, in which the anode is placed on the most central point of the nerve-trunk available, or on the vertebral column, and the cathode on different peripheral points. 3. The faradic current also frequently acts very well. We faradize the nerve either with a moderately strong "increasing" current, or we apply the wire brush to the skin over the affected nerves. The latter method is very painful, but it is often attended with excellent results. 4. Some electro-therapeutists, like Moritz Meyer, lay stress upon the stabile application of the anode of the constant current to any painful points on the vertebral column, such as have been [illegible] many cases of neuralgia by Trousseau.

[illegible] general rule, it is always well to begin with a mild and very cautious use [illegible] city, and not to go on to the stronger currents until later. Electricity [illegible] brilliantly at once, during the attack of pain, but sometimes no improvement is seen until after several sittings. If, after two or three weeks, we obtain no result at all, after employing different methods of applying electricity, it is the best plan to give up electrical treatment entirely as not suited to the case.

In the treatment of neuralgia we must consider a number of internal remedies, as well as electricity; some of these act symptomatically, like the narcotics, while others have obtained the reputation of having a specific action. Among the latter, quinine has decidedly the greatest value. In severe cases quinine may do excellent service, not only in malarial neuralgias, although most surely in those, but also in "idiopathic" neuralgias. It is in these cases that the remedy is given in large doses. We begin with fifteen to thirty grains (grammes 1–2) a day, best given in one dose, and in severe cases we may increase to sixty or seventy-five grains (grammes 4–5) or more. We see the best results from quinine in trigeminal neuralgia, while in other forms, as in sciatica, it is much less efficient. Beside quinine, salicylate of sodium is sometimes beneficial, especially in the rheumatic forms of neuralgia. Antipyrine, in fifteen- to thirty-grain (grammes 1–2) doses, antifebrine, in three- to seven-grain (gramme 0.25–0.5) doses, phenacetine, in seven- to fifteen-grain (gramme 0.5–1) doses, salipyrine, in twenty-to thirty-grain doses (grammes 1.5–2.0), and similar remedies, have lately been very often used. Next in rank, for continued use, come arsenic and bromide of potassium. The former is given in pill form or as Fowler's solution, five drops hree times a day, increasing gradually. Bromide of potassium acts only in large doses, forty-five to seventy-five or a hundred and fifty grains a day (grammes 3–5–10). Among the many other remedies which have been recommended in the past, but which at present are seldom used, we may mention here ergotine, internally and subcutaneously, oil of turpentine, oxide of zinc, valerianate of zinc, tincture of gelsemium, aconitine, phosphorus, subcutaneous injections of osmic acid, ten to fifteen minims (gramme 0.5–1) of a one-per-cent. solution, etc.

In all severe neuralgias the use of narcotics, especially of morphine, is unavoidable. Morphine is used almost exclusively during the attack, and is best given as a subcutaneous injection of one twelfth to one sixth of a grain (gramme 0.005–0.01) in the vicinity of the painful part. The anodyne effect almost invariably follows, but in obstinate and protracted cases the patient gradually becomes accustomed to the remedy. We must then resort to still larger doses, and these, oo, finally fail in their effect. Among the victims of the morphine habit we find many patients who have suffered or who still suffer from severe neuralgia, so that we must always be very cautious and conservative in the protracted use of morphine. We should be especially cautious before deciding to put the hypodermic syringe into the patient's own hand. Many physicians ascribe not only a palliative but a permanent benefit to injections of morphine in neuralgia. We sometimes see, in fact, that mild neuralgias recover under the exclusive use of morphine injections; but these are probably cases of spontaneous recovery. The internal use of morphine and opium preparations is decidedly inferior to the subcutaneous administration in certainty and rapidity of action. The external application of narcotic ointments, or embrocations, is much employed in practice, but it appears to be of advantage only in milder cases. We prescribe ointments with extract of opium (one to ten), extract of belladonna (two to ten), extract of opium and veratrine (one part of each to twenty of simple ointment), etc. We may also use chloroform, by applying blotting-paper wet with chloroform, or embrocations of chloroform oil, and ether as a spray. Chloral and also paraldehyde are often prescribed in chronic neuralgias for their hypnotic effect. Croton chloral has been especially recommended in neuralgia. Other hypnotics, sulphonal, etc., are also used. Finally, we must add that some physicians have praised the anodyne effect of subcutaneous injections of atropine, grain $\frac{1}{120}$ to

$\frac{1}{60}$ to $\frac{1}{20}$! (grammes 0.0005–0.001–0.003 !) at a dose, sometimes even in cases where morphine does not act.

In severe cases the surgical treatment of neuralgia is often of great importance—the section of nerves, neurotomy, or the excision of a portion of the nerve, neurectomy—in order to prevent the union of the divided nerve, or, finally, the drawing out of the nerve—so-called exairesis. A detailed account of the methods of operation is to be found in the text-books of surgery. We may state here merely that the surgical treatment of neuralgia, especially of severe trigeminal neuralgia, often has excellent results. The explanation of this success, however, is by no means easy. There is probably not only a break in conduction from the pain-exciting spot to the center, but an immediate or secondary change in the whole sensory neurone, by which the neuralgic change is made to cease. Of course a cure is not obtained in all cases. In any event, we should advise an operation only in severe cases, where all other remedies have been tried in vain, and the patient may be promised the possibility, or even the probability, of success, but never the certainty of it. Besides section of the nerves, nerve stretching has lately been frequently tried in neuralgia. We will return to this method of treatment in speaking of sciatica.

The chief question as to the use of baths arises only in the treatment of neuralgias in the domain of the nerves of the extremities, especially in sciatica, and therefore these and massage will be spoken of more in detail in connection with the separate forms of neuralgia.

We see, then, that a large number of remedies are at our command in the treatment of neuralgia, and that the choice among them is not always easy. In any given case we look for the causal indication, and try to fulfill it if possible. In the many cases, however, where we fail to find this, we must first of all try to alleviate the pain, for which purpose, if external derivatives do not suffice, our most effective remedy is morphine. We must then lay out a special plan of treatment. We try electricity, or, if this be not practicable, one of the other remedies mentioned above. We put most trust in quinine and antipyrine, especially in fresh cases; among other remedies, in anæmic persons, arsenic, and, in more robust persons, bromide of potassium are of service. If all these and similar remedies give no aid, in proper cases we may still hope for success from operative interference, or else we must confine ourselves simply to a purely symptomatic treatment with narcotics.

CHAPTER IV

THE INDIVIDUAL FORMS OF NEURALGIA

1. Neuralgia of the Trigeminus

(*Prosopalgia. Tic douloureux. Fothergill's Face-ache*)

Ætiology.—Trigeminal neuralgia is one of the commonest and most important neuralgias. In its origin many causes and predisposing factors of all sorts play a part, as was stated in detail in the preceding chapter. Many mild forms, especially neuralgia in the first branch, apparently are of acute origin and have no discoverable cause. In such cases some unknown infectious or auto-toxic influences may play a part. Not infrequently, especially in mild neuralgias of the first branch, we can not overlook the influence of exposure to cold (wind, wet) as a possible cause. Neuralgias after acute infectious diseases (influenza, typhoid), and especially malarial neuralgias, are situated most frequently in the st branch of the trigeminus, more rarely in the second, and only exceptionally the third. Trigeminal neuralgia may also be due to diseases of the cranial nes and periosteum, very often to diseases of the teeth (caries, exostoses, anom- ies of development and position of the teeth), and also to diseases of the nasal d frontal cavities and diseases of the middle ear and of the eye (inflammation d also strain of the eyes). Romberg found an aneurism of the internal carotid, essing on the Gasserian ganglion, as the cause in a severe and incurable case. e have seen a precisely analogous case. Finally, the severe trigeminal neural- as, which last for years, are of very great practical importance; for these we can ually find no cause at all, or at most a general nervous predisposition. This rm, which is the prototype of the genuine, severe, idiopathic neuralgias, comes usually in somewhat advanced life, and is perhaps somewhat more frequent in omen than in men. The special form of disease of the nerve in these cases is ill wholly obscure. The few anatomical reports as to resected nerve branches d extirpated Gasserian ganglions (*vide infra*) are contradictory and capable no positive interpretation. [Dana, Putnam, and others have found degenera- ve neuritis in the peripheral fibers of the excised nerve in these severe cases, metimes associated with obliterating endarteritis in the blood-vessels supplying e nerves. Spiller has found extensive degenerative changes in the nerve-cells of e Gasserian ganglion, but these may be secondary to the changes in the nerve- ers.—K.]

Symptoms and Course.—The attacks of pain in neuralgia of the fifth pair are ually quite intense, and in severe cases they may attain a most distressing and rrible severity. They arise either entirely without cause, or from some slight fluence, such as washing the face [making the slightest movement of the face in ting or talking.—K.], taking physical exercise, or mental excitement. They ten come on quite regularly at definite hours, especially in the forenoon, but in her cases they follow no rule. There are sometimes long, painless intervals tween the separate attacks, and sometimes the attacks follow one another in almost uninterrupted series. The lightning-like, cutting pains extend to the istribution of the affected branch of the trigeminus, but they sometimes shoot to the occiput, the back of the neck, the shoulders, etc. We can often perceive flex twitchings in the face, especially blepharospasm, and twitching of the rners of the mouth. The vaso-motor disturbances are noticed at first as abnor- al pallor, but later usually as quite an abnormal redness of the face and con- nctiva. In neuralgia of the upper two branches we often see, during the attack, n unusually great secretion of tears. An excessive flow of saliva and an in- eased secretion from the nasal mucous membrane are more rare. Sometimes, ut still quite rarely, we see herpetic eruptions in the course of the affected erve, zoster frontalis, herpes of the conjunctiva, etc. In some cases, too, more vere diseases of the eyes, belonging to the category of neuroparalytic ophthalmia, ave been observed, and also paralysis of the ocular muscles. Such complications lways point to some gross organic lesion of the trigeminus and its neighborhood. n neuralgias that have lasted longer, we often see still further trophic disturb- nces: changes (thickening or atrophy) in the skin and subcutaneous cellular tis- e, the hair turning gray or falling out in the frontal region, etc.

Most trigeminal neuralgias are situated, not in the whole distribution of the erve, but only in one or more of its branches (see Figs. 92 and 93, page 803). Ve accordingly distinguish: 1. *Neuralgia of the first branch* (*ophthalmic neural- ia*) which is especially frequent as supra-orbital or frontal neuralgia. The pain hoots chiefly from the supra-orbital foramen upward into the forehead, and metimes to the region of the internal angle of the eye and the bridge of the ose (ethmoidal nerve) or into the zygomatic region (lachrymal nerve). In this e find, as a rule, that pressure on the point of exit of the nerve at the supra-orbital foramen is more or less painful. More rarely we find painful points also on the nose, at the inner angle of the eye, or the parietal eminence, etc. 2. *Neuralgia of the second branch* (*supra-maxillary neuralgia*) is most frequent in the distribution of the infra-orbital nerve (infra-orbital neuralgia), with the chief painful point at the infra-orbital foramen, and others on the zygoma, on the upper lid, etc. The chief pain usually starts from the infra-orbital foramen, and is situated in the lower eyelid, the cheek, the upper jaw, the upper lip, and in part also in the temporal region. During the attack of pain tears are often seen in the eye. 3. *Neuralgia of the third branch* (*infra-maxillary neuralgia*) is situated chiefly in the branches of the mandibular nerve (inferior alveolar and mental nerves). The pain affects the region of the lower jaw and the chin; but sometimes the pain occurs in the temporal region, in the auriculo-temporal nerve, and in some cases it is especially distressing in the tongue, in the lingual nerve. The chief painful points are at the mental foramen and the lower temporal region.

The general course of neuralgia of the fifth pair differs very much in different cases. We see all forms, from the mildest, which rapidly passes off, to the severest and incurable types, which may drive the patient to despair. In general, neuralgia of the first branch usually belongs to the relatively milder forms; neuralgia of the second, and especially of the third branch to the severer forms. The above-mentioned "epileptiform" neuralgia (the true tic douloureux) may last for years with slight interruptions; this occurs chiefly in the second and third branches.

Diagnosis.—The diagnosis of trigeminal neuralgia is in general not difficult if only we consider the character and distribution of the pain, its occurrence in separate paroxysms, and the presence of any painful pressure points. Of course a careful local examination is always necessary to exclude the existence of inflammatory or other diseases of the bones, periosteum, and teeth. Neuralgia of the first branch may be confounded with other forms of headache, and especially with true migraine (*vide infra*), since in the latter the pain is often located in the region of the supra-orbital nerve. Only by a careful consideration of accompanying symptoms (scotoma, vomiting) can we avoid a mistake. In the severe neuralgias of the second and third branches the reflex muscular twitchings are very marked, so that, on very superficial observation, trigeminal neuralgia may be mistaken for clonic facial spasm ("tic douloureux" for "tic convulsif").

Prognosis.—The prognosis is never to be made with complete certainty. It is most favorable in fresh cases of supra-orbital neuralgia which have a manifest cause, which may be removed, as a basis; but if the affection is due to a coarse anatomical cause which can not be removed, or if we are dealing with chronic cases, especially of the second or third branch, the prognosis, unfortunately, is often very doubtful or utterly unfavorable.

Treatment.—The treatment of trigeminal neuralgia rests entirely upon the principles given in the preceding chapter. In searching for the causal indication we must look chiefly, in neuralgia of the second and third branches, for diseased teeth, and also in all cases for any affections of the nose, of the frontal sinuses, or of the middle ear. If such affections exist they should be treated in the appropriate way. Carious teeth, which are painful and which seem to have any relation to the neuralgia, should always be removed; but we must warn patients against permitting in their despair all their sound teeth to be extracted by unskillful dentists without any benefit—a thing which we have frequently known to happen. If there be a suspicion of syphilis or malaria, we should prescribe iodide of potassium or quinine. [Even in non-syphilitic cases of the severer type large doses of iodide of potassium (grammes 2.0–6.0, grain 30–90, thrice daily) combined with mercury may act very beneficially. Aconitine is of some benefit. The application of the positive pole of the galvanic battery, wet with a ten- to twenty-

-cent. solution of cocaine, may give temporary relief (electrical cataphoresis). na has obtained good results from absolute rest in bed and daily injections of ychnine (grammes 0.001–0.004, grain $\frac{1}{60}$–$\frac{1}{15}$) for several weeks.—K.]

In addition, in fresh "rheumatic" neuralgias, we should first try quinine, in es of fifteen to forty-five grains (grammes 1.0–3.0) or more, or one of the many w "nervines"—antipyrine, antifebrine, phenacetine, salipyrine, exalgine, etc. ey sometimes act very favorably, but in other cases the action is uncertain. xtures of the above-mentioned drugs (antipyrine with phenacetine, etc.) often better than a single drug. Many other remedies, which were formerly often ed in neuralgia, are at present only rarely prescribed; as, for example, butyl oton) chloral, tincture of gelsemium, aconitine, etc. Arsenic is more fre- ently prescribed at present, with somewhat more prospect of benefit.

Besides internal remedies, electricity is also to be considered. It is best given applying the anode to the painful points of exit of the affected nerve and to any ier painful points, while the cathode is held on the back of the neck. We uld use weak currents and avoid any sudden variations in the current. Other thods (faradic brush) may also be tried. Massage has also been recommended trigeminal neuralgia; but in really severe cases neither massage nor electric- is of any permanent benefit.

If a severe neuralgia persists in spite of rational treatment by electricity or igs, we should not delay too long in proposing to the patient operative treat- nt if possible. In frontal and infra-orbital neuralgia especially neurectomy is comparatively slight operation, which has many excellent results to show. en in the severe and otherwise incurable neuralgias of the third branch neurec- ny is sometimes attended with the best results. The full description of the hnicalities of the operation, as well as the description of nerve-stretching hich is much less to be recommended), must be left to the text-books on sur- y. We may add that Krause and others in very severe cases have extirpated Gasserian ganglion successfully and with benefit.

If we have to limit ourselves to a purely symptomatic treatment, injections of rphine become unavoidable in severe cases, but we should restrict their use much as possible. The above-mentioned nervines have sometimes a symptom- c action. We can sometimes lessen or shorten the paroxysm of pain by pres- e on the nerve or by compression of the carotid.

2. OCCIPITAL NEURALGIA

Of the neuralgias involving the sensory region of the upper four cervical rves, neuralgia of the occipitalis major (Figs. 92, 93) is relatively the most quent and practically the most important. Besides the factors to be considered all neuralgias, we must pay particular attention, in regard to ætiology, to dis- es of the upper cervical vertebræ—caries and new growths. The painful par- sms may attain the greatest severity. They are usually located in the two ipital nerves at once, although often more severe on one side than on the ier. Painful points are most frequently found midway between the mastoid cess and the upper cervical vertebræ. Vaso-motor disturbances, falling out of hair, etc., have been often observed.

The prognosis depends upon the variety of the disease. In primary neuralgic ections it is generally favorable. In such cases, if fresh, a rapid recovery may en be brought about by salicylate of sodium, antipyrine, the constant current, sometimes by strong cutaneous irritants to the back of the neck (blisters). In ier cases, of course, which are due to some severe disease (vertebral caries, mors, etc.), the prognosis is often utterly unfavorable, and treatment can be ly symptomatic (morphine, etc.).

Other neuralgias in the distribution of the cervical plexus are rare. They occur in the distribution of the occipitalis minor, when, according to Seeligmüller, they are quite frequently due to syphilis, and are easily cured by iodide of potassium, and in the distribution of the great auricular and the supra-clavicular nerves. A phrenic neuralgia even has been described, in which the pain extends along the course of the phrenic nerve to the insertion of the diaphragm; but this is at all events very rare. In these cases, as in many of the so-called visceral neuralgias, we probably mistake quite different forms of disease or hysterical symptoms for neuralgia.

3. Neuralgia in the Region of the Brachial Plexus

(Cervico-brachial Neuralgia)

Brachial neuralgia is, on the whole, rare, and it is hardly ever limited strictly to the distribution of a single nerve. In general, the radial and ulnar nerves are rather more frequently affected than the median. We also see at times neuralgia of the internal cutaneous nerve and other cutaneous branches, and neuralgia of the sensory branches of the axillary (circumflex) nerve. In regard to ætiology, we have to mention first wounds and contusions of the nerves, and also cicatrices and foreign bodies. The amputation neuralgias also belong to this class. Severe neuralgia in which the pain may extend over a greater part of the whole arm is sometimes seen after injury of the fingers (crushing, cuts, etc.). In some of these cases we may have to do with an ascending neuritis, starting from injury of a small nerve branch; in other cases there are probably cicatricial contractions, thickenings of the neurilemma, or small neuromata developing after the injury, which excite the pains. The "amputation neuralgias," which are often very severe in amputation stumps, are due to neuromata which form on the cut ends of nerves. Severe neuralgia often arises from pressure on the brachial nerves, as in cases of tumors in the axilla or neck (cancer, etc.), aneurisms of the aorta, etc. Double brachial neuralgia should always excite suspicion of disease in the neighborhood of the upper posterior nerve-roots, especially of cervical pachymeningitis, spondylitis of the lower cervical vertebræ, etc.

If we except the neuralgias secondary to trauma and compression already described, true primary neuralgia in the region of the brachial nerves is very rare. We may sometimes, and most frequently, speak of neuralgia coming on after exposure to cold, although the distinction between this form of neuralgia and true neuritis (*q. v.*) is often very slight. If we hold less strictly to the conception of neuralgia and include also the "nervous" pains not limited to the region of one special nerve and not due to any other discoverable local disease, we may find such "brachialgias" more frequently. They come on chiefly as one symptom of hysteria or neurasthenia in persons with a nervous predisposition, and are often very obstinate and apparently severe. We must also pay especial attention to the patient's calling and occupation. In seamstresses, piano-players, persons who write much or do much other work with the arms supported, etc., we sometimes see quite severe brachialgia for which we can find no other cause—the so-called "occupation neuralgias." A general nervous predisposition also comes into play not infrequently in such cases.

We must mention finally those brachialgias which are dependent upon arterio-sclerosis. In the true stenocardiac attack (*vide* page 343) the pain often shoots into the left arm. Many brachialgias, apparently associated with gout or diabetes mellitus, may also be dependent upon such vascular changes and the disturbances of nutrition caused by them in the peripheral nerves.

We have little to add concerning the special symptomatology of brachial neuralgia. The pain, as we have stated, is usually ascribed to the whole course of the nerves without being very exactly localized. It is greater either in the upper arm in the forearm and fingers. Painful points are sometimes found over the braiial plexus, over the radial nerve on the external surface of the upper arm, over ie ulnar nerve in the sulcus at the internal condyle, over the median nerve at the iner border of the biceps, and where the cutaneous nerves emerge from the fasciæ. aso-motor and trophic disturbances, such as "glossy fingers," a peculiar, shiny, rophic condition of the skin of the fingers, eruptions of herpes, etc., are often en in true organic neuralgias, and sometimes we also see a pronounced atrophy the whole arm. The diagnosis of neuralgia as such is usually easy; we must ar in mind only the risk of confusion with articular affections, and especially ith beginning arthritis deformans. It is sometimes much harder to determine ie cause of the neuralgia. This always requires a careful general investigation id close attention to all the conditions which may be involved.

The treatment of brachial neuralgia is often no easy task, since we frequently ive to do with protracted and obstinate diseases. The causal indications should met if possible; besides these we should make use chiefly of electricity (the deending galvanic current along the affected nerves). To relieve the pain we may so use salicylate of sodium, antipyrine, phenacetine, etc., and finally local arm packs, peat-baths, sand-baths, etc. We should be very guarded in using ircotics (morphine). If the brachialgia be associated with neurasthenia or rsteria, we can obtain success only by an appropriate general treatment with a ychical suggestive influence. In some severe cases of true neuralgia we must nsider the possibility of surgical interference, such as nerve-stretching, the tirpation of cicatrices, etc.

4. INTERCOSTAL NEURALGIA

(*Dorso-intercostal Neuralgia*)

The neuralgias of this class are almost always pure intercostal neuralgias, nce the posterior dorsal branches of the thoracic nerves are only exceptionally volved. The middle intercostal nerves, from the fifth to the ninth, are usually fected, one or more of them being attacked at the same time. The affection is uch more frequent on the left side than on the right.

In regard to ætiology, it is important to remember that obstinate intercostal uralgias are often a symptom, and for a long time the only symptom, of severe ganic disease, especially affections of the ribs; diseases of the vertebræ, such caries, carcinoma, and actinomycosis; diseases of the cord, such as tabes, mengeal tumors, syphilis, and aneurism of the aorta. Genuine idiopathic interstal neuralgia, however, is also met with, as well as these symptomatic forms, it, according to our own experience, less often than we might imagine from any of the statements. Intercostal neuralgia has been reported, especially in iæmic and nervous women, both in youth and middle age. Traumatic lesions the intercostal nerves and taking cold also play a part in the ætiology.

The pain in intercostal neuralgia may attain a remarkable severity, and it is iually increased by any considerable movement of the thorax. Hence the paent avoids deep inspirations, coughing, loud talking, etc., as much as possible. e usually find three painful points—one near the vertebral column, one somehere in the middle of the nerve, and a third near the sternum or over the rectus dominis. We may mention, among the trophic disturbances, the comparatively equent occurrence of herpes zoster. In such cases we probably always have an tual neuritis of one or more nerves. The pain precedes the eruption of zoster, comes on at the same time with it. It often lasts for a long time after the itaneous affection has healed. The special method of origin of the zoster vesles is still obscure. Recent careful anatomical investigations (A. Dubler) vor the hypothesis that the formation of vesicles arises simply from a direct extension of the inflammatory process from the terminal branches of the nerves to the skin. The hypothesis of special trophic disturbances would therefore be unnecessary. It is worthy of note that the attacks of zoster often exhibit a certain epidemic and sometimes even an endemic distribution, so as to suggest an infectious agency. The almost constant swelling of the neighboring lymph-glands, in the axillæ, at the lower border of the pectoral muscle, etc., also favors the theory of the existence of a true inflammatory neuritis in herpes zoster. We can often find a slight diminution of sensibility on the skin between the individual vesicles.

The course of intercostal neuralgia depends chiefly upon the ætiology of the affection. Primary neuralgias are often quite obstinate, but on the whole they usually give a favorable prognosis. This is especially true of the intercostal neuritis associated with herpes zoster. The differential diagnosis between genuine intercostal neuralgia and rheumatic affections of the muscles, incipient pleurisy, etc., is not always easy. In these cases a careful physical examination, a consideration of the localization of the pain, of the presence of painful points, and of the whole course of the disease, are needed to protect us from errors. Stenocardiac attacks (see page 343) are sometimes falsely regarded as intercostal neuralgia, and we must also remember that severe diseases of the vertebræ or the spinal meninges may at first assume the aspect of intercostal neuralgia.

The treatment is governed by the general rules given in the previous chapter. Cutaneous irritants, mustard plasters, or blisters often act very well in fresh cases. Local anæsthesia (chloroform, ether), especially a spray of methyl or ethyl chloride, may occasionally be of benefit. Electricity is given by the faradic brush or the constant current; with the latter, the cathode is placed on the vertebral column, and the anode on the lateral and anterior painful points, using quite a strong stabile current. All the usual anti-neuralgics (antipyrine, phenacetine, exalgine, etc.) may be used internally. In severe cases we can not avoid injections of morphine. Herpes zoster heals by simple treatment with lanoline or by dusting on powders, such as one part of zinc oxide to two of starch.

Mastodynia (*Neuralgia of the Mammary Gland*).—Mastodynia ("irritable breast" of Astley Cooper) is to be considered as a special form of nerve pain in the distribution of the intercostal nerves. It occurs almost solely in women, after the age of puberty, although it may occur in men, and is a very painful, distressing, and obstinate affection. The pain is either continuous, or it comes on in separate paroxysms, sometimes accompanied by vomiting. The whole breast, or more frequently different parts of it, is extremely sensitive to the touch. We know little that is definite as to its ætiology. Anæmia, hysteria, and traumatic action seem to have some influence on it. We sometimes feel little nodules in the breast, which are very painful (*tubercula dolorosa*, neuromata?), and which may sometimes give rise to the suspicion that a carcinoma is developing.

The disease may last for years. Treatment is difficult. Warm packs to the breast, bandaging the breast, and especially narcotics, may afford relief. Electricity may be of decided service. In the worst cases operative interference has been attempted—amputation of the breast, or extirpation of the painful nodules—but its results are uncertain. Nägeli highly recommends stretching of the whole breast *en masse*: the breast is seized with both hands and stretched and extended for several minutes with special regard to the tenderest points.

5. Neuralgia in the Region of the Lumbar Plexus

As the neuralgias of this class are rare, and show few peculiarities, we will content ourselves with a brief account of the most important forms.

Lumbo-abdominal neuralgia causes pain in the lumbar region, which shoots into the buttocks, the hypogastrium, and the genitals. Crural neuralgia is seated, part, in the region of the external anterior cutaneous nerve of the thigh, and in rt in the region of the cutaneous branches of the crural nerve, the internal and iddle cutaneous. Its distribution over the cutaneous distribution of the great phenous nerve, the inner portion of the calves, and the inner border of the foot, especially characteristic. In obturator neuralgia the pain extends along the side of the thigh, down to the vicinity of the knee-joint (see Figs. 90 and 91, ge 802).

In their individual characteristics, all these neuralgias agree with what has en said in the previous chapter. The diagnosis is not always easy, and we must y special attention to avoid confusing them with affections of the bones and ints, with lumbago, renal colic, etc.

We must mention here a special symptom-complex, *meralgia parœsthetica* Bernhardt, Roth), the chief symptoms of which are pain, paræsthesia, and paral anæsthesia on the outer side of one or both thighs—that is, in the territory the external cutaneous femoral nerve. Mechanical factors in particular (longntinued walking, the pressure of articles of clothing, hard work), or in some ses exposure to cold or acute infectious diseases, seem to excite this trouble, hich is sometimes very persistent. The treatment consists of embrocations, ectricity, baths, etc.

6. Sciatica

(Ischiatic Neuralgia. Ischialgia. Malum Cotunnii)

Ætiology.—Next to trigeminal neuralgia, sciatica is by far the most freuent, and practically the most important form of neuralgia. The anatomical urse of the sciatic nerve renders it especially exposed to mechanical injuries nd the influence of cold. Many cases of sciatica are certainly due to such uses as over-exertion of the leg in hard work, sitting for a long time on an ncomfortable seat, exposure to wet and cold, etc.; and this is why sciatica is in eneral far commoner in men than in women, and also much commoner in the boring classes than in the higher classes. Certain ætiological conditions may f course provoke sciatica in women also, especially the pressure of the gravid terus on the sacral plexus or mechanical injury of the plexus in difficult labor forceps, etc.). Venous stasis in the pelvic veins (hæmorrhoids) and habitual onstipation more rarely seem to give rise to the development of sciatica.

The relations of sciatica to certain constitutional and toxic general diseases re of practical importance. Thus a simple or even a double sciatica is not very re in diabetes mellitus, and it is sometimes seen in true gout (arthritis urica), articular rheumatism, following syphilis or gonorrhœa, in alcoholic subjects, tc. We must also bear in mind the occurrence of apparently pure sciatic pain tabes dorsalis.

Symptomatic neuralgia in the region of the sciatic nerve is also seen in pelvic imors, caries of the sacrum, and similar diseases.

Symptoms and Course.—Sciatica takes a somewhat different position from e other forms of neuralgia, since the sciatic pain does not usually show the ecial characteristics of true neuralgia in a fully pronounced fashion. In parcular, the individual paroxysms of pain are rarely so pronounced and so interipted by free intervals in sciatica as is the case, for example, in many trigemal neuralgias. Although an increase or a diminution of the pain is common in iatica, the pain on the whole gives us more definitely the impression of a peristent neuritic pain, and we can therefore scarcely be mistaken if we consider e anatomical cause of most cases of ordinary sciatica to be an actual sciatic euritis.

The pain usually begins in the lumbar and sacral regions and then gradually asses downward, following the course of the sciatic nerve, through the gluteal region and the posterior surface of the thigh to the hollow of the knee, and then down to the peroneal region (outer part of the leg, outer edge of the foot, and back of the foot), or more rarely to the tibial region (sole of the foot). The accuracy with which many patients point out with the finger the distribution of the pain, corresponding accurately to the anatomical course of the nerve, is a most characteristic feature for the diagnosis of sciatica. Individual cases, however, differ considerably, since at times the pain is felt to a marked degree only in the upper portion and hardly extends at all to the lower leg, while in other cases the severest pain is felt below the knee. This is probably due to the fact that the neuritis, which is the cause of the pain, affects one or another part of the nerve, being chiefly either in the upper root portion or in the terminal branches. The pains themselves are rarely described by the patients as "darting," but more commonly as "burning," "boring," etc. The pain is often worse at night than during the day. It is increased by different movements of the leg, by an uncomfortable position, by pressure, by cold, etc. Patients with severe sciatica are therefore almost completely bedridden, and they can take a few unaided steps only with the greatest pain. If we make the patient stand, there is often, but of course not always, a very characteristic way of holding the body. In order to take the weight of the body off the affected, tender leg, the trunk is bent over to the opposite side, and the spine thus becomes scoliotic, the convexity being usually toward the healthy side (sciatic scoliosis, see Fig. 94).

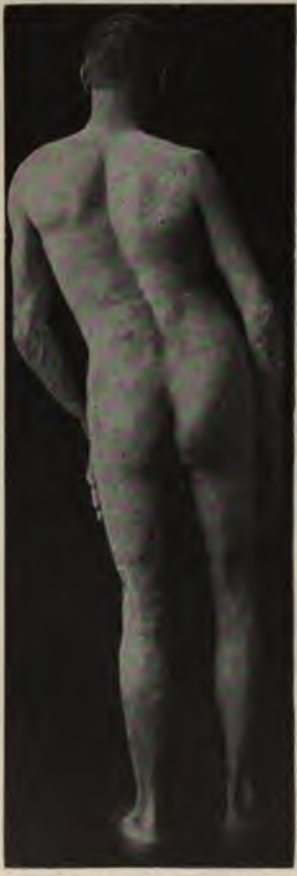

FIG. 94.—Sciatic scoliosis in severe sciatica. (Personal observation.)

If we examine the affected leg more closely, we shall notice first the sensitiveness of the sciatic nerve to direct pressure. This is often not so great as we might expect, probably because the upper part of the nerve, which is not so easily accessible to pressure, is chiefly affected; but we frequently find the nerve itself quite sensitive to pressure in the middle of the glutæus or at its lower border, in the hollow of the knee, or at the head of the fibula and elsewhere. If the patient be in a lying or sitting position and the thigh be strongly flexed, passively, on the trunk, while the lower leg is at the same time kept extended, the procedure gives rise to a gluteal pain which is characteristic. By this maneuver the sciatic nerve is apparently irritated by being stretched so much. Among the other nervous disturbances we may mention an actual paresis of the muscles supplied by the sciatic nerve (for example, the flexors of the lower leg); this is rare, and, as a rule, indicates more deeply-seated disease. The muscular hypertonicity (in part of reflex origin) is more striking; it sometimes takes the form of actual tremor or twitching of the painful leg. A peculiar muscle-wave (the so-called "myokymia") is also seen in sciatica. The patellar reflex is usually unchanged; but in severe sciatica we have quite frequently, although, of course, not always, seen the gastrocnemius reflex, obtained by striking the Achilles tendon, absent on the affected side; this is of value in diagnosis. [The patellar reflex is often somewhat exaggerated on the affected side in the early stages of sciatica.

and diminished in the later stages.—K.] On careful examination we sometimes find a slight disturbance of sensibility, but it is very rarely marked. In long-continued cases there is almost always a very noticeable atrophy of the affected leg. The circumference of both the thigh and calf is usually several centimetres less on the affected side than on the healthy side. This atrophy is possibly connected with vaso-motor disturbances, for the skin of the affected leg often feels decidedly cooler than that of the healthy side. The atrophy, however, is due mainly to the inactivity of the painful leg. [In a considerable number of cases quantitative or qualitative changes in the electrical reactions are found on the affected side. This would indicate that the atrophy was degenerative in character, due to neuritic changes in the nerve.—K.]

Sciatica rarely lasts less than several weeks, and in many cases months or even years may elapse before the pain entirely disappears and the leg completely regains its normal usefulness. The course, however, often shows very marked variations. In simple sciatica (not due to some other underlying disease) the final outcome is usually favorable. We must bear in mind, however, that the disease has a marked tendency to recur, so that any one who has once had sciatica must avoid with special care all exposure which may cause a return of the suffering. The general course of symptomatic sciatica is of course dependent mainly upon the nature of the underlying disease.

Diagnosis.—The diagnosis of sciatica is easy in the majority of typical cases, but it may sometimes be quite difficult. It is chiefly confused with lumbago, an acute coxitis, nervous coxalgia (*vide infra*), and psoas abscess. Even when the diagnosis of sciatica as such is definite, we must always consider with care the patient's general condition to determine whether we are dealing with a simple primary sciatica (primary sciatic neuritis) or with a symptomatic sciatica following some other underlying disease. In the section on ætiology above we have mentioned the possibilities which the physician must especially bear in mind. In cases where there is a suspicion of simulation (as in accident cases), we must note the accurate localization of the pain and the tender points, and especially the objective symptoms, the almost invariable wasting of the affected leg, and the frequent absence of the Achilles tendon reflex.

Treatment.—In ordinary primary sciatica and in fresh cases we usually begin with "anti-rheumatic" treatment. The patient should be put to bed and kept warm. We ourselves have treated many cases of sciatica with very good results by methodical sweating (hot-packs, hot-air baths). Internally, we would first prescribe salicylate of sodium, antipyrine, salipyrine, or phenacetine, from which we often obtain at least an improvement in the symptoms. We have never seen any good effect from oil of turpentine, which is often used in England. We have as little faith in the injections of osmic acid which are recommended (*vide* page 809). "Local derivatives," especially blisters along the nerve, are at present somewhat out of fashion, but we have often seen decided benefit from blisters, and we therefore use them quite frequently in obstinate cases. Warm fomentations, a Priessnitz's bandage about the entire leg, etc., usually give relief, and also embrocations with chloroform oil, a spray of chloride of methyl, etc. If the pain is very severe, so that sleep is constantly disturbed, we must use narcotics, especially subcutaneous injections of morphine. The author has not been able to bring himself to employ methodical treatment by morphine injections, as has been recommended from many sides.

Of other methods of treatment, besides those already mentioned, we may consider electricity, massage, nerve-stretching, and baths.

In electrical treatment we usually employ quite strong descending currents, with large electrodes, which we let act on the nerve for five or ten minutes a day, while we include one portion of the nerve after another in the current. Where there is much stiffness in the leg, we open and close the current a few times, in order to excite muscular contraction. Many cases are suitable for the use of the faradic current, especially for the wire brush. Besides electricity, massage has often given excellent results in sciatica. Details of the technique to be employed may be found in the special treatises on this important method of treatment.*

Baths (douches, rubbing) are beneficial in most cases of persistent sciatica. The regular use of simple warm baths (half an hour daily at a temperature of 27°–28° R. [93°–95° F.]) is of benefit, especially when combined with massage or electrical treatment. We see still more favorable results at certain bath cures, especially in Wiesbaden and also in Wildbad, Teplitz, etc. Peat-baths, and especially hot sand-baths (Köstritz, Blasewitz), may have a good effect.

Operative nerve-stretching may be tried only in very old and very obstinate cases. In some cases it has been attended by benefit. We have repeatedly employed bloodless nerve-stretching with satisfactory results. This is done by putting the patient on the back and then passively flexing the thigh against the trunk as much as possible, and keeping it there for two or three minutes each day. [This may be done, if necessary, under ether. If the lower leg be kept extended the stretching is greater.—K.]

It is very important always to pay attention to the general as well as the local treatment, in order to remove any active causative conditions. A change in the mode of life, avoidance of rheumatic, toxic, and mechanical injuries, regulation of the bowels, etc., are sometimes attended by visible results. Any underlying diseases must of course be especially treated (iodide of potassium in syphilis, anti-diabetic treatment, etc.).

[Mention should be made here of a method of treatment advocated by Weir Mitchell, which is occasionally useful in other forms of neuralgia as well as in sciatica. It consists in absolute rest of the affected limb by application of a splint. With this he combines the application of cold, by an ice-bag over the length of the nerve, which may be continued for a number of hours.—K.]

7. Neuralgia of the Genitals and the Rectal Region

Neuralgic affections of the parts named are not frequent, but still a number of cases have been described by different observers. Of course most of these observations are of ancient date and many of them are at present perhaps to be explained in some other way.

The pain in these cases has its seat either in the external genitals, or in the urethra, or in the anal and perineal region. The most frequent form is spermatic neuralgia ("irritable testis" of Astley Cooper), in which there is the most intense pain in the spermatic cord and the testicles, which is almost always associated with an extreme hyperæsthesia of the affected parts. The treatment of this form of neuralgia by narcotics and electricity is often unsuccessful, so that in severe cases resort has sometimes been had even to castration. In women, genuine uterine and ovarian neuralgia does occur, but most cases are either symptomatic neuralgia or a manifestation of hysteria.

Coccygodynia is the name of a form of severe pain in the coccygeal region, seen usually in women, which is much increased by walking, defecation, etc. The affection is so distressing that operations have been repeatedly performed on account of it to remove or to cut around the coccyx. We have twice seen this same symptom as a complication of tabes.

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* Busch, "Allgemeine Orthopädie, Gymnastik u. Massage," Leipsic, Vogel, 1882; Schreiber, "Praktische Anleitung zur Behandlung durch Massage," Vienna, 1883 [translated by Mendelson, Philadelphia, 1887]; Reibmayr, "Die Massage und ihre Verwerthung in der prakt. Medicin," Vienna, 1883; Hunerfauth, "Handbuch der Massage," Leipsic, 1887, etc. [Graham, "A Treatise on Massage," New York, 1890; Eccles, "The Practice of Massage," London, 1895; Kleen, "Handbook of Massage" (translated by Hartwell), Philadelphia, 1892].

8. Achillodynia. Talalgia. Tarsalgia. Metatarsalgia

For practical reasons we will mention briefly here a number of peculiar and often very painful affections of the feet.

Achillodynia is the name given to a pain at the insertion of the Achilles tendon, which is often very obstinate and a hindrance to walking. The trouble often seems to be connected with gonorrhœa, in other cases with gout, injury, over-exertion, hysteria, etc. We can sometimes make out a slight local swelling, but often there is no objective change. The treatment consists of rest, inunction of mercurial ointment, warm baths and packs, attention to any existing underlying disease, etc.

Pain in the heel is called talalgia or tarsalgia. Mechanical causes (long-continued standing or walking, the pressure of boots, etc.) often seem to be of significance, but sometimes we can not discover any cause. We must of course always consider inflammatory changes, flat-foot, etc. Baths, massage, and galvanism are most to be recommended.

Metatarsalgia (*névralgie métatarsienne, Morton's disease*) is an extremely distressing and severe pain, chiefly in the neighborhood of the fourth and fifth metatarso-phalangeal joints. The trouble is observed especially in women in middle life, and it may be sometimes connected with wearing ill-fitting shoes. In other cases injury, gout, etc., are regarded as causes. [It is often due to the pressure upon the nerve between the two phalangeal ends of the metatarsal bones, and it may be relieved by pressure upon the tarsal ends, which causes the phalangeal ends to separate.—K.] The treatment consists of prescribing proper shoes, rest, local fomentations, baths, etc. In some severe cases the painful bones have been resected with success.

There are also other forms of pain in the feet besides these mentioned, which are usually hard to explain. Hysterical conditions certainly play a part in some cases, and are to be taken into account in treatment, but in other cases we often have to deal with mechanical and inflammatory conditions which are hard to remove.

9. The so-called Articular Neuralgias (Articular Neuroses)

Neuralgia of the joints, first described by the English physician Brodie, was first generally known in Germany after Esmarch proved, by publishing many observations, that apparently severe and very painful diseases of the joints are often found, which are due to no discoverable anatomical disease of the joint, and which we are therefore justified in regarding as nervous affections. Since the localized pain in the joint is the chief symptom in most of the cases of this class, the somewhat unfit term "neuralgia of the joints" has been chosen, although we do not find here, as a rule, such typical and paroxysmal attacks of pain as in genuine neuralgia, and although, too, a number of other symptoms are usually present, which do not occur in genuine neuralgia.

We see neuralgia of the joints chiefly in nervous persons disposed to hysteria, and therefore more frequently in women and girls than in men, but it is also quite common in children. We can very often make out a psychical cause for the origin of the disease. The affection is often caused by injuries which affect the joint, and which would be in themselves without significance, if they were not associated with a decided fright, and did not direct the patient's thoughts to the affected limb. Articular neuralgias do not, therefore, belong to the pure neuralgias, but are symptoms of hysteria (traumatic hysteria).

Either immediately after such an occurrence, or often only some weeks later, the patient begins to complain of pain. The knee- or hip-joint is almost always affected, only rarely the joints of the upper extremity. The pain is continuous, but it is more severe at times, especially on motion, or mental excitement. At other times, especially if the patient's attention be diverted from the trouble, it seems to diminish to a considerable extent. It is localized mainly in the joint, but the whole leg is often painful. The patient is usually very sensitive to pressure or jarring, and sometimes we may even discover some painful points on pressure over the joints. The patient can not walk at all, or at least walking is very painful, and she limps badly. In severe cases, especially if the excessive sympathy of those about her reduces the patient's power to resist her suffering, she becomes completely bedridden for weeks or months. There is usually a decided weakness in the affected leg, which is almost always associated with great muscular rigidity and tension. The leg is extended, or flexed, and rotated inward, in just the same way as in genuine coxitis.

The diagnosis of articular neurosis is often quite difficult, but it is almost always possible, with long observation of the case. At first, of course, the disease often seems to be a severe affection of the joint, on account of the great pain, the rigid position, and the complete inability to use the leg. The experienced physician, however, is usually struck by the absence of any definite physical changes in the joint, especially of swelling, and also by the changes in the intensity of the complaint, by the influence of mental emotion on the suffering, and finally by the general impression he gets from the patients, the way they behave, and the contrast between their evidently exaggerated complaint and their usual (though, of course, not invariable) good appearance, appetite, and undisturbed sleep. If the patient's attention be diverted, the same pressure on the joint, which before was unendurable, is not even felt. In doubtful cases an examination under chloroform is very advisable. With this, contractures apparently the most severe vanish, and the normal character and mobility of the joint become plainly manifest.

As soon as the diagnosis of an articular neurosis is made the treatment has quite definite indications. All embrocations, poultices, bandages, etc., are to be laid aside. The patient is to be brought to the conviction that the pain is dependent only upon the morbid anxiety, and that walking is indeed quite possible if she will only first learn to will to walk again. We therefore make the patient practice walking methodically; these attempts at first prove very poor and apparently distressing to the patient, but they often lead very speedily to better results. These exercises are very much aided by electrical treatment of the joint, passing a strong current through it or using the faradic brush, and also by local cold douches and massage. Under some circumstances the use of internal remedies may be indicated, in many cases with regard only to the mental condition. We give iron to anæmic patients, and also the nervines. (See the chapters on hysteria and traumatic neuroses.)

10. ACROPARÆSTHESIA

Certain symptoms of sensory irritation, to which F. Schultze has given the name of "acroparæsthesia," have only of late years aroused general attention (Laquer and others), although they have long been familiar to all experienced physicians and have been repeatedly described in medical literature. It is, in fact, quite a common complaint, the chief symptom of which consists of an unpleasant and often even very painful sensation at the tips (ἄκρα) of the extremities, especially in the hands, finger-tips, feet, and toes. This sensation is almost constant, or at least it recurs very frequently.

The trouble usually appears in middle life, and it is said to be somewhat more frequent in women than in men. In many cases we can find no special cause, but sometimes we can discover certain injurious influences which have affected the hands or feet, such as past exposure to cold, freezing, much washing in cold water, fatiguing mechanical occupations, chemical irritants, etc.

The abnormal sensations are usually described as darting, prickling, tearing, etc. Their chief seat is, as has been said, in the fingers and finger-tips. Both hands are usually affected, but the trouble is often more pronounced upon one side than upon the other, and certain fingers of the same hand are sometimes more painful than others. The feet (toes) are more rarely affected than the hands; in such cases venous stasis (varicose veins) seems to us to have a certain significance in the ætiology. We must also consider arterio-sclerotic changes in the vessels (see page 373). The pain is not equally severe at all times; it is often worse at night and toward morning, ceasing during the day.

If we examine the hands or feet closely we can sometimes detect no objective abnormality, but in many cases certain "vaso-motor" phenomena are first of all manifest. The painful parts are cooler and more cyanotic than under normal conditions, but we have also seen very obstinate cases in which the hands usually felt hot and sweated constantly and profusely. The sensibility is normal, or there may be in some parts a very slight blunting of tactile sensibility, or more rarely a marked hyperæsthesia to touch. Motion, as a rule, is undisturbed, but many patients complain of a certain stiffness of the hands. In general, however, it is the pain, often very severe, which limits the usefulness of the hands, and often greatly disturbs the patient's work. There are no special changes in the reflexes. In some cases we have seen very marked trophic changes, especially a manifest smallness and tapering of the terminal phalanges, marked curving of the nails, a shininess of the skin (glossy skin), etc. A short time ago in a very obstinate case, which had lasted for years, we saw torpid ulcers develop on the toes without any external cause; they were very painful and showed no tendency at all to heal.

The course of the disease is in some cases benign, recovery taking place after several months; but in many other cases acroparæsthesia is a very obstinate, chronic condition, lasting for years, and, in spite of its lack of danger, causing the patient great discomfort and distress.

Nothing definite is known as yet as to the nature and special origin of the disease. It is generally assumed that it is an irritative condition of the sensory terminal nerve-fibers and terminal apparatus—that is, that it is a purely peripheral trouble; but it is still uncertain whether we have to do with a primary disease of the nerves themselves or with a disease of the smallest terminal arteries and consequent circulatory disturbances with their sequelæ.

The diagnosis of acroparæsthesia is in general easy, since the picture of the disease is in fact very characteristic; but we must always bear in mind that such paræsthesiæ sometimes occur as symptoms or precursors of organic central diseases (for example, as precursors of apoplexy, in other brain diseases, in tabes, diabetes, etc.).

Symptomatic treatment is all that is possible, and in this regard the local use of electricity, especially the faradic brush, seems to give the best results; but the constant current, electrical hand-baths, etc., are sometimes of benefit. Experience must decide in the individual case as to the influence of thermic procedures (warm and cold hand-baths, cold douches, the Scotch douche, etc.). Besides local treatment, we must also pay attention to the patient's general condition (iron, quinine, arsenic in anæmia, etc.). [Drugs which act upon the blood-vessels, such as ergot, nitroglycerine, or the nitrites, are sometimes of service.—K.]

CHAPTER V

HABITUAL HEADACHE

(*Cephalæa. Cephalalgia*)

In addition to the neuralgias, we must speak here of habitual or "nervous" headache, an affection which is very often met with in practice, but in regard to whose precise causes or whose special nature our knowledge is still in many respects very unsatisfactory.

We do not term the symptomatic headaches, so often observed, "nervous headaches." The former come on in acute febrile infectious diseases, in pronounced general anæmia, in the different anatomical diseases of the brain and its membranes, of the skull, the frontal sinuses, etc. Just as little ought we to confuse habitual headache with other painful and well-characterized affections, especially typical neuralgia in the frontal branch of the trigeminus, or in the occipital nerves, or with genuine migraine or hemicrania (*vide infra*). The cases which belong to this class are rather those in which the headache forms to a certain degree a disease in itself, and is the sole, or at least the chief, symptom of which the patient complains, and for which he seeks aid. We know no definite anatomical basis for them. We usually assume disturbances of circulation and of the fine nutrition as the special causes of headache; but the nature of these changes is still wholly unknown. We can also state little that is definite as to the special place where these pains arise. We do not know positively whether painful irritations may arise in the brain-substance itself. In case pain should be produced by irritation of the sensory parts of the brain, it would probably not be felt in the head, but it would be projected toward the periphery of the body. The cerebral meninges, however, especially the dura mater, are decidedly sensitive, and hence they may usually be regarded as the special seat of headache.

The manifold character of the circumstances under which headaches arise renders it probable that the causes of headache are very different in different cases. We have to do either with persons who seem perfectly healthy in other respects, or with weak and anæmic people, or again with robust, very well nourished, "full-blooded" persons with red faces. Hence we look for the cause of the pain, according to the general constitution of the patient, either in an abnormal hyperæmia or in an anæmia of the brain and its membranes—hyperæmic or anæmic cephalalgia. We very often find headache, too, as the chief symptom in nervous, neurasthenic people—neurasthenic cephalalgia. To this class belong especially people who are overworked physically and mentally—scholars, officials, students before an examination, etc. If we believe that we can make out "rheumatic" influences, such as taking cold, or toxic influences, such as alcohol, nicotine, or chronic lead poisoning, we speak of a rheumatic or a toxic cephalalgia; but, strictly speaking, "rheumatic" headache is probably not a nervous headache at all, since it seems to be in many cases, at least, a slight rheumatic inflammation of the external soft parts of the head. Patients with habitual headache often suffer at the same time from chronic gastric disturbances or habitual constipation, so that the latter disorders, in many cases, stand perhaps in a causal relation (toxic influences?) to the headaches. Finally, it is a very important point that headache is sometimes associated with chronic diseases of neighboring organs, especially of the nose, the naso-pharynx, and the ear. In very many cases, however, we can find no definite cause at all for the affection, so that we have to do with a genuine idiopathic disease. Habitual headache, existing from childhood, is certainly in most cases the expression of a general "nervous constitution," which is due to congenital conditions which of course are still wholly unknown in their details. Habitual headache, therefore, is often an hereditary disease. [One of the frequent causes of persistent headache is eye-strain, due to the persistent unconscious effort of the muscles of accommodation to overcome some error of refraction, such as astigmatism or hypermetropia. The pain is often referred to the frontal region, and it is brought on or made worse by using the eyes for near work. In some cases this eye-strain may be associated with insufficiency of some of the external muscles of the eye. This condition may be associated with normal visual power, so that the patient often maintains that there can be no trouble with the eyes; this is especially true where the refractive error is slight, less than one dioptric. It is therefore essential, in every case of persistent headache, to have a thorough examination of the eyes, in order to determine whether there is astigmatism or hypermetropia. The adjustment of proper glasses, or, in rare cases, tenotomy of some of the external muscles, will often give marked relief, although even in these cases attention must also be paid to the general condition. In many cases the error of refraction is but one out of many causes which give rise to the headache, so that we should never be too confident in our prognosis of cure, and we should also never be content with finding a single cause, but we should search for all possible causes. Referred pains in the head from disease elsewhere have been spoken of on page 806.—K.]

Habitual headache is always a chronic disease. It may last for months or years, or even through the whole life, either being present continually, or, what is more frequent, coming on in separate attacks and lasting several hours or days. These attacks sometimes come without any evident cause, but they may often be referred to definite influences, to mental excitement, physical exertion, errors of diet, etc. The pain is felt either in the forehead or in the occiput, or sometimes over the whole head. It is sometimes limited to a definite and quite sharply defined part of the head. The precise form of the pain is described in different ways, either as boring, or tearing, or as if the head were pressed together from without, or as if it would split open. In many cases the intensity of the pain is not great; there is merely a dizziness and a feeling of "pressure" in the head, but in other cases the pain is very severe. In such cases there is also at times a pronounced hyperæsthesia of the skin of the head, so that it may cause pain even to touch the hair.

The general health is almost always disturbed in headache. The patient can not work, he is often ill-tempered and irritable, and he loses his appetite. We sometimes see more marked gastric symptoms, especially nausea and vomiting (transition forms to migraine, *q. v.*), and sometimes copious perspiration. Severe cases of the disease are of great importance, since by an attack the patient is rendered almost wholly unable to attend to his business. For many women, especially, habitual headache is a factor which dominates their whole life as a restraint and disturbance.

The treatment of headache is a very difficult and usually a thankless task. In the first place, of course, we should try to adapt our treatment to the ætiology of the disease if it is evident. We should therefore never neglect to make a thorough examination of all the organs which are to be considered (nose, ear, stomach, heart, kidneys, [eyes], etc.), and pay especial attention to any existing underlying diseases (syphilis, gout, alcoholism, organic nervous disease, etc.). The general physical constitution also requires the greatest attention. For anæmic patients we prescribe iron, arsenic, a country residence, strengthening diet, etc. We order full-blooded persons, especially if they also suffer from indigestion, to follow a restricted diet, to have gymnastic treatment, or to drink bitter waters, or we send them to such health-resorts as Marienbad or Carlsbad. Nervous headaches in hysterical and neurasthenic patients require, above all, a rational general treatment: mental treatment, a regulation of their mode of life, cold-water cures, electricity, general faradization, galvanism to the head or to the sympathetic, etc. For persons who suffer from nervous headache in consequence of excessive mental strain and excitement, we most urgently advise complete physical and mental rest for a long time. We send them to the country or make a trial of sea-bathing.

The number of symptomatic remedies recommended to relieve headache is very considerable. In most of the tedious cases the patient himself has learned to know his disease perfectly well. Many know that there is no remedy for "their old headaches," and they merely desire to rest, waiting until the pain ceases of itself. Others have become accustomed to employ certain domestic remedies: they put poultices on the head, take a cold or a hot foot-bath, put a mustard plaster to the back of the neck, bathe the forehead with cologne-water, use a menthol pencil, bind a towel tightly about the head, drink strong tea, smell ammonia or "smelling-salts," etc. We sometimes see good results, although frequently we do not, from the internal remedies. These may be used, either during the attack or for a longer period, to prevent the return of the pain. A new remedy often works well, but the benefit is not lasting. There are no special indications for the different remedies, so that we must try to see which one is the most efficacious. Antipyrine is most used of late in doses of seven to twenty grains (gramme 0.5–1.5). Its efficacy in migraine is undoubted, but it sometimes gives distinct relief in other forms of headache. We may also use antifebrine, in doses of three to seven grains (gramme 0.25–0.5), phenacetine, seven to fifteen grains (gramme 0.5–1), and quinine, five to twelve grains (gramme 0.3–0.75). In "rheumatic" headaches which come on after exposure to cold, draughts, etc., we may try sodic salicylate, half a drachm to a drachm (grammes 2–4). It is of practical importance that a mixture of these remedies (for instance, equal parts of antipyrine and phenacetine) is often more efficient than a single remedy by itself. Of other drugs we may mention paullinia serbilis (guarana, which contains caffeine), in seven- to thirty-grain powders (grammes 0.5–2), three to six one-grain ergotine pills (gramme 0.05) a day in hyperæmic headache, potassic bromide half a drachm to a drachm (grammes 2–4), arsenic, [cannabis indica, caffeine], etc.

Electrical treatment (*vide supra*) has given decidedly good results in many cases, although the mental factor perhaps plays the chief part here. We must always begin electrical treatment with great caution, and try first what method is best borne. Cold-water cures, too, are sometimes beneficial, as we have said above, or residence in the country, at the seashore, or in the mountains.

We can sometimes do the patient good service with the remedies mentioned, but in other cases the evil obstinately defies all attempts at cure. Then, however, the patient has still the encouragement left that the disease often ceases at last spontaneously in advanced age, after lasting years and years.

CHAPTER VI

ANOMALIES OF THE SENSE OF SMELL

Anomalies of smell, which depend upon a disease of the olfactory nerve itself, of its terminal apparatus, or of its central termination, are not infrequently observed, but they have no great practical interest. It is well known that only the upper two turbinated bones and the upper part of the septum of the nares, the olfactory region, are supplied by fibers of the olfactory nerve. The odors excite the special olfactory cells which lie between the epithelial cells. The processes of these cells—that is, the olfactory nerves—pass through the openings of the lamina cribrosa to the olfactory bulbs in which they split up. The ganglion cells of the bulb form neurones of the second order for the olfactory tract. Their nerve-fibers pass through the olfactory tract to the brain. The fibers for smell either enter into relation with the optic thalamus or terminate in the cortex of the uncinate gyrus and the cornu Ammonis. A part of the olfactory fibers, however, undergo a decussation in the anterior commissure. The fornix and the corpora albicantia have also relations with the olfactory apparatus, which in man is only a poorly developed organ, while in many animals it has a very decided morphological development.

In testing the sense of smell, we use substances which do not at the same time irritate the sensory fibers of the trigeminus in the nasal cavity. The best substances to use are cologne-water, ethereal oils, such as oil of cloves or oil of bergamot, oil of turpentine, camphor, musk, valerian, asafœtida, etc.

Hyperæsthesia of the sense of smell (hyperosmia) makes itself manifest either by a remarkably fine perception of odors, or by an abnormal sensitiveness to them. The latter symptom is often noticed, especially in the hysterical. Patients have headaches, or attacks of fainting, from slight odors, which healthy persons notice but little. Subjective sensations of smell (hallucinations of smell) are quite frequent among the insane, and sometimes during the aura of an epileptic attack.

A diminution of the power of smell (olfactory anæsthesia, anosmia) is not infrequently seen in the different diseases of the nose—coryza, severe inflammation, etc.; also in affections of the base of the skull, such as tumors, and acute and chronic meningitis, which involve the trunk of the olfactory sympathetically, and also in cerebral disease, tumors, etc. In right hemiplegia and aphasia, for example, co-existing anosmia of the left nasal cavity has sometimes been observed. In far-advanced tabes dorsalis we have sometimes found pronounced anosmia, dependent, perhaps, upon an atrophy of the olfactory nerve. Complete or unilateral anosmia, however, is most frequently observed in the hysterical (hysterical hemianæsthesia, traumatic hysteria, etc.). It is important to state that in every marked enfeeblement of the smell the "taste" for many forms of food suffers, since it is well known that their "aroma," as in roast meats, wines, and the different sorts of cheese, is due chiefly to the co-existing sensations of smell.

The treatment of anomalies of smell almost always coincides with the treatment of the primary disease. In case the disturbance of smell makes special interference desirable, we can try electrization of the nasal mucous membrane, or painting it with very irritating or very odorous solutions.

CHAPTER VII

ANOMALIES OF THE SENSE OF TASTE

Sensations of taste are obtained from two nerves—the glosso-pharyngeal, and the lingual nerve from the third branch of the trigeminus. The glosso-pharyngeal is the nerve of taste for the posterior third of the tongue and the palate, the lingual for the anterior two thirds of the tongue. The gustatory fibers of the lingual, all, or at least a great part of them, pass over to the chorda tympani, and reach with this the trunk of the facial; they do not remain in the facial, however, as many pathological observations have most plainly shown, but they finally come back to the trigeminus, and, as has been believed up to the present time, probably chiefly by the great superficial petrosal nerve and the Vidian nerve to the spheno-palatine ganglion, in this way passing to the second branch of the trigeminus; or, as is rendered probable by more recent investigations, they pass by means of the otic ganglion to the third branch of the trigeminus and to the Gasserian ganglion (*vide* page 802). We know nothing definite as to their further course and their central termination.

Hyperæsthesia of taste is rare, and it has been seen almost exclusively in the hysterical. Paræsthesia of taste is sometimes found in patients with facial paralysis, who complain of an abnormal taste in their mouths. In the insane, too, subjective sensations of taste (hallucinations of taste) may occur. Anæsthesia of the gustatory nerves (gustatory anæsthesia, ageusia) is, however, quite frequent. As follows from what has gone before, this may be seen: (1) In affections of the peripheral terminal organs of the gustatory nerves, as in disease of the mucous membrane of the tongue; (2) in affections of the glosso-pharyngeal, such as compression; (3) in affections of the lingual nerve, and of the trigeminus within the cranial cavity; (4) in affections of the chorda tympani, from diseases of the middle ear; (5) in affections of the facial nerve between the entrance of the chorda tympani and the geniculate ganglion; but we know from experience that any obstacle to conduction in this nerve, above or below the points named, causes no disturbance of the sense of taste. Central disturbances of taste are said to have been observed in affections of the posterior portion of the internal capsule. Disturbances of taste are very common in hysteria and allied conditions (traumatic neuroses, etc.).

The test of the sense of taste must be made separately for all the different varieties of the sensation of taste, since partial paralyses of taste are not infrequent. The test is performed by putting small amounts of the substance to be tasted in solution on the tongue with a glass rod or a brush. The anterior and posterior parts are to be tested separately. A solution of quinine or tincture of nux vomica serves as a test for bitter, a solution of sugar for sweet, vinegar or dilute muriatic acid for sour, and a solution of common salt for salt. We may also use as a test for taste the well-known galvanic taste, which is strongest at the anode, but is also detected at the cathode in even very weak currents, and hence is so often noticed from by-currents in galvanizing the head, neck, etc.

An accurate diagnosis as to the seat and cause of disturbances of taste can be made only by considering the other symptoms which are also present. Direct treatment may best be employed by the aid of electricity.

SECTION II

Diseases of the Motor Nerves

CHAPTER I

GENERAL REMARKS UPON THE DISTURBANCES OF MOTILITY

1. Paralysis

General Classification of Paralyses.—By "paralysis" we mean the loss of voluntary motion in the muscles of the body controlled by the will. We usually distinguish between the complete loss of the power of active motion (paralysis) and the mere weakening of it (paresis). In complete paralysis of any part of the body, or of a single muscle, the slightest voluntary motion can not be produced in it; while in paresis of a diseased part certain movements are still possible, but they are more or less below the normal in strength, extent, and duration.

In every portion of the tract that leads from the motor portions of the cortical gray matter of the brain to the muscles—that is, in every part of the so-called great "cortico-muscular conduction-path" or "pyramidal tract"—disease may lead to paralysis if it takes away from the part affected its power to conduct voluntary motor excitations. Every destruction or inhibition of function of the motor centers in the cerebral cortex, with whose integrity the initiation of voluntary innervation is associated, must also lead to a paralysis in the corresponding muscular region. Finally, diseases of the muscles even may cause paralysis, since the muscles may either have their contractile substance injured, or lose their power to respond by a contraction to any nervous excitation that reaches them ("myopathic paralyses").

If we represent to ourselves briefly the precise course of the chief tract for exciting voluntary movements, so far as it is now known, we must put the beginning of this tract, according to all recent experiments, in the region of the central convolutions of the cerebrum and of the paracentral lobule. Here we find the so-called psycho-motor centers (see the details in the chapter on cerebral localization), from which the motor fibers in the corona radiata converge and pass downward. The latter, after they have united into quite a compact bundle, enter the internal capsule, which they traverse obliquely. As we see in a horizontal section through the cerebral hemispheres (see Fig. 95), the internal capsule consists of two limbs—an anterior, between the lenticular and the caudate nuclei, and a posterior, between the lenticular nucleus and the optic thalamus. The two limbs form an obtuse angle, opening outward, whose top—that is, the junction of the anterior and posterior limbs of the internal capsule—is termed the "knee of the capsule." The motor tract (*Py*) lies in the posterior limb of the

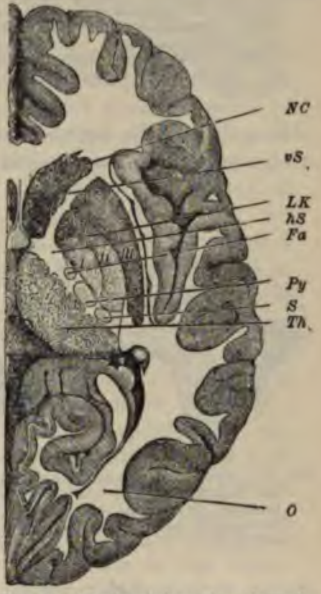

Fig. 95.—Horizontal section through the right cerebral hemisphere. *NC.* Caudate nucleus. *Th.* Optic thalamus. *LK.* Lenticular nucleus (first, second, and third divisions). *vS.* Anterior limb of the internal capsule. *hS.* Posterior limb of the internal capsule. *Fa.* Fibers belonging to the facial nerve. *Py.* Pyramidal tract (motor). *S.* Sensory tract (probably cutaneous nerves and those of special sense). *O.* Occipital lobe.

internal capsule about the posterior end of its middle third. The fibers for the cranial motor nerves (facial, hypoglossus) lie farther forward than the fibers for the extremities. The tract runs downward rather obliquely, so that in the upper part of the internal capsule it lies somewhat farther forward than in the lower. From the internal capsule the pyramidal tract enters the crusta. It lies first in the third quarter, counting from the inside, then farther down in the middle third of the crusta (see Fig. 96). The fibers for the cranial motor nerves here lie nearer the median line than the fibers for the extremities. Farther downward the tract passes into the anterior half of the pons. In the pons the fibers of the pyramidal tract are somewhat separated, but they come together below it into the compact bundle of the pyramids on the anterior surface of the medulla. In this course from the internal capsule to the medulla oblongata all the nerve tracts for the muscles of the head branch off laterally, and, after they have decussated in the median line, they go to the nuclei of their respective nerves (oculo-motor nuclei under the aqueduct of Sylvius, facial nuclei in the pons, hypoglossal nuclei in the medulla, etc.). The motor fibers for the trunk and extremities remain in the pyramids, at the lower end of which the decussation of these fibers (motor decussation of the pyramids, *decussatio pyramidum*), takes place. The fibers of each pyramid pass over, for the most part, into the lateral column of the opposite half of the spinal cord, and here from the distinct bundle of the lateral pyramidal tract (*PyS*, see Figs. 97 and 98). A small part of the pyramids, which sometimes seems to be entirely wanting, remains uncrossed, and passes downward as the so-called anterior pyramidal tract (*PyV*, Fig. 97) in the anterior column of the cord on the same side. From the lateral column, or the anterior column of the cord, the motor fibers pass into the anterior gray columns of the cord, and are here connected with the large motor ganglion-cells of the anterior cornua. We now know that this connection does not take place in the sense of a direct passage of the lateral tract fibers into the cells of the anterior cornua, but that each fiber breaks up into a "nervous terminal arborization," which surrounds the protoplasmic processes of a ganglion-cell of the anterior horn and in this way enters into

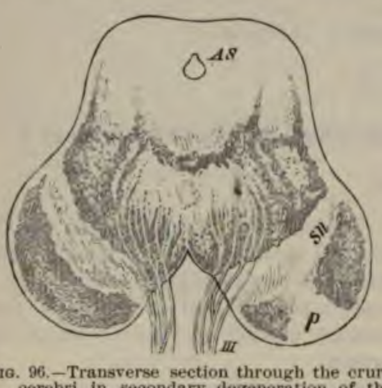

FIG. 96.—Transverse section through the crura cerebri in secondary degeneration of the right pyramidal tract. (From CHARCOT.) *sn.* Substantia nigra. *p.* The degenerated and therefore translucent pyramidal tract. *III.* Oculo-motor nerve. *AS.* Aqueduct of Sylvius.

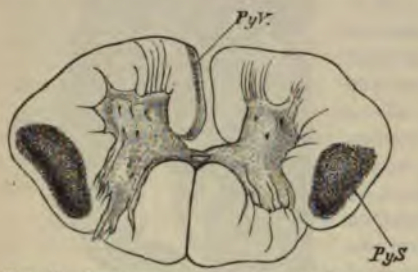

FIG. 97.—Transverse section through the cervical enlargement of the spinal cord. *PyS.* Lateral pyramidal tract. *PyV.* Anterior pyramidal tract (in this case present only on one side).

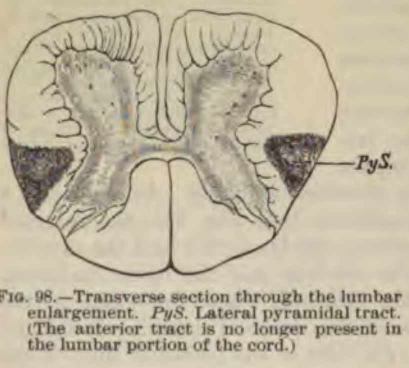

FIG. 98.—Transverse section through the lumbar enlargement. *PyS.* Lateral pyramidal tract. (The anterior tract is no longer present in the lumbar portion of the cord.)

the closest relation with it. From the ganglion-cell of the anterior horn its axis-cylinder process passes off directly as the anterior root-fiber, and the anterior spinal roots are further continued as the peripheral motor nerves. In the muscle, as we know, the nerve-fiber again breaks up into a terminal arborization ("motor end plate"), which finally transmits the irritation coming from the cerebrum to the contractile muscular fiber.

The long motor tract, just described, the cortico-muscular path or the pyramdal tract, whose course has been quite accurately determined in its details by he results of pathological observations (Türck, Charcot) and investigations into the history of its development (Flechsig), is composed of two neurones connected with each other: the first (central) motor neurone extends from the cell of origin n the cerebral cortex to the terminal arborization of the fiber in the motor nuclei (nuclei of the cranial motor nerves, motor ganglion cells in the anterior cornua); he second (peripheral neurone) extends from the above-mentioned ganglion cells o the muscles, and is accordingly formed essentially by the ganglion cell and the peripheral nerve-fiber. It forms, at all events, the chief path for the conduction of voluntary innervation. It is possible that there are other motor paths of conduction besides this tract, but we know nothing definite concerning them.

If we pay attention to the course of the motor tracts described, we shall easily understand certain peculiarities in the distribution of motor paralyses, which are of great importance in diagnosis. Since the motor centers for separate parts of he body, such as the face, the arm, or the leg, are separated from one another in he cerebral cortex, and are distributed over a comparatively large surface, as we hall see more fully later on, it is easily explained why affections of the cortex, if hey are not very extensive, may lead to paralysis of only a single part of the body. We call such an isolated paralysis of one part of the body monoplegia, and hus we speak of a cortical facial or brachial monoplegia. Farther downward in he brain, in the internal capsule and the crus cerebri, however, as we have seen, ll the motor fibers are collected into one bundle, whose diameter takes up comparatively little space. Hence we perceive that any disease of the brain, which s situated in this part of the motor tract, may easily make this tract incapable of conduction throughout its whole extent, or at least throughout the greatest part of it. The result must be, then, a more or less complete paralysis of the facial muscles, the arm, and the leg at the same time—that is, of the entire half of the body—a form of paralysis which is termed hemiplegia or unilateral paralysis. We may note here that, as a result of the passage of the motor fibers to the other half of the cord in the decussation of the pyramids, the paralysis must develop on the side of the body opposite to the focus of disease in the brain. Farther down in the medulla and the cord the fibers coming from both hemispheres, and belonging to the two sides of the body, lie quite near each other. Since many diseases of the cord have a tendency to affect the two halves of the cord at once, or gradually to extend over the whole transverse section of the cord, a simultaneous paralysis of the corresponding portion of the two sides of the body will be apt to arise as a result of this. This form of paralysis we call paraplegia. Diseases in the cervical cord may have as a result a paralysis of all four extremities or of the two arms—cervical, brachial, or superior paraplegia; diseases in the dorsal and lumbar cord a paraplegia of the two legs—inferior paraplegia, often called simply "paraplegia," without further prefix. In diseases of the peripheral nerves we have, of course, again a limitation of the paralysis to the region of the affected nerve. The paralysis may be quite extensive in diseases of a nervous plexus—paralysis of a peripheral plexus; or it may confine itself wholly to the region of a single nerve, or even of a single branch of the nerve—paralysis of a peripheral nerve.

We will have to add many more details to what has just been said, but as a fundamental principle we may now note that hemiplegia is the chief form of cerebral paralysis, while paraplegia is the chief form of spinal paralysis. Monoplegias are usually either cortical cerebral paralyses, or peripheral paralyses.

General Ætiology of Paralysis.—The kind of lesion which leads to paralysis may, in the different cases, be very diverse. From easily understood reasons we can very rarely decide as to the kind of lesion from the intensity and extent of the paralysis, but only from the manifest ætiological factors, from the development and course of the paralysis, from other morbid symptoms that are also present, etc. In general, we may divide the paralyses into two groups, according to the nature of their cause: into paralyses from causes that can be discovered anatomically, and into the so-called functional paralyses, in which no anatomical cause for the paralysis can be found; but since our anatomical, and especially our histological, methods of investigation have become better developed and are more employed, the domain of functional paralyses has become gradually more and more restricted, and a definite anatomical cause has now been found for many paralyses which were once regarded as functional.

All the diseases of the nervous system may be anatomical causes of paralysis, if they lie in a spot where they damage or destroy the paths of motor conduction. Inflammation, degeneration, new growths, hæmorrhages, and severe disturbances of circulation, with their results, especially embolic and thrombotic softening, are found in the brain, the cord, and the peripheral nerves, and under some circumstances give rise to the phenomenon of paralysis. Mechanical lesions of the nervous system also play a great part in the pathogenesis of paralysis, especially injuries, and compression of the brain, the cord, and the peripheral nerves by swellings, new growths, and other diseases in their vicinity.

We also know certain toxic substances which cause direct injury (degeneration) of the motor-nerve territory (chiefly the peripheral motor nerves), and consequent paralysis. Of these toxic paralyses, alcoholic paralysis (*vide infra*) and lead paralysis (*vide infra*) are the most important in their clinical relations; but other poisonous substances, such as copper, arsenic, and certain vegetable alkaloids, may also cause paralysis.

We may group a large number of paralyses together under the term of "paralysis after acute diseases." Since in these cases we always have to do with acute infectious diseases, we may assume, as their most probable cause, certain changes in the nervous system, sometimes in the brain or cord, but much more frequently in the peripheral nerves, which stand in direct relation to the specific infectious material. There seem to be chemical poisons ("ptomaïnes") which form in the body in infectious disease, and cause degeneration of certain nerve-fibers, in the same way, for example, as lead. We most frequently see paralysis appear after diphtheria, diphtheritic paralysis (*vide infra*), or more rarely after typhoid fever, small-pox, dysentery, the acute exanthemata, etc. The paralyses which may arise in certain chronic infectious diseases, especially in syphilis and tuberculosis, usually have another origin. Here we usually have to do with the specific morbid product itself (gumma, tubercle), which may invade various parts of the nervous system, but, on the other hand, we must under some circumstances consider the action of toxines even in paralyses in syphilis and tuberculosis. Finally, certain paralyses which occur in certain general constitutional diseases, such as diabetes and gout, are also due probably to toxic influences.

Those paralyses which come on most manifestly because of exposure to cold are termed paralyses from exposure to cold, or "refrigeratory," or frequently "rheumatic" paralyses. Although many spinal diseases, such as myelitis, may perhaps be referred to exposure to wet or cold, we usually reckon among rheumatic paralyses only certain peripheral paralyses, such as that in the domain of the facial nerve. The functional disturbance of the nerves in these cases probably depends upon mild inflammatory changes in them produced by cold, and is accordingly of an anatomical and not merely of a functional nature.

There are, however, quite a comprehensive group of paralyses which we must to-day still term functional paralyses. To this class belong the hysterical paralyses, the paralyses from psychical causes, such as paralysis from fright, the "paralyses from imagination," etc. We shall learn to recognize these more fully in the chapter on hysteria.

In conclusion, we must bear in mind the so-called "reflex paralyses," whose ætiology is not yet fully explained—that is, paralyses which may come on in the course of diseases of certain internal organs, especially of the intestine, and of the urinary and sexual organs, as a result of centripetal irritations acting reflexly. An attempt has been made to explain their origin, from analogy with well-known physiological experiments, by the idea that a "reflex inhibition" is excited in certain motor regions by a sensory irritation arising in the diseased organs, a theory which has not yet been fully confirmed. Leyden's hypothesis is scarcely more probable, and it is by no means proved. According to it the paralyses of this class are explained by an ascending neuritis, arising from the organs originally affected (see the chapter on neuritis). Generally speaking, the whole conception of "reflex paralyses" is still far from clear, and we will do well to be extremely guarded in their diagnosis. Many cases which were formerly regarded as reflex paralyses are to be classed as hysteria, especially as traumatic hysteria (*q. v.*). Lépine has also regarded the paralysis of the arm of the affected side, seen in some cases of empyema, especially after operative interference, as a "reflex paralysis," an explanation which may be proper in some cases, but in regard to which we should be the more cautious, as metastatic abscesses of the brain are not very infrequently found in empyema (see the chapters on purulent meningitis and cerebral abscess).

General Symptomatology of Paralyses.—We can recognize the existence of a paralysis, independently of the patient's statements as to the impossibility of performing certain motions and functions, only by a careful and thorough physical examination of the power of voluntary motion. This examination in patients with nervous disease must extend to all parts of the body, and demands an accurate knowledge of all the movements that can normally be executed by the different joints, and of the muscles and nerves requisite to produce them (see the tables at the end of this chapter).

In each individual case of paralysis some other symptoms must be considered besides immobility—first the condition of the paralyzed muscles, and then certain accompanying symptoms that are often present with the paralysis.

In regard to the first point, the trophic condition of the paralyzed muscle is of the greatest diagnostic and practical importance. In comparing a large number of paralyses, a very evident difference in this respect strikes us at once. We see on the one hand paralyses where the paralyzed muscles retain their normal volume for a comparatively long time and only gradually waste as a consequence of disease and the lack of the normal influences of innervation, although this atrophy never goes beyond a certain degree. On the other hand, however, we also see cases in which there is a considerable atrophy in the paralyzed muscles in a few weeks or months, finally progressing to an almost complete disappearance of the muscles. This distinction is so comprehensive that all the last-named paralyses have been classed together under the name of "atrophic paralyses."

If we once more represent to ourselves the whole course of the motor tracts, from the cerebral cortex to the voluntary muscles, we shall remember that these tracts are composed of two connected yet independent neurones. The peripheral neurone directly innervating the muscle has its ganglion-cell in the anterior cornua of the spinal cord. Clinical and anatomical experience teaches us that, in all those paralyses where the cause—that is, the break in conduction of the motor fibers—lies in the first (central) neurone from the cortex up to, but excluding the cells in the anterior cornua, there is, as a rule, only a slow and relatively slight amount of atrophy in the paralyzed muscles, while in those paralyses where the cause is situated in the second (peripheral) neurone—that is, in these ganglion-cells themselves, or in the motor nerves peripheral to them—a pronounced muscular atrophy rapidly develops. The only possible interpretation of this fact is, that the large motor ganglion-cells of the anterior cornua have, as we express it, a trophic influence on the muscles innervated by them. As we have seen above, the peripheral motor-nerve fiber is nothing but the direct, long cellular process of a motor ganglion-cell in the anterior horn. It is therefore easy to understand that this process atrophies when it is separated from the cell-body and its cell nucleus, or when the cell itself is destroyed. If, however, the motor nerve degenerates, the muscle belonging to it is entirely separated from all influences of innervation; neither voluntary nor reflex nerve irritations can act upon it any longer. This lack of innervation apparently causes such a change in the trophic conditions of the paralyzed muscle that its fibers disappear one after the other, until finally, in long-continued paralyses, only a slight trace of muscular tissue is left. We can therefore consider the peripheral motor neurone and the muscular fiber as, in a certain degree, a trophic unit.

It has been generally believed that the anatomico-histological processes in the rapidly atrophying muscles were also different in diseases of the peripheral motor neurones from the processes of slow and slighter atrophy in paralyses from disease of the central motor neurones. In the first case we were dealing with a so-called "degenerative" atrophy of the fibers—that is, with an atrophy associated with granular and fatty destruction of the fibers—and in the second case only with a "simple" atrophy. Such a histological difference, however, can not be strictly maintained. We are always dealing, essentially, with a diminution in the size of the fibers, where, however, the transverse striation of the muscle remains plainly visible. We also find a great increase in the nuclei of the muscle, and, finally, we often see an increase of the interstitial connective and fatty tissue.

We observe a further distinction in the condition of the paralyzed muscles if we perform passive motion in the paralyzed parts. There are paralyses where we can perform passive motion of the paralyzed parts at any joint with perfect ease and freedom, and without perceiving the slightest resistance. We term such paralyses "flaccid paralyses," but there are also paralyses where passive motion meets with considerable muscular resistance, so that it can be performed only with a certain greater or less amount of exertion, or not at all, or only within certain limits: spastic paralyses. This distinction is dependent upon the state of muscular tonus in the paralyzed muscles. If this tonus be increased (hypertonia of the muscles) and the muscles be found in a state of marked and permanent contraction, the paralyzed parts show a peculiar stiffness, or even an almost complete spastic rigidity. If the muscular tonus be diminished or wholly lost (hypotonia of the muscles), the paralyzed limbs are flaccid and sometimes even "flapping." Since the muscular tonus is in all probability of reflex origin, it is not remarkable that spastic paralyses are usually associated with an increase, and flaccid paralyses often with a loss of the reflexes, especially the tendon reflexes (*vide infra*). States of marked permanent shortening of the muscles, whereby certain parts are fixed in an abnormal position, are called muscular contractures. The cause of their development will be spoken of later.

Finally, in every case of paralysis, we must consider the other nervous symptoms which accompany it, since these may also be of great importance in judging of the cause of the paralysis. We must first of all investigate the condition of he reflexes (*vide infra*) in the paralyzed parts, already mentioned, from which nany conclusions can be drawn as to the seat of the lesion which causes the aralysis. We must also test the state of the sensibility, both in the skin and in he muscles themselves. Certain attendant trophic and vaso-motor symptoms re also to be regarded. The skin over the paralyzed parts sometimes appears yanotic, or marble-like; it feels cool, is œdematous, and sometimes is peculiarly lry, hard, and scaly. The examination of the electrical excitability of the aralyzed nerves and muscles is of especial importance. The phenomena ocurring in these conditions will be particularly described below.

2. SYMPTOMS OF MOTOR IRRITATION

As we have termed the symptoms of motor deficiency "paralysis," we group he symptoms of motor irritation in general together under the name of "spasm." Ve mean by this all the morbid movements occurring in the muscles involuntaily and even against the will. Although we may find spasm in the smooth musles, which generally are not controlled by the will, as in spasm of the bronchial nuscles, spasm of the vessels, etc., we will concern ourselves here only with the pasmodic movements in the voluntary muscles. We must look for the cause of hese in abnormal irritation exerted in some way on the motor tracts, but we now very little of the precise nature and character of this irritation in most ases. The abnormal irritation often acts directly on the motor nervous region, specially in the frequent spasms in affections in the neighborhood of the cortical notor centers; but the motor irritations often seem to be excited secondarily hrough some reflex channel—reflex spasms.

For a long time two kinds of spasm have been distinguished symptomatically. Ve term those spasms clonic where the abnormal muscular contraction lasts nly a short time, and then is interrupted by a short period of relaxation, to come n again afresh. The affected parts of the body are thus put in a constant conulsive motion. In distinction from this we term those abnormal muscular conractions tonic spasms where the muscle remains spasmodically contracted for a nger time—minutes, hours, or days. The affected part of the body is thus kept notionless in some abnormal position. Both forms of spasm, however, show nany transitions and combinations, so that we must often speak of "tonic-clonic" pasms.

A more careful examination of the symptoms of motor irritation, however, ives a still greater number of different forms. We will here group together the nost important varieties of morbid involuntary movements.

1. *Epileptiform convulsions* are severe, and usually clonic spasms, at times onic-clonic, either involving the whole body, and then always associated with disurbances of consciousness, or limited to one half or one portion of the body. By hem the whole body or the affected part is put into violent motion, usually hrashing and shaking movements. The pure epileptic spasm in epilepsy is the ype of this form of spasm, but precisely analogous spasms, "epileptiform" pasms, are seen in organic diseases of the brain, in uræmia, etc.

2. *Persistent rhythmical contractions* in single groups of muscles are someimes seen in certain cerebral diseases, such as apoplexy and sclerosis, and also, s we have observed, after acute myelitis. In these the part of the body affected s put in motion by continuous, separate, weaker or stronger thrusts, which follow ne another in a regular rhythm. Rhythmical contractions are also seen as preursors or at the end of epileptiform spasms.

3. *Trembling motions*, or *tremor*, as we say in ordinary parlance, are moderate notions, rapidly following one another, with a not very marked excursion. If the remor is more pronounced, we term it "shaking." Tremor is an important sympom, almost pathognomonic in many nervous diseases, such as paralysis agitans,

but we know almost nothing in regard to the more intimate manner of its origin. We often see pronounced tremor, especially in the hands, in exophthalmic goître. We know that tremor is often present in old people—senile tremor; and in alcoholic subjects—alcoholic tremor. Tremor sometimes appears in muscles at rest, that is, not innervated by the will, and sometimes only in those which are moved voluntarily. This latter form of tremor, which is seen most frequently in multiple sclerosis (*vide infra*), is termed "intention tremor." Very marked intention tremor, which is increased by any mental excitement, is also seen as a symptom of chronic mercurial poisoning (mercurial tremor), particularly in mirror-makers, etc.

We may mention here the so-called essential tremor, that is, a condition where the tremor, which is most marked in the hands, is the only morbid symptom and can be referred to no known cause. On energetic voluntary innervation of the muscles the tremor, as a rule, ceases. This form of tremor is sometimes seen in comparatively young people, and even in children. A distinct hereditary predisposition is often present, so that several "tremblers" are found in the same family (hereditary tremor).

We very often find trembling in "nervous" persons, which is at once increased by any emotion (nervous, neurasthenic tremor). Marked tremor is also frequent in hysteria, especially in traumatic hysteria.

4. *Single contractions*, either sudden twitchings, or in the form of a slow contraction of the muscle, are seen with especial frequency in diseases of the cord. They are either single, or frequent and persistent. Their mode of origin is not always plain. They may depend on direct motor irritation or they may have a reflex origin. In the latter case each twitching is sometimes associated with a sudden feeling of pain (symptoms of irritation of the posterior roots?).

5. *Fascicular and fibrillary muscular contractions* are little contractions in the separate muscular bundles, which may be seen on a close examination of the muscles, but which do not have any special influence on motion. If the fibrillary contractions in a muscle are very pronounced, there may develop an actual "wave" in the muscular substance. We see this symptom especially in atrophied muscles, particularly in spinal progressive muscular atrophy (*vide infra*) and other spinal amyotrophies, but there also seem to be independent forms of diseases of the muscles, in which we may see for a long time a persistent and extensive muscular wave, associated with pain, profuse sweating, etc.—the so-called *myokymia* of F. Schultze.

6. *Choreic movements* are either slight contractions or quite complicated and extensive, but brief and involuntary movements, which usually appear in the face, in one limb, or sometimes over the whole body, without regard to rule. In severe cases they are almost continuous, but in milder cases they are interrupted by shorter or longer pauses. They form the chief symptom of chorea proper, but they are also present in other cerebral affections, such as symptomatic chorea, posthemiplegic chorea, etc.

7. *Movements of athetosis* is the name we give to peculiar involuntary and usually quite slow movements, which are seen especially in the arms and hands, but also in the head, trunk, etc. The fingers make slow but often very extensive movements, are extended, spread apart, flexed, and moved over and around one another in the most remarkable way. This form of motor irritation occurs as a special disease, "*athetosis*," or as a symptom in certain central nervous diseases, especially the cerebral paralysis of children (*vide infra*).

8. *Constant* or *co-ordinated spasms* are symptoms of motor irritation in which the patient performs complicated movements by compulsion—forced movements. Among these are classed the compulsory going forward or moving in a circle, the turning about the axis of the body (forced attitudes), and also certain peculiar

complicated forms of spasm, such as spasms of jumping, laughing, screaming, with all sorts of spasms of the respiratory, pharyngeal, and laryngeal muscles, associated with hiccoughing and belching sounds, etc. They are seen most frequently in severe cases of hysteria, but epilepsy may also occur exceptionally in the form of co-ordinated spasms. The forced movements and attitudes mentioned above are seen chiefly in affections of the cerebellum and the cerebellar peduncles.

9. *Tonic spasm*, as has been said, is the name for all morbid muscular contractions that continue for a long time. Tonic spasm in the muscles of mastication, the masseters, is termed trismus. Tonic spasm in the muscles of the back and neck, by which the whole body is drawn backward, and the vertebral column is bent into an arch with the convexity in front, is called opisthotonos. Tonic rigidity of the whole body is termed tetanus. Tonic spasms are seen not infrequently as an idiopathic condition, but also in tetanus, tetany, hysteria, etc.

10. *Cataleptic rigidity* is the name of that tonic condition of the muscles in which the limbs are deprived of the influence of the will, but are held in position by the muscles in any position given to them passively. It is seen chiefly in certain cases of hysteria, but cataleptic states are also present in other cerebral diseases, especially if associated with general mental stupor, such as tumors, meningitis, etc. (see the chapter on catalepsy).

11. *Associated movements* are abnormal movements which appear, while making voluntary movements, in muscles which have no connection with the movement willed. Thus in hemiplegia associated movements sometimes take place in the arm when the patient wills to move the leg alone. In spinal cases the movement of one leg is sometimes accompanied also by an unintentional movement of the other leg. Associated movements are commonest in the muscles of the same limb. Thus we often see in hemiplegia or in spastic spinal paralysis that patients can not flex the leg up toward the body without at the same time producing a marked dorsal extension of the foot as an associated movement. In old peripheral facial paralysis (*q. v.*) we very often see associated movements in the facial muscles.

Besides the conditions of motor irritation, other attendant nervous symptoms often occur at the same time. Symptoms of motor paralysis and irritation are very often combined with each other, since the different forms of spasm may appear not only in groups of muscles whose motion is otherwise normal, but also in paretic or paralyzed muscles. In general convulsions the state of the consciousness deserves special attention. Genuine general epileptic attacks are associated with complete loss of consciousness, hysterical attacks are often associated with clouding of consciousness, but in most of the other forms of spasm the consciousness is unaffected. Finally, it is worthy of mention that tonic spasms especially are attended by a feeling of decided pain, which is probably due to an irritation of the intra-muscular sensory nerves. Such painful tonic muscular contractions are termed cramps. Among them are the well-known painful spasms in the calves, etc.

3. Ataxia

In executing all normal complicated movements we need the simultaneous action of several muscles. Consider the numerous muscles which must be put in activity in walking, in grasping, and in all the manifold employments of the hands. Hence, in order to perform such movements correctly, it is not only necessary that all the muscles concerned should be innervated by the will—that is, that they be not paralyzed—but that we should also be able so to modify the innervation of each individual muscle that its contraction corresponds precisely to the special part of the work belonging to it. A prescribed voluntary motion can take place only when, first, all the muscles requisite for it come into no less but also no greater action; second, when each individual muscle contracts only so far and so much as its special task requires; and, third, when the conditions in the time of innervation take their normal course—that is, when all the muscles involved contract at the same time or in the proper order one after another. A movement which is executed in such a prescribed manner we call a co-ordinated movement, and the process of modifying properly the innervation of the different muscles necessary for a complicated movement we call the co-ordination of motion. We must especially bear in mind that the simultaneous action of several muscles is so far necessary, even for what seem to be the simplest movements, that muscles antagonistic to those moved must also come into activity. Only by the aid of the ever-ready antagonistic muscles can we grade our movements as finely, or check or hasten them as rapidly, as is demanded for the execution of almost all complicated movements.

Nervous pathology is rich in facts which can make the idea and the necessity of the co-ordination of motion clear to us. We often see disturbances of motility which make the patient incapable of any fine motor acts, and yet which do not depend at all upon any motor weakness or paralysis, but only upon a disturbance in the co-ordination of motion. Such a disturbance we call ataxia, and we speak of an ataxia of the arms, of the legs, etc., when the parts named can perform all the motions and often even retain their full strength, but these movements show, usually at once, a striking, disordered, uncertain " ataxic " character.

Many theories have been advanced as to the precise cause of ataxia. In general, however, there can be hardly a doubt that ataxia depends upon a disturbance of centripetal influences. We must suppose that our movements are constantly kept in order and control (co-ordinated) by centripetal excitations coming from the skin, muscles, and joints, although they are not all by any means consciously perceived. By such excitations the child learns gradually to change his original uncontrolled and unregulated movements into orderly movements. In the adult most movements have already been so long practiced as to be sure and steady. The learning of a new movement, a new dexterity, however, takes place in precisely the same way. Even a long-practiced movement (walking, grasping) needs the constant control and regulation exerted by centripetal excitations. If these controlling influences are lacking by reason of any disease of their corresponding centripetal tracts, ataxia is the necessary result. Ataxia may occur both in peripheral nervous diseases and also in diseases of the spinal cord, cerebellum, and cerebrum. It is therefore evident that the special circumstances which give rise to ataxia may be very diverse. We will return to this point in the description of the individual diseases which lead to ataxia. (See especially the chapters on tabes dorsalis, multiple neuritis, and affections of the cerebellum.)

4. General Remarks upon testing the Reflexes and upon the Condition of them

In testing the reflexes, which should never be omitted in any case of nervous disease on account of its frequent great diagnostic importance, we must distinguish the two chief groups of reflexes from each other: the cutaneous reflexes, and the " tendon reflexes."

Cutaneous Reflexes.—We term the muscular contractions, excited reflexly by irritation of the sensory centripetal cutaneous nerves, cutaneous reflexes. These are usually present only to a slight degree in the upper extremities; but we can sometimes excite reflexes even here by pricking or pinching the skin, especially the skin of the fingers. The very marked reflex in many people caused by tickling the axilla is well known. The test of the cutaneous reflexes in the lower extremities is much more important. The soles of the feet are the most sensitive parts for exciting a reflex, whose arc lies in the first and second sacral segments of the spinal cord. Simply tickling the soles with the finger is a sufficient irritation (the tickling reflex), and so is the prick of a pin (the prick reflex), or stroking the skin hard with a blunt object, usually the handle of a percussion-hammer (the stroking reflex). Thermal irritants are also very suitable for exciting a reflex, especially bits of ice held to the skin (cold reflex). It is often advisable to try all these methods, since with diminished reflex irritability a reflex contraction in the leg can often be excited by some one of them, alone. We should also examine the reflex irritability of the rest of the skin, as well as the soles of the feet, by a pin-prick, by pinching a fold of the skin, etc. We should especially remember that in nervous diseases there is often a delay in the reflex, so that the reflex contraction appears only when the irritation has lasted for a certain time. Thus in many diseases of the cord, as we have often seen, the reflex follows only after we have kept a needle sticking into the skin for some time or have pinched a fold of skin continuously for several (ten or fifteen) seconds, a delay which has a connection with the fact of the " time summation of reflex irritation " known from physiology. The summation of reflex irritation not infrequently coincides with the summation of irritation from the sensation of pain (*vide supra*, page 793), so that, after the irritation has acted for a certain length of time, the sensation of pain and the reflex contraction occur at the same time. In the stroking reflex (a rapid, vigorous stroking of the sole of the foot or of other parts of the skin with the handle of a percussion hammer) we have a local summation of reflex irritation, since many different parts of the skin are rapidly irritated one after another. In the tickling reflex the local and the time summation of reflex irritation are united. All parts of the skin do not show the same " reflex sensibility." The reflexes are usually set free more readily from the sole of the foot than from the skin of the leg or thigh. The reflex contraction in such cases consists usually of a dorsal extension of the foot and a coincident flexion of the knee and hip. In the toes, stroking the sole usually causes in healthy persons a flexor reflex, but in the paralyzed there is often a dorsal extension of the great toe. After irritating the skin of the thigh we sometimes see reflex contractions in the extensors of the lower leg. The stronger and more persistent the irritation, the more energetic and widespread is the reflex contraction. Where the reflex irritability is actively increased, sticking a needle into the sole will often produce a vigorous flexor contraction in the irritated leg, and also a weaker contraction at the same time in the other leg.

[The researches of Babinski, confirmed by many other observers, have shown that dorsal extension of the great toe, following stroking of the sole, is in adults a valuable sign as indicating disturbance of the central motor neurones, the pyramidal tract. It is rarely, if ever, obtained in health, except in young children, but it is found in the majority of cases of old hemiplegia, spastic paralysis, etc., where there is degeneration of the pyramidal tract, and it may occur before there is any notable increase in the tendon reflexes. The first response on irritating the sole of the foot in health is usually a plantar flexion of the great or small toes; in some instances there is only a contraction of the tensor vaginæ femoris muscle, which may readily escape observation. In other cases, still, the irritation at once causes a sudden withdrawal of the leg. We rarely, if ever, fail to elicit some form of response, which is equal on the two sides, in health.—K.]

We must also mention two special forms of cutaneous reflex which are often examined: the abdominal reflex and the cremaster reflex. The abdominal reflex is best excited by stroking the abdominal wall rapidly and with some force with the handle of a percussion hammer. If we stroke from without inward on the linea alba, we can distinguish, according to the level of the irritation, an upper, middle, and lower abdominal reflex. On stroking from above downward, these contractions unite. In health the abdominal reflex is only very rarely absent, chiefly in persons with a very lax abdominal wall and in the corpulent. In cerebral hemiplegia the absence of the reflex on the paralyzed side is an important sign. In spinal diseases the reflex may be absent on one or both sides, which should lead us to bear in mind that the centripetal portion of the reflex arc of the lower and middle abdominal reflex enters the spinal cord through the posterior roots of the tenth to twelfth intercostal nerves, and of the upper abdominal [epigastric] reflex through the posterior roots of the ninth intercostal nerve (Dinkler). The cremaster reflex is a reflex ascent of the testicle when the inner side of the thigh is forcibly stroked or when sudden deep pressure is exerted by the hand a hand's breadth above the internal condyle. This reflex is almost always present in healthy boys and men, although in varying strength. We must take care not to confuse the cremaster reflex with the contraction of the dartos. The clinical significance of the cremaster reflex is similar to that of the abdominal reflex. The reflex arc of the cremaster reflex is at the level of the first and second lumbar nerves. Other cutaneous reflexes—the gluteal, the mammillary, etc.—have at present no special practical significance.

Tendon Reflexes.—Of almost greater practical importance than the investigation of the cutaneous reflexes is the test of the phenomena classed under the name of the "tendon reflexes," and first carefully investigated and described by Erb and Westphal in the year 1875. We understand by these those muscular contractions which arise from the brief mechanical irritation of the tendons and analogous parts, such as the periosteum and fasciæ. Westphal originally held that these contractions were the result of direct mechanical irritation, but later accurate clinical and experimental work has shown that the "tendon reflexes," as Erb had maintained from the beginning, are true reflex phenomena, starting from the mechanical irritation of the sensory nerves in the tendons, periosteum, etc. The tendon reflex of most practical importance is the patellar reflex, "knee phenomenon" (Westphal) [or "knee-jerk" (Mitchell)]—that is, a contraction occurring in the quadriceps after mechanical irritation of the ligamentum patellæ by a quick blow with a percussion hammer. In order to evoke this reflex it is especially necessary for the person examined to avoid all active muscular tension in the leg, especially in the extensor. If we examine patients who are not in bed, we can test the patellar reflex by making the patient while sitting cross the leg to be tested over the other, and by striking the patellar tendon as the leg hangs loose; but it seems to us more convenient to direct the patient to extend the leg until it forms an obtuse angle with the thigh with the opening of the angle below. If the sole be set fully on the floor the extensor cruris is made tense in this position, and we can very easily and plainly excite the contraction of the quadriceps by striking the patellar tendon. When the patient is in bed it is best to raise the leg to be tested into a semi-flexed position by putting the left hand under the hollow of the knee; the foot remains lying on the bed, and the patient should avoid any active muscular tension. If the patellar reflex be weak and hard to detect, we should try the proceeding advised by Jendrássik, which consists in testing the reflex while the patient grips the two hands together, and tries with all his strength to pull them apart. By this or any other vigorous tension of the muscles of the upper extremities the muscles of the legs are probably greatly relaxed, while any voluntary innervation is avoided, and thus is explained the increase of the reflex, which is often pronounced. If by the help of this "Jendrássik's test" no reflex is to be elicited, we may regard it as absent, a condition which is almost always of pathological significance, since complete absence of the patellar reflex practically never occurs in health. The reflex arc in the cord for the patellar reflex is found in the second lumbar segment.

The second important tendon reflex to be provoked in the lower extremity is e Achilles tendon reflex. If we give to the foot of the person examined (who ould lie on his side in bed) a passive position of slight dorsal extension, so that e tendo Achillis is a little tense, and then strike the tendon a quick blow, a arked contraction of the gastrocnemius follows. Precisely the same reflex is sually to be elicited by striking the plantar fascia. The Achilles tendon reflex is ot quite so constant in healthy persons as the patellar reflex, but it is usually esent and well marked. Its reflex arc in the cord lies at the level of the third the fifth sacral segments. Where the tendon reflexes are abnormally increased, owever, the Achilles tendon reflex is very vigorous, and then we can very often oduce it in the following especially characteristic manner. If we make a sud- en, short, vigorous, passive, dorsal extension of the foot, the tendo Achillis is ddenly made tense, and thus is irritated mechanically. As a result of this, ere is a reflex plantar flexion of the foot. If now, by persistent passive dorsal tension of the foot, the tendo Achillis is again made tense, there follow by rns new plantar and dorsal movements of the foot, so that the foot is thus t into a vigorous tremor. This symptom, which can only exceptionally be pro- ked in healthy persons, is termed ankle clonus (foot clonus), or "foot phenom- on" (Westphal). Where there is a very great increase of the tendon reflexes e tremor is sometimes not confined to the foot, but the whole leg falls into a gorous clonus, a symptom which was once given the unsuitable name of spinal ilepsy, since it apparently came on spontaneously as a result of some tension of e tendons. We can also obtain the patellar reflex in the form of a clonus if we ll the patella firmly down with the finger and force it downward by a sudden ow on the finger.

The two symptoms described—the patellar reflex, and the Achilles tendon re- x or foot phenomenon—are practically the most important, and are the most ten tested, but they are by no means the only reflexes in the lower extremity. esides the reflexes from the special tendons, we also frequently obtain muscular ntractions by striking the periosteum and the fasciæ, which have been termed he periosteal and fascia reflexes. Thus a contraction in the quadriceps often ollows a blow on the anterior surface of the tibia when the reflex excitability is ncreased. We also see contractions frequently in the adductors of the thigh on triking the internal condyle of the femur, contractions in the muscles of the osterior aspect of the thigh in striking the calves, etc.

Under normal conditions the tendon reflexes in the upper extremities are metimes quite weak or entirely absent, but where the irritability is abnormally reased we see even here the most various and vigorous reflexes. The most portant and most constant are the periosteal reflexes, from striking on the er end of the radius and ulna. As a rule, in our observations, we have usually ained a contraction in the supinator longus and biceps from a blow on the d of the radius, and a weaker contraction in the same muscles from the head he ulna, and also, very frequently, pronation of the forearm and flexion of the t and fingers. The deltoid also contracts not infrequently from a blow on the r end of the ulna. Direct tendon reflexes in the biceps and triceps on strik- their own tendons are also almost always to be obtained. [The triceps reflex as the most constant of all the reflexes of the upper extremity.—K.] We may not infrequently obtain a contraction in the biceps by a blow on the clavicle. rsistent clonus in the hand upon passive volar flexion sometimes occurs, but it are.

A more detailed account of the behavior of the cutaneous and tendon reflexes he individual diseases of the nervous system will be given later in the special pters, where we shall also learn the present theories as to the course of the ex tracts and the influence of other nerve tracts upon them.

Mechanical Muscular Irritability and Paradoxical Contraction.—"*Direct mechanical irritability of the muscles*" is shown by the occurrence of energetic brief contractions of single muscular bundles or of the whole muscle from a direct blow on the belly of the muscle with a percussion hammer. In such cases, however, we are dealing, in my opinion, not with a direct muscular irritation, but with a mechanical irritation of the nerves which enter the muscle, since the muscular contractions are always strongest when we strike the corresponding point of entrance of the nerves, precisely the same as faradic irritation of the muscle. The ensuing muscular contraction, however, may sometimes be a reflex, evoked by mechanical irritation of the fascia stretched over the muscle. These contractions just described are to be distinguished from the so-called idiomuscular contractions. We see these most plainly if we give a vigorous blow with the ulnar side of the hand or with a percussion hammer to the belly of a muscle, such as the biceps. A small transverse, circumscribed muscular swelling forms at the point struck, and gradually disappears again. The test for mechanical muscular irritability has not yet attained any special practical importance.

"*Paradoxical contraction*" is the name which Westphal has given to a symptom seen especially in the tibialis anticus, and rarely also in the flexors of the leg and forearm. It is when the foot, after being put in passive dorsal extension, remains in this position even after the expiration of a considerable time (several minutes), and a marked prominence of the tendon of the tibialis anticus is usually visible. We can not at the present time give a definite explanation of this phenomenon, which so far has been observed in various spinal and cerebral diseases, multiple sclerosis, paralysis agitans, etc.

5. GENERAL REMARKS UPON THE CHANGES OF ELECTRICAL EXCITABILITY IN THE MOTOR NERVES AND MUSCLES *

Electricity, since the investigations of Duchenne, Remak, Benedikt, Moritz Meyer, von Ziemssen, Brenner, Erb, and others, has become not only one of the most prominent therapeutic aids in the treatment of nervous diseases, but it also plays an extremely important part in the examination of nervous patients, since the test of the electrical excitability of diseased nerves and muscles gives us a large amount of valuable information in regard to diagnosis and prognosis.

Every complete electrical examination must be made with both currents—the faradic or induction current (usually the secondary current), and the galvanic or constant current. One "indifferent" pole is usually put on the sternum or the back of the neck, and the other "testing" pole on the nerve or muscle to be tested. For the indifferent pole we use a large electrode (about twelve centimetres long), and as a testing pole a smaller electrode, the best being Erb's "normal electrode," with a surface of ten square centimetres. The excitement of the muscle from the nerve is called indirect; the excitement from placing the electrode on the muscle itself (where, of course, the excitement of the intramuscular nerves can not be excluded) is called direct. Those points on the human body where the different nerves and muscles are most easily accessible to the electrical excitement are to be found in Figs. 99 to 104, taken from Erb's handbook.

In faradic examination the normal condition is that we can provoke marked muscular contractions both from the nerves and from direct excitement of the muscles at the points generally accessible to excitement. As von Ziemssen has found, the points at which the muscle can most easily be made to contract by

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* In regard to all the details of electrical diagnosis and electro-therapeutics we would refer to Erb's "Handbuch der Elektrotherapie." Leipsic, Vogel, 1886. [Translated by De Watteville. New York: Wm. Wood & Co., 1887.] E. Remak's "Grundriss der Elektrodiagnostik und Elektrotherapie." Vienna, 1895, etc.

"direct" excitement correspond exactly to the points where the motor nerves enter the muscle. The direct faradic muscular excitation is therefore always a nerve excitation. In order to determine the excitability quantitatively we measure the distance between the two coils of the induction apparatus (coil distance, *Rollenabstand*) at which the first minimal contraction of the muscle occurs. On increasing the current, the minimal contraction gradually passes into a vigorous tetanic contraction of the muscles.

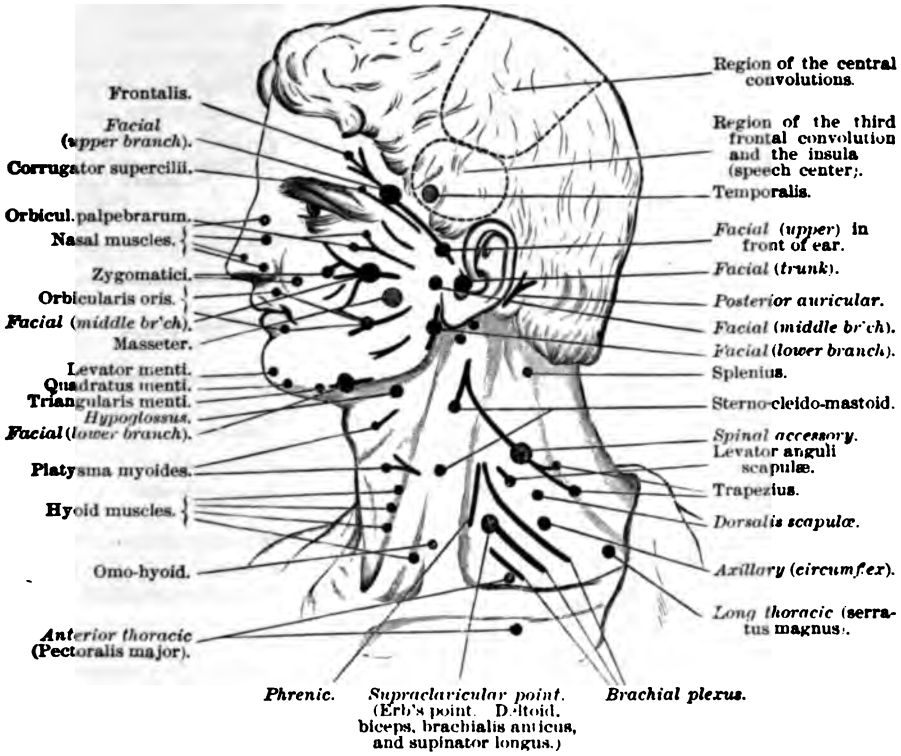

FIG. 99.

Galvanic examination is performed by the aid of a "current reverser," by which the current at any desired strength can be rapidly opened and closed and the testing pole can be made either the negative pole (the cathode or zinc pole) or the positive pole (the anode or copper or carbon pole).

The nerves and muscles are excited not by the continuous flow of the electrical current, but only by its variations and the sudden opening and closing of the current.

By the help of this "polar method of investigation" (Brenner) we can easily determine the law of contraction, which holds equally for the normal motor nerves and for the muscles.

With a very weak current no noticeable excitement takes place. If we gradually increase the strength of the current, the first weak contraction of the muscle occurs at the closure of the cathode—that is, when the current is closed so that the cathode is made the testing pole. On opening the cathode, or on closing or opening the anode, nothing follows. If we increase the strength of the current still more, the cathodic closure contractions become stronger, and the anodic closure and anodic opening contractions appear, now the one being earlier and stronger, and now the other. Opening of the cathode has still no effect. Only with a very strong current, in which the cathodic closure contractions have already become tetanic—that is, they still persist after the closure of the current—can we provoke weak cathodic opening contractions. Expressed in the abbreviations now in general use in electrical diagnosis, the law of contraction for normal muscles and nerves in man is as follows:*

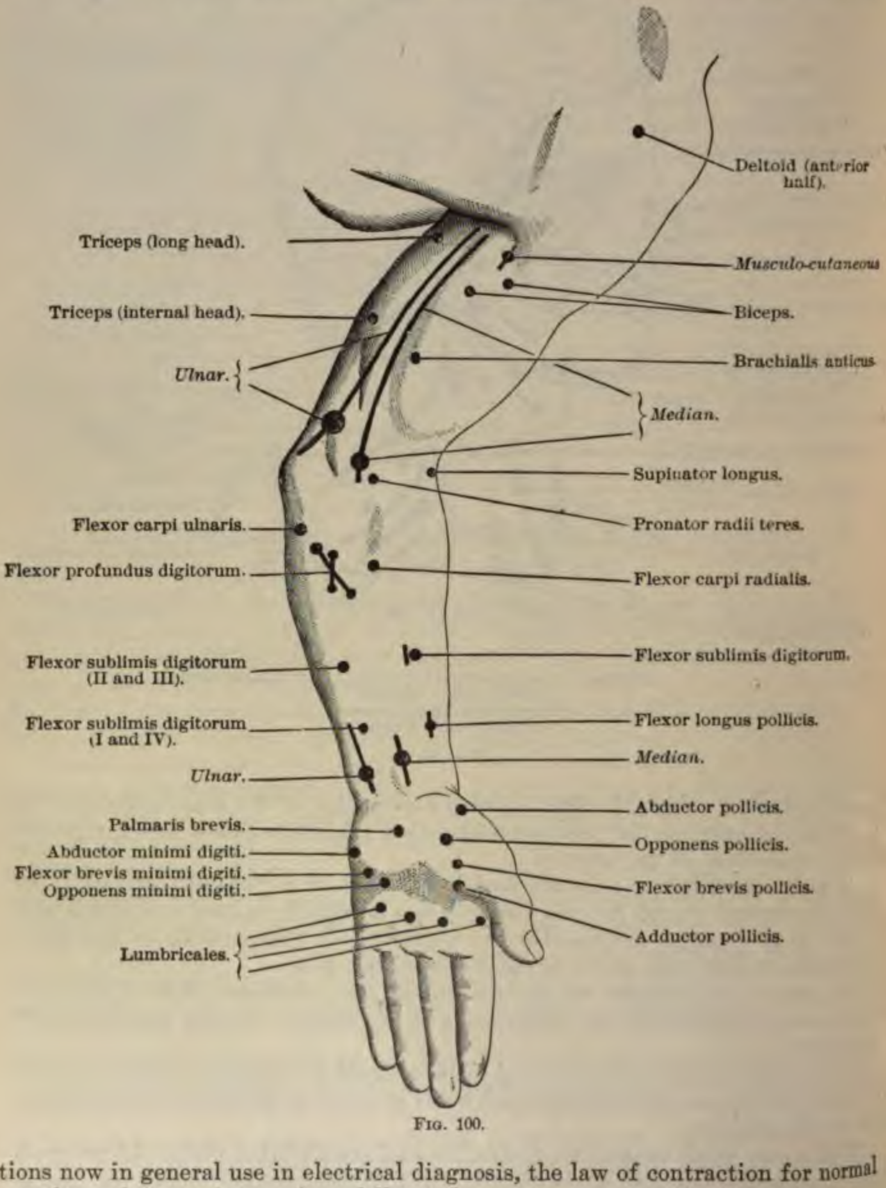

FIG. 100.

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* Ka signifies cathode, An = anode, S = closure, O = opening, z (Zuckung) = weak contraction, Z = stronger contraction, Te = tetanus. Sometimes the increasing strength of the contractions is abbreviated by the signs Z, Z′ and Z″. [Many English and American writers on electricity use letters

1. Lowest degree with weak currents: **KaSz**, KaO—, AnS—, AnO—.
2. Middle degree with stronger currents: **KaSZ**, KaO—, **AnSz**, **AnOz**.
3. Highest degree with very strong currents: **KaSTe**, **KaOz**, **AnSZ**, **AnOZ**.

The variations from the normal state seen under pathological conditions consist of quantitative and also of qualitative changes in the law of contraction. We term the simple increase or diminution of the electrical excitability in nerves or muscles, without simultaneous changes in the quality and order of the occurrence of muscular contractions, quantitative changes. The discovery of increased or diminished excitability of nerve and muscle can be made most easily in unilateral diseases, where we can compare the strengths of current required to obtain the minimal contraction on the diseased and healthy sides with each other. This is now determined without any special difficulty, since the general introduction of the absolute galvanometer. With the galvanometer thrown out of circuit we find with how many cells or at what position of the rheostat the first distinct cathodic

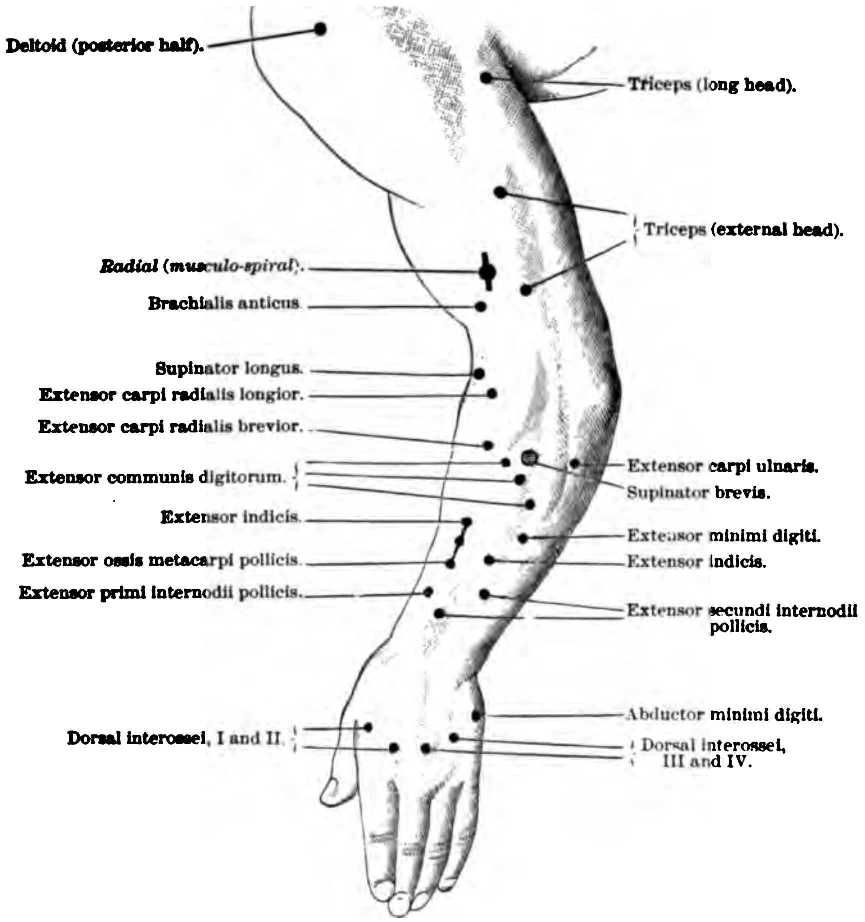
Deltoid (posterior half).
Triceps (long head).
Triceps (external head).
Radial (musculo-spiral).
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor communis digitorum.
Extensor carpi ulnaris.
Supinator brevis.
Extensor indicis.
Extensor minimi digiti.
Extensor ossis metacarpi pollicis.
Extensor indicis.
Extensor primi internodii pollicis.
Extensor secundi internodii pollicis.
Abductor minimi digiti.
Dorsal interossei, I and II.
Dorsal interossei, III and IV.

FIG. 101.

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derived from the English names. Thus C stands for cathode, closure, and contraction. A and O have the same meaning. It seems to us clearer and conducive to greater harmony to retain the German abbreviations, which are simple and definite.—TRANS.]

closure contraction (KaSZ) occurs. Then the galvanometer is put into the circuit and the strength of the current in milliampères is read off directly. By

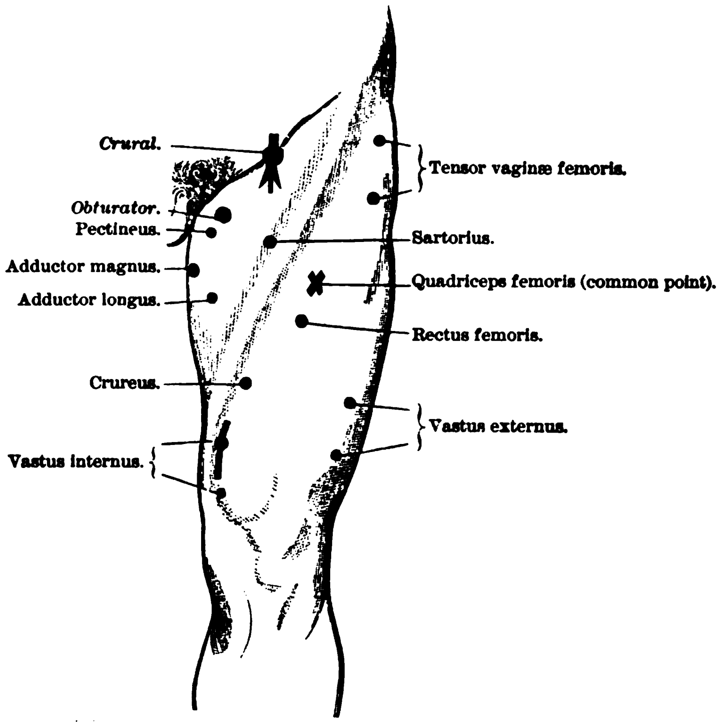

FIG. 102.

extensive investigations on healthy persons Stintzing has found certain limits within which the normal excitability of the different nerves varies. Deviations from these figures above or below normal accordingly permit us to recognize an increase or a diminution of the galvanic excitability. It is to be borne in mind that Stintzing uses a small "normal electrode" with a surface of three square centimetres. Some of the most important averages found by Stintzing are shown in the following table:

Facial nerve	1.0–2.5	milliampères.
Accessory nerve	0.1–0.4	"
Ulnar nerve	0.2–0.9	"
Median nerve	0.3–1.5	"
Radial nerve	0.9–2.7	"
Crural nerve	0.4–1.7	"
Peroneal nerve	0.2–2.0	"
Tibial nerve	0.4–2.0	"

An accurate measurement of the absolute strength of the faradic current is possible, but it has as yet been but little introduced into practice. We usually content ourselves, as we have said, with the statement of the coil distance at which the first perceptible contraction occurs. Stintzing found as an average position for the frontal nerve, 128.5 millimetres; for the accessory nerve, 137 millimetres; for the radial nerve, 105 millimetres; for the median nerve, 122.5 millimetres; for the ulnar nerve, 130 millimetres; for the crural nerve, 111.5 millimetres; for the peroneal nerve, 115 millimetres; and for the tibial nerve, 107.5 millimetres. We must refer to the special works for further details.

Changes in the quantitative electrical excitability are not uncommon in pathological conditions, but they have no very great practical diagnostic significance. A pathological increase of electrical excitability is seen especially in tetany, a diminution in many cases of neuritis, and sometimes also in myelitis, pressure paralyses, etc. The apparent diminution of excitability is often due only to increased resistance (dry, thick skin, thick layer of fat, etc.).

Much more important, however, than the simple quantitative changes of electrical excitability are those not merely quantitative but also qualitative deviations from the normal law of contraction, which were first discovered in certain forms of paralysis by Baierlacher in 1859, and were soon generally confirmed. Erb has given these the name of the "reaction of degeneration," because they are closely connected with the progress of certain anatomical changes in the paralyzed muscles and nerves.

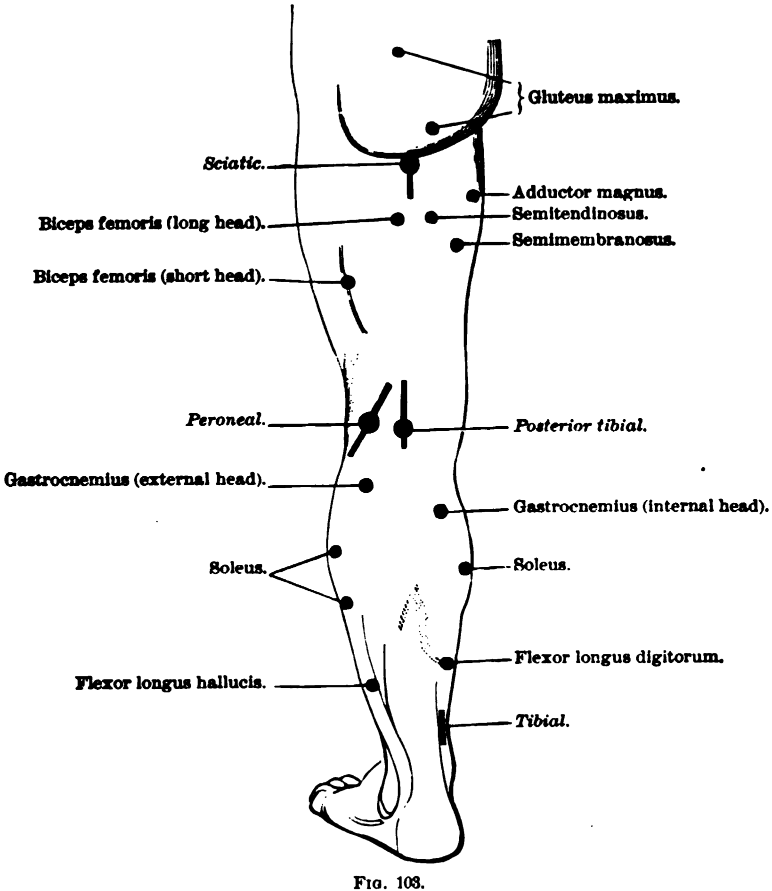

FIG. 103.

In order to make the relations of the reaction of degeneration clear, let us select as an example any fresh peripheral paralysis and follow the changes in excitability to the two currents in the nerves and muscles. In a short time (two or three days) after the onset of the paralysis a gradually increasing decline in the faradic and galvanic excitability in the nerve begins. After one or two weeks the excitability is completely lost, so that from the nerve we can no longer provoke any trace of muscular contraction with the strongest faradic or constant current. During this time the excitability of the paralyzed muscles to the faradic current has also rapidly diminished, and finally has wholly disappeared. The case is quite different with galvanic excitement of the muscles. Here we find at first a slight diminution, which in the second week passes to a decided increase of the galvanic muscular excitability. We now obtain marked muscular contractions with relatively very weak currents. Besides that, two other very important peculiarities are to be noted: 1. The muscular contractions are first of all not short and lightning-like, as under normal conditions, but they seem quite sluggish, protracted, "worm-like," and often persist during the whole duration of the closure of the current. 2. The muscular contractions occur not only chiefly at cathodic closure (KaS), as under normal conditions, but the anodic closure contractions (AnSZ) are as strong as the cathodic closure contractions (KaSZ), or even plainly exceed them. With stronger currents anodic closure tetanus (AnSTe) is readily obtained. The cathodic opening contraction (KaOZ) is also frequently stronger. 3. It may finally be mentioned here that the mechanical irritability of the muscles in such cases is usually increased, and the muscles when struck also show a slow, worm-like contraction.

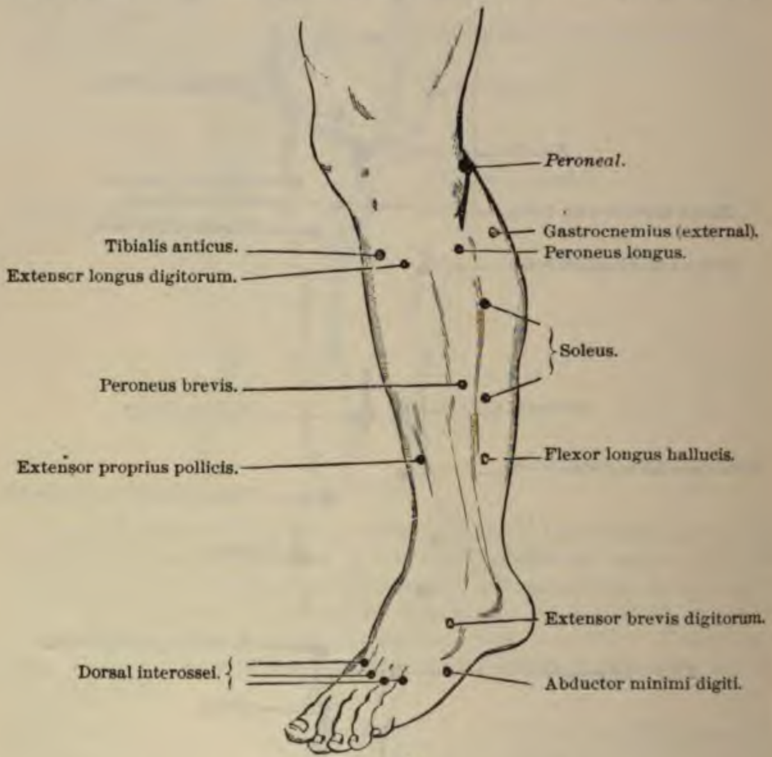

FIG. 104.

This second degree of the reaction of degeneration lasts from four to eight weeks. If the paralysis be severe and long continued, or incurable, at the end of this period comes a decline of the galvanic muscular excitability. The contractions become weaker, the strength of current necessary to produce them greater, and, finally, in incurable cases, even with the strongest currents, we can obtain only a little slow anodic closure contraction (AnSZ), or none at all. It is different, however, in the milder, curable cases. In these the passage to the normal condition gradually follows either the increase of the galvanic muscular excitability, or, in more protracted cases, its secondary decline. The contractions become more vigorous and shorter, the cathodic closure contraction (KaSZ) again predominates, the faradic muscular excitability and the faradic and galvanic excitability of the nerves finally return, and with them the old normal conditions are restored. A fact to be observed in these cases is of great interest, namely, that the voluntary motion in such cases often returns decidedly earlier than the electrical excitability of the peripheral nerves. We see, then, that a diseased nerve may be capable of conducting excitations coming from the brain, while the taking up of excitation, its direct excitability, is still completely lost. In such cases we can obtain a muscular contraction by electrical excitation of the nerve above the point of lesion.

Besides the complete reaction of degeneration just described, there is also a so-called partial reaction of degeneration, which is not infrequent in milder cases. This may show itself in several forms, but it is chiefly when the diminution of the faradic and galvanic excitability in the nerves and the diminution of faradic excitability in the muscles is only of a slight degree, while the characteristic changes in the direct galvanic excitement of the muscles—ready appearance of anodic closure contractions (AnSZ), and especially the noticeably slow character of the ensuing muscular contractions—are fully developed. In general the slow muscular contraction on direct galvanic irritation of the muscles is practically the most important symptom of reaction of degeneration. We sometimes see the occurrence of slow contractions on faradic excitement of nerves and muscles—"faradic reaction of degeneration," and in some cases we even see slow muscular contractions on indirect galvanic irritation of the nerve (partial reaction of degeneration with indirect slowness of contraction). In the course of atrophic paralyses we often see that the different varieties of reaction of degeneration pass into one another as the process advances or improves (Stintzing).

Anatomical Changes of the Nerves and Muscles in the Reaction of Degeneration; its Significance in Diagnosis and Prognosis.—As we have previously seen, all paralyses may be divided into two great groups—into atrophic paralyses, and paralyses without marked atrophy of the affected muscles. We have learned to recognize the necessary hypothesis of the "trophic" influence of the ganglion-cells in the anterior cornua of the spinal cord as the foundation of this distinction, an influence which is easily to be understood when we consider that ganglion-cell and peripheral motor nerve-fiber form an anatomical unit with which the muscular fiber is directly connected as a terminal organ. In all cases where the disease affects these ganglion-cells, or is situated in the peripheral nerves, so that the trophic influence of the ganglion-cells on the muscles can no longer be of influence, we have a degenerative atrophy of the peripheral portion of the nerve, and also a relatively rapid atrophy of the muscle belonging to it. This secondary degeneration of the peripheral nerves and the consequent atrophy of the muscles are the anatomical causes of the phenomena of electrical reaction of degeneration.

If we have to do with a peripheral paralysis, such as a traumatic lesion of a nerve-trunk, the portion of the nerve peripheral to the point of lesion is separated from its cell of origin in the cord, and begins to undergo secondary degeneration. The first anatomical sign of the degeneration is a breaking down of the medullary sheath at first into large and gradually into smaller flakes and drops. The axis cylinder is also soon destroyed, so that the sheath of Schwann finally incloses only approximately homogeneous fluid contents, which are in great part rapidly absorbed. At the same time there is an increase of the nuclei in the sheath of Schwann, and this increase, when the process is long continued, leads to a decided increase of the interstitial connective tissue in the nerve. The diminution and final loss of electrical irritability in the nerve are perfectly parallel to these anatomical changes, for we can readily understand that such a completely destroyed nerve can be excited neither by the faradic nor by the galvanic current.

The degeneration of the nerve involves its finest terminal branches in the muscles; but the muscle itself, as we have said, does not remain unchanged. The muscular fibers undergo a marked atrophy. They become much smaller, their transverse striation may be preserved for a long time, but it often becomes somewhat less distinct. Some muscular fibers show a peculiar yellow homogeneous consistency which is called "waxy degeneration." Fatty or marked granular degeneration of the fibers occurs only in the later stages. The motor end-plates of the nerves in the muscles are spared for a comparatively long time, and they disappear only when the degeneration of the muscles has reached the highest degree. There is also a considerable increase in the muscle nuclei and in the later stages a considerable new formation of interstitial connective tissue, often associated with an abundant deposit of fat. Muscles thus altered react only to the galvanic current and in the slow manner described above. We recognize from this that the muscle deprived of its nerve still retains a certain trophic independence. The slow galvanic contraction is the expression of the purely muscular irritability which is still retained by itself in the muscle deprived of its nerve and consequently atrophied, while the faradic irritability is lost because the faradic irritation of the muscle can be accomplished only by means of the terminal branches of the nerve in the muscle.

In the incurable cases the processes of degeneration just described gradually advance, but, in the cases that recover, a number of processes of regeneration begin sooner or later. We can not here go into the finer details. We would mention merely that the "regeneration" in all probability takes place only from the central nerve-stump; that it consists, therefore, essentially of a new outgrowth from the maimed neurones. The old nerve-sheaths of the cut-off or degenerated peripheral portion serve as conduction tubes. The axis cylinders grow out first, and then new medullary sheaths are formed. The end-plates of the nerves are restored very early, according to Gessler, even before the nerve-fibers. The muscular fibers also increase in volume at the same time.

Voluntary motion in the previously paralyzed muscle returns hand in hand with the processes of regeneration. It appears, as has been already mentioned, sooner than the normal electrical excitability of the nerves and muscles, because it can take place as soon as the axis cylinders are again formed, while the normal electrical excitability of nerves and muscles can take place only after the restoration of the medullary sheaths. We usually express this fact by saying that the reception of peripheral irritations depends upon the persistence of the medullary sheath. The destroyed medullary sheaths perhaps form a sort of isolating layer about the axis cylinders, which is only a poor conductor of the electrical current. This is the reason why in diseases of the peripheral nerves we sometimes find reaction of degeneration in muscles and nerves which have lost none of their voluntary motility. In such cases there is probably disease of the medullary sheaths without much degeneration of the axis cylinders.

The same anatomical changes, which we have just described as a secondary degeneration in lesions of the peripheral motor nerves, also develop, if the primary disease has its seat in the anterior cornua of the gray matter of the spinal cord—that is, in the trophic centers themselves. In these cases, of course, the form of the disease has nothing to do with it. Both in the different forms of inflammation and of primary atrophy, and also in new growths, which affect the anterior gray matter of the cord, a secondary degeneration, with pronounced reaction of degeneration, develops from the anterior roots of that portion of the cord affected to the ends of the peripheral nerves, and even to the corresponding muscles. We shall also learn to recognize a number of primary degenerations of the peripheral nerves, such as primary neuritis, diphtheritic and toxic paralyses, etc. In such cases we find almost precisely the same anatomical changes as in secondary degenerations, and, in consequence of these changes, there is also electrical reaction of degeneration in the paralyzed nerves and muscles. In all cerebral paralyses, however, and in those spinal paralyses where the cause of the paralysis is situated above the part of the anterior gray cornua concerned, the degeneration of the peripheral nerves, and also the reaction of degeneration, are entirely wanting.

We thus see that the reaction of degeneration, in regard to diagnosis, at once permits us to decide that the disease is situated in the anterior gray cornua, in the anterior roots, or in the peripheral nerves. It does not permit any further distinction. In regard to prognosis, it teaches us that anatomical changes have taken place in the nerves and muscles, from which, under some circumstances (that is, if the ganglion-cell bodies are still preserved), a restoration is still very possible, but at all events it can take place only after the lapse of a considerable time, at least two or three months. We shall soon learn to recognize a number of mild peripheral paralyses in which there is generally no reaction of degeneration. From the absence of reaction of degeneration we can then draw the conclusion, with certainty, that no coarse anatomical changes are present in the nerve, and that we may expect a much more rapid recovery from the trouble, perhaps in three or four weeks. The partial reaction of degeneration, above mentioned, is also an important symptom in regard to prognosis. It shows probably that slight changes have occurred only in the terminal branches of the muscle nerves, while the larger nerve-trunks have remained approximately normal. The latter have therefore retained their electrical excitability while the terminal branches of the nerves have lost it. The partial reaction of degeneration therefore always permits a more favorable prognosis in regard to time than the complete reaction of degeneration.

6. Table of the Different Forms of Voluntary Motion and the Muscles and Nerves concerned Therein

In order to obtain an accurate knowledge of the extent of the paralysis in complicated paralytic conditions (hemiplegia, paraplegia, muscular atrophy, etc.), it is imperatively necessary to test all the muscle groups in order. The performance of these tests and their diagnostic value and significance will be materially aided by the use of the following table, arranged according to movements.

I. MUSCLES OF THE HEAD AND TRUNK

1. *Facial Muscles and Muscles of Mastication*

1. Wrinkling the forehead transversely (Mm. frontalis et occipitalis, N. facialis).
2. Wrinkling the forehead longitudinally (M. corrugator supercilii, N. facialis).
3. Closing the eyes (M. orbicularis palpebrarum, N. facialis).
4. Dilating the nostrils (M. compressor nasi et M. levator alæ nasi, N. facialis).
5. Drawing the mouth outward and upward (Mm. levator labii superioris, zygomatici, risorius, N. facialis).

2. Drawing the shoulder-blade toward the median line (Mm. rhomboidei, Mm. thoracici posteriores from 4 and 5 C. N.).

3. Fixation and rotation of the shoulder-blade in raising the upper arm to a vertical position (M. serratus anticus major, N. thoracicus major from 5 C. N.).

2. *Movements at the Shoulder*

1. Raising the arm forward and outward (M. deltoideus, N. axillaris* [circumflex] from 4 and 5 C. N.).

2. Adduction and drawing downward (Mm. pectorales major et minor, N. thoracici anteriores from 6 C. N., M. latissimus dorsi, N. subscapularis longus from 5 and 6 C. N.).

3. Outward rotation (Mm. supraspinalis, infraspinalis, teres minor, N. suprascapularis from 4 C. N.).

4. Inward rotation (Mm. subscapularis et teres major, N. subscapularis from 6 C. N.).

3. *Movements at the Elbow*

1. Flexion (Mm. biceps et brachialis anticus, N. musculo-cutaneus; 5 and 6 C. N., M. supinator longus, N. radialis [musculo-spiralis]; 6 and 7 C. N.).

2. Extension (M. triceps, N. radialis, 6 and 7 C. N.).

3. Pronation (Mm. pronator teres et pronator quadratus, N. medianus; 6 C. N.).

4. Supination (M. biceps, N. musculo-cutaneous, M. supinator brevis, N. radialis; 5 C. N.).

4. *Movements at the Wrist*

1. Dorsal extension (Mm. extensor carpi radialis longus et brevis, extensor carpi ulnaris, N. radialis; 6 and 7 C. N.).

2. Volar flexion (Flexor carpi radialis, N. medianus. Flexor carpi ulnaris, N. ulnaris; 7 and 8 C. N.).

3. Radial flexion (M. extensor carpi radialis longus).

4. Ulnar flexion (Mm. flexor et extensor carpi ulnaris).

5. *Movements of the Fingers*

1. Extension of the proximal phalanges (Mm. extensor digitorum communis, extensor indicis, extensor minimi digiti, N. radialis; 6 and 7 C. N.).

2. Extension of the distal phalanges [phalangines and phalangettes] (M. interossei, N. ulnaris).

3. Flexion of the proximal phalanges (Mm. interossei et lumbricales, Nn. ulnaris et medianus; 7 and 8 C. N.).

4. Flexion of the distal phalanges (Mm. flexor digitorum sublimis et profundus, the latter for the third phalanx [phalangettes], N. medianus; 7 and 8 C. N.).

5. Spreading the fingers, abduction (Mm. interossei externi [dorsales], N. ulnaris; 8 C. N.).

6. Adduction of the fingers (Mm. interossei interni [palmares], N. ulnaris).

7. Flexion of the proximal phalanx and abduction of the little finger (Mm. flexor brevis et abductor digiti minimi in the hypothenar eminence, N. ulnaris).

6. *Movements of the Thumb*

1. Extension of the metacarpal and both phalanges (Mm. extensor pollicis brevis et longus, the latter for the second phalanx, N. radialis; 8 C. N.).

2. Abduction of the metacarpus (M. abductor pollicis longus, N. radialis).

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* We have followed Henle's nomenclature for the peripheral nerves.—K.

3. Adduction of the metacarpus (M. adductor et caput profundum flexoris brevis, N. ulnaris).

4. Flexion and opposition of the metacarpus, flexion of the first phalanx with simultaneous extension of the distal phalanx (Thenar muscles: Mm. opponens, abductor brevis et caput superficiale flexoris brevis, N. medianus; 8 C. N.).

5. Flexion of the second phalanx (M. flexor pollicis longus, N. medianus).

III. LOWER EXTREMITY

1. *Movements at the Hip*

1. Flexion (Mm. ilio-psoas, sartorius, N. cruralis, M. tensor vaginæ femoris, N. glutæus superior; 2 and 3 L. N.).

2. Extension (M. glutæus maximus, N. glutæus inferior from the sciatic plexus; 1 S. N.).

3. Abduction (M. glutæus medius et minimus, N. glutæus superior; 1 S. N.).

4. Adduction (Mm. adductor brevis, adductor longus, adductor magnus, pectineus, gracilis, N. obturatorius from lumbar plexus; 3 L. N.).

5. Outward rotation (Mm. pyriformis, obturatur internus, gemelli, quadratus femoris, N. ischiadicus, M. obturator externus, N. obturatorius from lumbar plexus; 5 L. N.).

6. Inward rotation (Mm. glutæus medius et minimus, N. glutæus superior; 1 S. N.).

2. *Movements at the Knee*

1. Extension (M. extensor cruris quadriceps, N. cruralis; 3 and 4 L.N.).

2. Flexion (Mm. biceps, semimembranosus et semitendinosus, N. ischiadicus; 4 and 5 L. N.).

3. *Movements at the Ankle and of the Toes*

1. Dorsal extension of the inner edge of the foot (M. tibialis anticus, N. peroneus; 2 S. N.).

2. Dorsal extension of the outer edge of the foot (Mm. peroneus longus et brevis, N. peroneus; 2 S. N.).

3. Plantar flexion of the foot (Mm. gastrocnemius et soleus, N. tibialis; 1 S. N.).

4. Adduction of the inner edge of the foot (M. tibialis posticus, N. tibialis).

5. Abduction of the foot (M. peroneus brevis, N. peroneus).

6. Dorsal extension of the toes (Mm. extensor digitorum communis et extensor hallucis longus, N. peroneus; 1 and 2 S. N.).

7. Plantar flexion of the toes (Mm. flexores digitorum et hallucis longi et flexor digitorum brevis, N. tibialis; 1 and 2 S. N.).

CHAPTER II

THE DIFFERENT FORMS OF PERIPHERAL PARALYSIS

1. PARALYSIS OF THE OCULAR MUSCLES

Ætiology.—The largest part of all the ocular paralyses arise from affections which involve either the peripheral oculo-motor nerves or their nuclei in the brain-stem. We accordingly make a distinction between peripheral and nuclear paralyses of the ocular muscles. As we shall take up the latter more fully in the description of chronic bulbar paralysis, we have here to mention only the most important and most frequent causes of the peripheral ocular paralyses. These are as follows:

1. Injuries which directly affect the nerve-trunks or their branches: blows on the eye, knife-stabs, fractures of the skull involving the orbit or the base of the skull, and the like.

2. Compression of the nerves from morbid processes in their neighborhood. Tumors of the base of the skull, especially, very often lead to ocular paralyses. Periostitis at the base of the skull or in the orbit may also cause similar symptoms, and so may syphilitic diseases of the nerves and their surroundings, the meninges or periosteum, aneurisms of the basilar artery, acute or chronic meningitis in its different forms, etc. In all these cases we usually have to do with a pure mechanical compression of the affected nerves by the morbid new growths in their immediate vicinity. More rarely the pathological process directly invades the nerves themselves.

3. The so-called rheumatic ocular paralyses are quite common. These arise after some decided exposure to cold, such as a draught from an open window, and are in all probability very largely of a peripheral nature. They depend, as is supposed, upon an acute neuritis of the affected nerve, and hence correspond completely to the other rheumatic paralyses, such as rheumatic facial paralysis. Among the "rheumatic paralyses" we usually class the paralyses which apparently come on spontaneously and completely recover, for which no other special cause can be made out.

4. The ocular paralyses that sometimes arise after certain acute diseases are also of a peripheral nature, and are due to degenerative neuritis of the affected nerves. They are most frequent as a result of diphtheria, and are very much more rare in typhoid fever, acute rheumatism, etc. Of chronic diseases, diabetes mellitus may sometimes give rise to ocular paralyses, especially to paralysis of accommodation. The ocular paralyses occurring as a symptom of alcoholic polyneuritis or polioencephalitis may be referred to a toxic influence that is recognized.

5. Finally we often see ocular paralyses as a symptom of more extensive nervous disease, especially in tabes, multiple sclerosis, etc. The details of these conditions will be found in the description of the several diseases.

Symptoms.—Since we must refer to the text-books of ophthalmology in regard to the more precise symptomatology, and the more special methods of ophthalmic investigation, we will here give only a brief review of the chief symptoms of ocular paralyses which are important in nervous pathology.

The disturbance in the mobility of one eye is usually first noticed by the patient himself from the appearance of double images—double vision, or diplopia. These arise because, on looking to one side, the eye on the paralyzed side can not be brought into the corresponding position (strabismus), and consequently the retinal images no longer fall upon the same spots, the two images are no longer projected outward in a corresponding fashion, so that two different images are therefore produced of the same external object. In pathological convergence of the visual axes homologous double images arise, in pathological divergence crossed images—that is, in the first case, on closing one eye, the image disappears on the same side, in the second case it disappears on the opposite side. By alternately fixing the gaze on one or the other of two fingers held in line with each other, and by regarding the disappearance, on closing one eye, of the double image of the finger not fixed, we can easily demonstrate this on ourselves. If, then, crossed double images arise, for example, on looking to the right, we must have a divergent strabismus—that is, an imperfect function of the left internus;

but if there are homonymous double images, there must be a convergent strabismus, and consequently a weakness of the right abducens. It makes it much easier to test the double images if we put a colored glass before one of the patient's eyes. False projections of the visual field arise, in connection with the double images and with the abnormal strength of innervation which the patient has to use for his ocular muscles, so that the patient's judgment of the position of external objects is uncertain. In the more extensive ocular paralyses this often leads to a pronounced feeling of dizziness. In order to avoid this unpleasantness many patients confine themselves to monocular vision, close the affected eye, or put their heads in a position to avoid the double images.

Finally, we must mention a symptom to be observed in almost every ocular paralysis—the so-called secondary deviation of the healthy eye. If, after the sound eye has been covered, we have the paretic eye fix itself upon a point which it can not reach at all, or which it can reach only after the utmost exertion, we see, when the covering hand is taken away from the sound eye, that the latter has been moved much too far in the corresponding direction. The abnormal exertions of innervation with the affected eye, somewhat after the analogy of certain associated movements, pass over to the associated muscle of the healthy side and cause in it too extensive a contraction.

Physical examination gives the following results, according to the extent of the paralysis:

In complete paralysis of one **oculo-motor nerve** (the levator palpebræ superioris, the superior, inferior, and internal recti, the inferior oblique, the sphincter of the iris, and the ciliary muscle) the first thing that is noticed, besides the disturbance in the movements of the eye, is the more or less complete drooping of the upper lid—ptosis. If the patient wishes to lift the upper lid he can not do so at all or only to a slight degree. If he tries very hard there is usually an evident contraction of the corresponding frontalis muscle. If we ask the patient to follow with his eyes the motion of any object, such as the finger, held before him, while he keeps his head still, we notice at once that the affected eye does not move upward, downward, or inward. The pupil is dilated (mydriasis) and no longer contracts to light. Accommodation is lost, and distinct vision for near objects is impossible. As a rule, the whole eye seems rather prominent (paralytic exophthalmus), because the backward traction of the recti is very largely absent. In old oculo-motor paralysis there is often a secondary contracture in the unparalyzed external rectus (and superior oblique), by which the eye is persistently drawn outward. Partial oculo-motor paralyses are not infrequent, especially isolated ptosis, also paralysis limited to the levator palpebræ and superior rectus, paralysis of the internal, inferior, or superior rectus, or finally isolated paralysis of accommodation, and they may usually be easily recognized from what has been said.

Paralysis of the **abducens** is characterized by the inability to move the external rectus. The eye can no longer be moved, or it can be moved only imperfectly, outward beyond the median line. In old paralysis the eye is drawn inward from a secondary contracture of the internal rectus, and convergent strabismus arises. Paralysis of the abducens may often be isolated, but it is sometimes bilateral, or combined with other ocular paralyses.

Paralysis of the **trochlear nerve**, the superior oblique muscle, is not perfectly easy to recognize; but it is rarely of special practical importance. The action of the superior oblique coincides mainly with that of the inferior rectus. If the trochlear be paralyzed the eye turns somewhat inward on looking downward, because the superior oblique acts also to abduct the eyeball, and therefore acts chiefly on looking downward and outward. The paralysis of the trochlear nerve is sometimes made manifest by the failure of the eye to revolve, which rotation normally takes place on looking downward, and is due to the superior oblique muscle. This latter movement takes place in each eye about a sagittal axis, in such a way that the left eye is turned from the left and up to the left and downward, and the right eye from the right and up to the right and downward. In regard to diagnosis, it is also characteristic that the double images in trochlear paralysis appear only in the lower half of the field of vision, and especially on looking downward. Hence it happens that the disturbance of vision is especially manifest in going up or down stairs, because in such cases the double images of the steps are disturbing.

The following must be added in regard to the separate clinical forms of ocular paralysis: Rheumatic ocular paralysis affects the abducens most frequently, and not infrequently the oculo-motor or one of its branches, as in isolated ptosis. We have seen a rare case in which exposure to severe cold had been followed by a complete paralysis of all the external muscles of the right eye, with complete ptosis, and almost absolute immobility of the eye in all directions. The rheumatic ocular paralyses are almost always acute in their onset, and they are often during the first period associated with sensations of pain about the eye and in the head. Vomiting (of reflex origin?) is also not rare at the beginning of the affection. The course of most cases is favorable, since they usually completely recover in a few weeks, though sometimes not for months. In some cases the paralysis may remain stationary.

The diphtheritic ocular paralyses usually appear, like the other diphtheritic paralyses, a week or two after the termination of the disease. They most frequently affect the muscles of accommodation, so that the patient complains chiefly of indistinct vision for near objects; but we sometimes see paralyses of the external ocular muscles also, the abducens or the internal rectus. The prognosis of diphtheritic paralyses is almost invariably favorable.

Finally, we must mention here the so-called "**recurrent or periodical oculo-motor paralysis,**" to which Möbius and others have lately called attention, and whose nature is still almost wholly inexplicable. In the cases of this sort repeated paralyses of one oculo-motor nerve, often associated with headache and vomiting, just as in migraine, come on at longer or shorter intervals (in women sometimes at the menstrual period) in the same individual, who has often had them since childhood. All branches of the oculo-motor are usually affected alike, but sometimes the internal ocular muscles are spared. In milder cases there may be only ptosis. The individual attack sometimes lasts only a few days, but often some weeks. The attacks usually become gradually more severe later on. The nature of recurrent oculo-motor paralysis is still quite unknown, but it seems to be growing more probable that in a part of the cases at least the disease is closely related to migraine (*q. v.*), and forms in a measure only an unusual development of migraine—*migraine ophthalmoplégique.* The genuine cases of the disease, however, must, of course, be distinguished from the recurrent oculo-motor paralysis that sometimes occurs in tabes, cerebral syphilis, tumors of the base of the skull, etc. [The relation to migraine seems very doubtful. There is rarely complete recovery between the attacks, and there is a tendency for the attacks to increase in frequency and duration as well as in severity. In three cases which have come to autopsy lesions have been found affecting the nerve—tumors or inflammatory thickening. Some of the cases have also had anæsthesia in one or more branches of the distribution of the trigeminus during the attack. Besides the recurrent oculo-motor paralysis and recurrent facial paralysis (*vide infra*), recurrent trochlear, and abducens paralyses and recurrent paralysis of several cranial nerves at once have been reported.—K.]

Nothing generally applicable can be said as to the course and prognosis of the other forms of ocular paralyses, since in them everything depends upon the form of the underlying disease.

Treatment.—In regard to the fulfillment of any possible causal indication we must remember especially that ocular paralysis is not very infrequently of syphilitic origin. Iodide of potassium and energetic mercurial inunction may sometimes give very good results in such cases. Hence these remedies must also be tried in doubtful cases.

Of other remedies galvanic treatment gives the speediest relief. We pass weak currents transversely through the temples, or, what is usually better, we put the anode to the back of the neck and apply the labile cathode to the closed eye, especially to the region corresponding to the paralyzed muscles. Great caution, weak currents, and the avoidance of any great variations in the currents are of course necessary. We may also try preparations of strychnine internally, or, better, subcutaneously in the vicinity of the eye. We must refer to special treatises with regard to a correction of the double images by covering the affected eye, spectacles with one dark or ground glass, or prismatic spectacles, or in regard to operations, such as tenotomy, that are sometimes performed.

2. PARALYSIS OF THE MOTOR BRANCH OF THE TRIGEMINUS

(Paralysis of the Muscles of Mastication)

Paralysis of the muscles of mastication, the masseters and temporals, supplied by the third branch of the trigeminus, is a rare affection. It is most frequently seen in diseases of the base of the skull which compress the motor branch of the fifth. We shall also learn later on to recognize paralysis of the muscles of mastication as a rare symptom of chronic bulbar affections.

The chief symptom of motor paralysis of the trigeminus is the difficulty or impossibility of chewing. In unilateral paralysis the patient can chew only on the healthy side; in bilateral paralysis he can no longer chew at all. Paresis of the muscles of mastication can be readily determined by the lack of strength in resisting the attempt to draw down the lower jaw or by the patient's inability to make a deep imprint in biting a bit of wood (lead-pencil, etc.). In complete paralysis of the muscles of mastication the lower jaw hangs loosely down, and it can also no longer be moved sideways, from the co-existing paralysis of the pterygoids. We can not usually detect clinically disturbances in the other muscles supplied by the trigeminus (mylohyoid, buccinator, tensor palati, and tensor tympani), but there are often sensory disturbances in the distribution of the trigeminus at the same time.

The prognosis and treatment depend upon the primary disease. Local faradization or galvanization of the paralyzed muscles is to be tried.

3. FACIAL PARALYSIS

(Mimetic Facial Paralysis. Bell's Palsy)

Ætiology.—Facial paralysis is one of the commonest peripheral paralyses, as we can understand from the superficial position of the nerve, exposing it to external injuries, and from its course through the narrow Fallopian canal. We consider it improbable that a special "neuropathic family predisposition" plays a part in the occurrence of peripheral facial paralysis, as Charcot's school maintains. The most important causes of facial paralysis are: 1. Exposure to cold, draughts, sleeping by an open window, riding in the cars with the window open, etc. The paralyses arising in this way are termed "rheumatic," and we also include in this class those peripheral paralyses which are apparently spontaneous—that is, those without any marked exposure to cold that can be discovered. In all these cases we probably have to do with a neuritis of the nerve-trunk, which comes on in a way as yet unknown (infectious? toxic?). [In a recent autopsy of a case of so-called "rheumatic" facial paralysis, Minkowski found a simple degenerative parenchymatous neuritis, without any swelling of the nerve-sheath or increase of connective tissue.—K.] 2. Diseases of the middle ear and especially caries of the petrous bone. As the facial passes through the Fallopian canal, which is in the immediate vicinity of the tympanic cavity, we can easily understand how so often in caries of the petrous bone, and often perhaps in purulent affections of the middle ear, inflammation may invade the trunk of the facial, or how the facial may be compressed by inflammatory exudations, etc. 3. In rare cases a pressure paralysis of the facial nerve arises in swelling of the parotid gland or tumors in its vicinity. 4. Diseases of the base of the skull or brain, tumors, syphilitic new growths, and acute or chronic inflammations, often give rise to the development of a facial paralysis by invasion of the trunk of the facial or compression of it. 5. We shall have to speak repeatedly in the following sections of the frequent implication of the facial nerve in diseases of the brain and medulla and of the implication of the facial muscles in multiple neuritis, in juvenile muscular dystrophy, and other diseases.

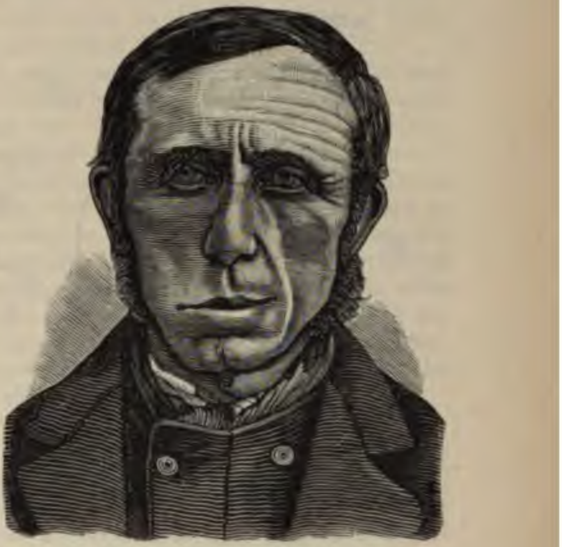

FIG. 105.—Right facial paralysis (after SEELIGMÜLLER). The folds are smoothed out and in part entirely absent on the paralyzed side, while they are strongly marked on the left. The mouth and nose are drawn to the left.

Symptoms and Course.—The manifold character and different functions of the nerves, which unite in the trunk of the facial, are the causes of the quite large array of symptoms in facial paralysis. Most of these symptoms are manifest only in peripheral facial paralysis, and the following description, therefore, refers chiefly to this.

The paralysis of the facial muscles of expression is the most striking and characteristic (see Fig. 105). The paralyzed half of the face is lax and expressionless, the wrinkles in the forehead are obliterated, the eye is abnormally wide open, as the weight of the lower eyelid draws it down, and the eye also waters (epiphora), the nasolabial fold is obliterated, the corner of the mouth droops, and saliva frequently flows from it. The paralysis becomes still more marked on any movement of the face, on wrinkling the forehead, turning up the nose, laughing, talking, whistling, or inflating the cheeks. The eye can be only partly closed. On attempting it, the upper lid sinks down from its weight (a weakening of the levator palpebræ superioris), the eye is turned upward, so that the pupil is covered, but quite a wide space is left between the eyelids (lagophthalmus). This defective closure of the lids facilitates the entrance of dust, etc., into the eye, and sometimes gives rise to conjunctivitis, or even to severer inflammation of the eye. Speech is rendered difficult and indistinct from defective movements of the lips, and mastication becomes impaired from the imperfect movement of the cheeks. The patient can no longer whistle because he can no longer pucker the lips sufficiently on the affected side. If he tries to puff out the cheeks or keep them inflated we notice plainly the flaccidity of the cheek on the paralyzed side. On drawing down the lower lip the paralysis of the platysma myoides is sometimes very plainly seen. The tongue is often apparently protruded to one side, but this is due merely to the distorted position of the mouth. Very often, however, we see, when the mouth is open and the tongue is at rest, that the half of the tongue corresponding to the paralyzed side lies deeper than the other half, a condition which is probably connected with a paralysis of the stylo-hyoid and digastric muscles. In some cases in which the geniculate ganglion or the facial trunk above it were affected, some writers have claimed to find a paresis of the soft palate on the affected side; the fibers from the facial are said to pass through the superficial petrosal nerve to the spheno-palatine ganglion, and thence to the soft palate. It droops more, and on phonation the soft palate is raised obliquely to the healthy side. According to more recent investigations, however, the soft palate is apparently innervated only by fibers from the vaso-accessory and not from the facial.

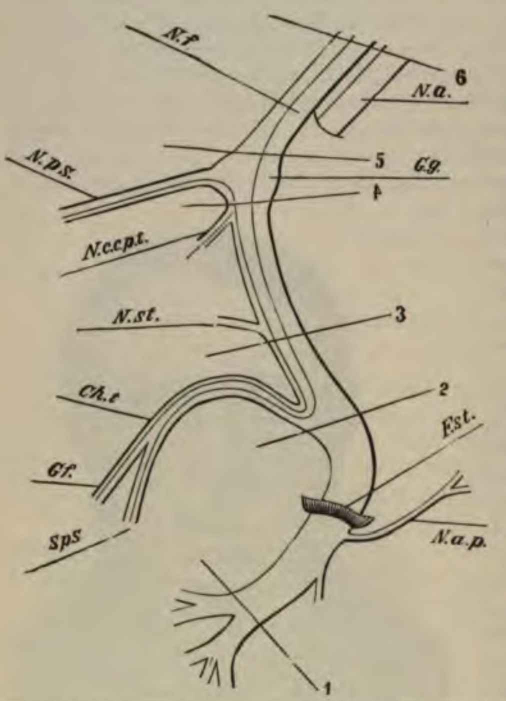

FIG. 106.—Schematic representation of the trunk of the facial from the base of the skull to the pes anserinus. Different localizations of the lesion in paralysis. *N.f.* Facial nerve. *N.p.s.* Great superficial petrosal. *N.c.c.p.t.* Nerve communicating with the tympanic plexus. *N.st.* Stapedius. *Ch.t.* Chorda tympani. *Gf.* Fibers of taste. *Sps.* Nerve governing the secretion of saliva. *N.a.* Acoustic nerve. *G.g.* Geniculate ganglion. *F.st.* Stylomastoid foramen. *N.a.p.* Posterior auricular nerve.

Disturbances of taste in the anterior two thirds of the tongue have been repeatedly found on the paralyzed side, but they usually attain only a slight degree. They are explained by an affection of the fibers of the chorda tympani, which run for some distance in the facial, as has been described on page 827. At the beginning of the paralysis many patients complain of subjective sensations of taste. Later on the dullness of taste may often be discovered by careful testing. Tactile sensibility in the tongue is only exceptionally diminished (sensory fibers in the chorda?). A diminished secretion of saliva (fibers in the chorda), and consequently an abnormal feeling of dryness in the patient's mouth on the paralyzed side, are sometimes said to have been observed. Disturbances of hearing are frequent, but they are usually due to some complicating aural trouble (*vide supra*), or to a co-existing affection of the acoustic nerve. Paralysis of the stapedius muscle, however, sometimes seems to cause symptoms, including a marked sensitiveness to all loud sounds, and even an abnormal acuteness of hearing, especially for low notes (hyperacusis, oxyokoia). These symptoms are due to the fact that in paralysis of the stapedius its antagonist, the tensor tympani, causes a greater tension of the membrana tympani. Reflex movements, winking, etc., are, of course, lost in complete peripheral facial paralysis. For the special reflexes, which are often seen in the later stages of facial paralysis, *vide infra.*

By testing all the symptoms described, in most cases we can decide with accuracy upon the place where the break in conduction in the facial must occur. If we examine the accompanying plan of the facial (Fig. 106), devised by Erb, we can easily understand the following chief symptomatic forms of facial paralysis. Of course we must not forget that many points (innervation of the soft palate, of the secretion of saliva and tears, etc.) are still very much in need of further investigation.

1. Paralysis of the facial muscles; but taste, secretion of saliva, hearing, and soft palate normal; seat of the affection in the portion between 1 and 2, usually the trunk of the facial below the Fallopian canal.

2. Paralysis of the facial muscles, disturbance of taste, and eventually diminished secretion of saliva; but hearing and soft palate normal; seat of the affection within the Fallopian canal between 2 and 3.

3. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing; but soft palate normal; seat between 3 and 4.

4. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing, and paresis of the soft palate (? *vide supra*); seat in the geniculate ganglion between 4 and 5.

5. Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, paresis of the soft palate (?), but no disturbance of taste; seat above the geniculate ganglion between 5 and 6.

The changes in electrical excitability, and some other changes, may be best described in connection with the course of facial paralysis. Ordinarily rheumatic paralysis usually begins quite suddenly; less frequently it is more gradual. Sometimes there are, for a short time, subjective prodromata, such as abnormal sensations of taste, slight ringing in the ears, and very often painful sensations in the ear, behind the ear, and in the face, which symptoms may be referred to the beginning of acute inflammatory processes in the nerve. In a few cases the occurrence of herpes vesicles has been observed in the distribution of the affected facial, a condition which may be most readily explained, in accordance with what was said on page 815, by reference to the numerous anastomoses between the branches of the facial and those of the trigeminus. Among other rare complications of simple peripheral facial paralysis we may mention neuralgic pain in the different areas of the trigeminus and slight optic neuritis.

In regard to the further course we distinguish the three following forms:

1. *The mild form of facial paralysis*, to which especially many rheumatic paralyses belong. The affection is usually referred only to the facial muscles, disturbances of taste, etc., being wholly absent. Electrical excitability in the facial and the paralyzed muscles remains entirely normal. Recovery is rapid, usually in two or three weeks. In these cases we may certainly suppose that there are no severe and deep-seated anatomical changes in the nervous or muscular fibers.

2. *The middle form of facial paralysis* (Erb). In this there is no complete reaction of degeneration, but only a partial one. The excitability of the nerve is somewhat diminished, but it is not lost. In the muscles, however, in about two or three weeks, there appears a decided increase of galvanic excitability to direct excitement. The anodic closure contraction (AnSZ) is also greater than the cathodic closure contraction (KaSZ), and the contractions are slow. In regard to prognosis we may decide from this that the recovery will still be quite rapid. It usually follows in from four to six weeks.

3. *The severe form of facial paralysis* is that in which there is a complete reaction of degeneration in the nerve and muscles, the details of which we have learned in the previous chapter—loss of faradic and galvanic excitability of the nerve, loss of faradic excitability of the muscles, and quantitative and qualitative changes in the galvanic excitability of the muscles. In this form there are always coarse processes of degeneration in the nerve and atrophy of the muscles, so that recovery follows, if at all, only after three to six months, or even later, because

the processes of regeneration require at least as much time for their accomplishment. We often see in the later stages of these cases special symptoms of motor irritation (Hitzig). These consist, first, of a slight or sometimes very pronounced tonic contracture of the paralyzed muscles, which is sometimes very striking; if such cases are examined carelessly, we may easily mistake the (flaccid) healthy side of the face for the paralyzed side; second, of single spasmodic contractions of the muscles, a true facial tic (*vide infra*) following the paralysis of the facial; third, of special associated movements—if the patient closes his eyes, winks, etc., there always follows a marked distortion of the corner of the mouth, which can not be suppressed; we have seen this symptom frequently in students in whom a peripheral branch of the facial has been injured in a duel; fourth, of an increased reflex irritability—on pricking the skin, or blowing on it, vigorous muscular contractions follow. We have often ourselves seen contractions in the affected facial muscles following a blow on the bridge of the nose, on the nasal bone, or on the forehead on the healthy side. These reflexes come from the skin, or perhaps in part from the periosteum and the fasciæ also. All these symptoms—the contracture, the abnormal twitchings, and the associated movements—may last for a very long time—for years in incurable or in imperfectly cured cases.

Prognosis.—The prognosis of facial paralysis depends, of course, in the first place, upon the primary disease, if any exists. Paralysis in tumors of the base of the brain, caries of the petrous bone, etc., is almost always incurable. The course of the paralysis in affections of the middle ear depends upon the curability of the latter disease. Very important data for the accurate prognosis of rheumatic paralysis are given by the electrical examination, as has been carefully described above. Of course, we can never form a definite judgment from this at the beginning of the paralysis, but only at the end of the first week. If, at the end of the first week or fortnight, the electrical excitability of the nerve still remains normal, we can almost certainly prophesy a rapid and favorable course. If reaction of degeneration appears, we can not count upon recovery in the most favorable cases before two or three months. Relapses almost never follow directly upon the course of a paralysis, but it is by no means rare to see peripheral facial paralysis occur several times in the same patient after intervals of some years. We have seen a man of about thirty who had a peripheral facial paralysis four times within a few years, which disappeared each time after a few weeks—a condition which possibly is to be regarded as analogous to the "periodical oculo-motor paralysis" (see page 857).

Diagnosis.—The symptoms of facial paralysis are so pronounced that the paralysis itself can always be easily recognized. In regard to the precise form of the paralysis and its cause, we can often decide only by considering the ætiological factors, such as injuries, exposure to cold, or aural affections. In distinguishing between peripheral and central (bulbar or cerebral) paralyses, the other coexisting bulbar or cerebral symptoms must first of all be considered. We shall learn to recognize more accurately later the different modes in which, in these cases, facial paralysis may be combined with paralysis of the other cerebral nerves, or the nerves of the extremities. In doubtful cases electrical examination is often of decisive value. Reaction of degeneration can be present only in peripheral paralysis, or in such bulbar paralyses as affect the fibers of the facial below the facial nucleus or affect the nucleus itself. In all cerebral paralyses the electrical excitability is perfectly retained. It may also be mentioned here briefly that in all cerebral facial paralyses the frontal muscles and the orbicularis palpebrarum are usually unaffected, while in peripheral paralyses they also are paralyzed.

Treatment.—The treatment of the underlying disease is of the greatest importance in all cases where any aural affection, any removable compressing swelling, as of the parotid, or syphilis, lies at the bottom of the trouble. The methods of treatment indicated in such cases are self-evident. In fresh rheumatic facial paralysis it is advisable at first to give a few doses of salicylate of sodium or antipyrine. In other cases electricity is the only remedy which can give sure results, although we must not overestimate its efficiency. In the earlier stages of the paralysis we may recommend the stabile conduction of a weak constant current through the auriculo-mastoid fossæ, four to six times a week for two or three minutes, at first the anode, then the cathode, to the affected side. Later on the chief treatment is peripheral galvanization, or eventually faradization of the muscles. We place the anode in the auricular fossa and slowly stroke the different nerve-branches and the muscles with the cathode. We can often confirm the fact, immediately after each sitting, that the eye closes better after galvanization of the orbicularis. Faradization excites a reflex irritation of the nerves from irritation of the skin, and hence is perhaps of service. Internal remedies (strychnine, etc.) are probably quite useless.

In secondary contractures we may obtain some favorable results by methodical stretching of the muscles by wooden wedges inserted under the cheeks and by massage.

4. Paralyses in the Region of the Muscles of the Shoulder

Isolated paralyses of these muscles are rare, with the exception of the practically important paralysis of the serratus. Disturbances in their functions are more frequent, as one symptom in complicated paralytic states, especially in progressive muscular atrophy, and in diseases in the neighborhood of the foramen magnum or of the upper cervical vertebræ. In syringomyelia paralyses in the distribution of the accessory nerve are not uncommon.

Paralysis of the Sterno-cleido-mastoid (*spinal accessory nerve*).—The chin is somewhat raised and turned toward the affected side in consequence of the antagonistic contracture of the other sterno-mastoid. Motion in the opposite direction is difficult. In bilateral paralysis of this muscle it is very difficult to turn the head with the chin raised, and it can be done only imperfectly. The head readily falls backward and, when the patient is lying down, it can be lifted only with the greatest difficulty if at all.

Paralysis of the Trapezius (*spinal accessory nerve*).—The shoulder sinks downward and forward so that the supra-clavicular fossa becomes deeper. The median border of the scapula moves outward and is not parallel to the vertebral column, as under normal conditions, but it runs obliquely from below and inward, upward and outward. Voluntary raising of the shoulder, "shrugging the shoulder," is impaired, and it can be done only by the levator scapulæ. The arm sinks down from its own weight, and there is often pain in the shoulder from stretching the ligaments of the joint. If the clavicular portion is paralyzed, the shoulder does not move on deep breathing. Raising the arm above the horizontal is also affected, from the impaired fixation of the scapula.

Paralysis of the Pectoralis Major and Minor (*anterior thoracic nerves*).—Abduction of the upper arm is difficult, the hand can be placed on the shoulder of the healthy side only by the help of the anterior bundle of the deltoid, and the palms can not be clapped together hard when the arms are stretched forward. The function of the pectoral in forcibly drawing down the arm when raised to the perpendicular (as in chopping wood, pulling down a hanging rope, etc.) is very important.

Paralysis of the Rhomboidei (*N. dorsalis scapulæ*) causes the inner edge of the scapula to be somewhat raised from the thorax while its lower angle moves outward. It becomes difficult to approximate the scapula to the vertebræ, and wholly impossible to do so if the trapezius be also paralyzed. The movements of the extended arm inward and backward are also impaired on account of the defective fixation of the scapula. Isolated paralysis of the levator anguli scapulæ causes no noticeable symptoms, but if the trapezius be also paralyzed it becomes wholly impossible to raise the scapula. Then the approximation of the scapula to the vertebral column (rhomboidei) and the raising of the scapula (levator anguli scapulæ) are completely abolished.

Paralysis of the Latissimus Dorsi (*subscapular nerves*).—There is no deformity when at rest, but the arm can not be strongly adducted inward and backward or held firmly to the trunk, and the hand can not be placed on the sacrum. The latissimus, like the pectoralis major, acts in drawing down vigorously the raised arm. Finally, when the latissimus is paralyzed, it affects the throwing back of the shoulders, as in the position of the soldier.

Paralysis of the Rotators of the Humerus Inward and Outward.—Movements of rotation at the shoulder-joint are best tested when the forearm is flexed at a right angle. In paralysis of the inward rotators (especially the subscapular, innervated by the subscapular nerve), the arm, when rotated outward, can not be brought back again to its normal position. All manipulations, too, which the paralyzed arm tries to make on the opposite side of the body, are considerably impaired. In paralysis of the outward rotators—the infra-spinatus, innervated by the supra-scapular nerve, and the teres minor, innervated by the axillary — rotation of the arm outward is abolished. In writing and sewing (using the needle), the paralysis causes very marked disturbance. Paralysis of the teres major (subscapular nerve) causes hardly any noticeable disturbance, since this muscle acts merely as an aid to the latissimus dorsi and pectoralis major.

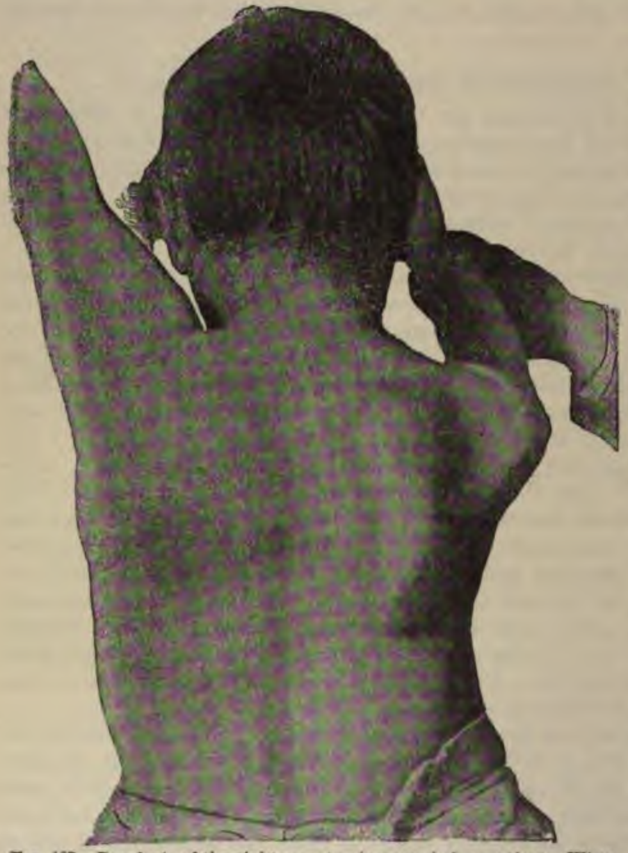

Fig. 107.—Paralysis of the right serratus (personal observation). Wing-like protrusion of the right scapula when the arm is stretched forward.

Paralysis of the Serratus Anticus Major (*paralysis of the long thoracic nerve*).—This paralysis is quite common, and therefore is of practical importance. Its most frequent cause is mechanical injury of the long thoracic nerve, such as may occur especially in porters, field-hands who carry sacks, soldiers in fighting, etc. We also see at times a neuritic paralysis of the serratus, coming on with pain in the shoulder either without any discoverable cause, or as the result of taking cold ("rheumatic serratus paralysis"). Finally, serratus paralysis may follow acute infectious diseases, especially typhoid fever, where it is probably also of neuritic origin. We have seen serratus paralysis after acute articular rheumatism and once after gonorrhœa. Bilateral paralysis of the serratus is seen as one symptom of extensive muscular atrophy, especially in juvenile muscular dystrophy. A congenital absence of both serrati also occurs with the corresponding disturbances of function.

As the arm hangs down quietly, the scapula on the paralyzed side is somewhat raised and stands out a little from the chest-wall, from the action of the antagonists (the traction of the levator anguli scapulæ, the trapezius, and the pectoralis), its lower angle is a little approximated to the vertebral column, and therefore its median edge runs obliquely upward and outward (traction of the rhomboidei). This faulty position of the scapula, however, is in many cases only of slight degree.

If the patient wishes to raise his arm, he can raise it only to the horizontal position, and we fail to see the projection of the tense indentations of the serratus on the lateral wall of the chest; but as soon as we seize the scapula firmly and push it forward—that is, supply the missing action of the serratus—the patient can at once raise the arm. If the arm is raised outward to the horizontal line, the scapula approaches the vertebral column. The somewhat projecting lower angle then comes almost behind the spinous process. The muscular masses of the trapezius and rhomboidei are crowded together and form a swelling which is absent on the healthy side when the arms are in the same position. If the patient moves his arm forward, which has been raised outward, the scapula turns about its vertical axis and makes a wing-like projection almost perpendicular to the surface of the back (*vide* Fig. 107). We can now take the scapula between two fingers and grasp its whole inner surface. The latter forms a deep hollow with the middle part of the back. Serratus paralysis can almost always be recognized at the first glance from this extremely characteristic and peculiar position of the scapula. In some cases the patient can still raise the arm almost completely to the perpendicular in spite of paralysis of the serratus. This is probably done by the help of the middle portion of the trapezius. Disturbances of sensation are often entirely absent, but, on careful testing, we can often find a slight disturbance in the region of the scapula and shoulder in traumatic and neuritic paralysis.

The course of serratus paralysis is usually quite tedious, but, as a rule, in ordinary peripheral paralyses, recovery takes place after several months. Many cases are incurable. Treatment consists chiefly in the application of electricity to the paralyzed nerve and muscles.

5. Paralyses of the Muscles of the Back and Abdomen.

The muscles of the back and abdomen are rarely paralyzed except as a complication of more extensive disease. Paralysis of these muscles is seen most frequently in the different forms of juvenile muscular dystrophy (*q. v.*). If the erector spinæ on both sides is atrophied and paralyzed, the vertebral column when the patient is sitting forms a curve with the convexity backward. The patient supports himself by putting the hands on the thighs, lest the trunk fall forward. When the trunk is bent forward it is difficult or impossible to regain the upright position. On standing, the trunk sinks backward, so that a plummet dropped from the upper thoracic vertebræ would fall behind the sacrum. The anterior portion of the pelvis is raised by the contraction of the abdominal muscles necessary to maintain the equilibrium. If the abdominal muscles are paralyzed, the patient can no longer raise the trunk to an erect position, when lying on his back, without the aid of the arms. On standing erect, the belly is very prominent, the anterior portion of the pelvis is sunk, and the spinal column is bent in marked lordosis. A plummet dropped from the upper thoracic vertebræ would strike about in the middle of the sacrum. The implication of the smaller spinal muscles, not mentioned here, is difficult to determine positively. If the extensor of the back as well as the abdominal muscles are paralyzed, it is no longer possible to keep the trunk erect.

6. Paralyses in the Region of the Upper Extremity

Paralysis of the Deltoid Muscle (*axillary* [*circumflex*] *nerve*).—Deltoid paralysis occurs either as one symptom of complicated paralyses involving the brachial plexus, or as an isolated traumatic and rheumatic paralysis; that is, neuritic, beginning with pains in the region of the shoulder. After acute articular rheumatism (*q. v.*), when the shoulder has been involved, we have also repeatedly seen deltoid paralysis ("arthritic or arthropathic paralysis") with atrophy of the muscles. Finally deltoid paralysis is a common symptom in the most varied spinal and cerebral diseases. The characteristic symptom of deltoid paralysis is the impossibility of raising the upper arm when it is hanging down vertically, but we must remember that the different parts of the deltoid need not always be equally affected. The anterior bundle of the muscle raises the arm obliquely forward and inward, the middle bundle outward, and the posterior bundle backward; the latter also serves chiefly to draw the arm outward and backward when it has been raised forward. The arm is raised forward and outward by the action of the deltoid only up to the horizontal, and it is raised backward only to an angle of about forty-five degrees. To raise the arm completely to the perpendicular the serratus anticus must act (*vide supra*). If the anterior bundle of the deltoid be chiefly paralyzed, the patient can no longer put his hand to his head, raise the arm to the opposite shoulder, etc. If the posterior bundle be paralyzed he can no longer put his hand in his trousers' pocket. In complete paralysis the arm usually hangs down loosely. If the patient, nevertheless, makes a vigorous effort to raise his arm, the shoulder is usually raised and the scapula turned from the contraction of the trapezius and serratus. In a few cases only has it been observed that, where the deltoid is wholly paralyzed, the patient can still raise his arm by the aid of the upper portion of the pectoralis major or of the supra-spinatus and trapezius. It is easy to avoid mistaking anchylosis of the shoulder-joint for deltoid paralysis by making a few passive movements of the arm.

Paralysis of the Biceps and Brachialis Anticus (*musculo-cutaneous nerve*) is only exceptionally an isolated paralysis, but it is quite often seen combined with other paralyses. The forearm, when in supination, can not be flexed, but in pronation the supinator longus can still display its action of flexion. The action of supination by the biceps, which it exerts when the forearm is flexed, is also absent. We can readily recognize this action on ourselves by making a rapid and forcible movement of supination when the forearm is flexed and then feeling the contraction of the biceps with the fingers. We sometimes see at the same time a disturbance of sensibility on the radial side of the forearm from an affection of a cutaneous branch of the musculo-cutaneous nerve.

Radial [Musculo-spiral] Paralysis.—The anatomical course of the radial nerve causes pressure paralysis of this nerve to be among the commonest peripheral paralyses. It is seen especially when the nerve is pressed against the humerus during sleep by the body or head lying on it, in drunkenness, sleeping on a wooden bench, on the hard ground, etc. The paralysis is usually noticed immediately on waking. Other traumatic influences, direct injuries of the nerve, compression in dislocation of the shoulder, in fractures of the humerus, in pressure from crutches, in bandaging or fettering the arm, etc., are also frequent causes of radial paralysis. It is worthy of note that paralysis of the extensor digitorum has been repeatedly seen to follow subcutaneous injections of ether on the extensor side of the forearm. Exposure to cold (rheumatic radial paralysis) plays no very great part in the ætiology of paralysis. Radial paralysis is also rare after acute infectious diseases. For lead paralysis, which is localized chiefly in the distribution of the radial, *vide infra.*

The radial innervates the triceps and the muscles on the extensor side of the forearm. Paralysis of the triceps is present only in the cases where the point of lesion is situated quite high up, as in crutch paralyses, dislocation paralyses, plexus paralyses, etc., but it is absent, or at least it is only faintly manifest, in most of the ordinary pressure paralyses, in which the place where the radial turns about the humerus is the point of compression. Triceps paralysis is readily recognized by the impossibility of extending the forearm, but we must always make the experiment with the upper arm raised, so as to exclude the action of gravity in extending the forearm.

Paralysis of the muscles on the extensor side of the forearm may at once be recognized, since the hand hangs down relaxed in a flexed position (see Fig. 108). Any dorsal extension by the extensor carpi ulnaris and the extensores carpi radialis longus et brevis is impossible, and the lateral movements of the hand in abduction and adduction are rendered difficult. The fingers are flexed, the first phalanx can not be extended by the extensor communis digitorum, extensor indicis, and extensor minimi digiti; but if the first phalanges be extended passively and supported, the extension of the terminal phalanges is perfectly normal, from the action of the interossei which are supplied by the ulnar nerve. The thumb is flexed and adducted, and can neither be abducted nor extended actively (extensores ossis metacarpi et primi et secundi internodii pollicis). If the forearm be extended and pronated it can not be supinated (supinator brevis), but the flexed forearm can be supinated by the biceps. Flexion of the forearm in supination, which is done by the biceps and brachialis anticus, is retained, but flexion when half pronated ("middle position") is weakened, from the paralysis of the supinator longus. If we have the patient make short and rapid movements of flexion of the forearm in this position we do not see the characteristic normal prominence of the tense supinator longus. The very characteristic prominence of this muscle is also wanting if the patient tries to hold his pronated and semiflexed forearm firm against forcible attempts at extension. The supinator longus is only exceptionally spared in radial paralysis.

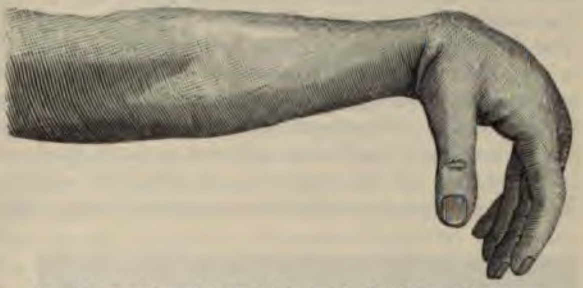

FIG. 108.—Position of the hand in paralysis of the radial nerve. (From SEELIGMÜLLER.)

The functional disturbance of the hand in radial paralysis is very considerable. The action of the flexors is also weakened, since their points of insertion are approximated, on account of the constant drooping of the hand. We often see, too, sensory disturbances, as well as motor, in the distribution of the radial, but these are usually slight. The chief seat is on the radial half of the back of the hand and the dorsal surface of the thumb, and index and middle fingers (compare Fig. 87, page 801). The electrical excitability of the paralyzed parts corresponds to the laws that generally obtain. At the onset, and in mild cases, it is normal; at a later period, in severe cases, there are pronounced atrophy and reaction of degeneration. It is worthy of note that in all forms of radial paralysis, especially in lead paralysis, we very often find a peculiar chronic thickening and swelling of the tendons on the back of the hand, the chief cause of which is probably the mechanical tension of the tendons.

Ulnar Paralysis.—Except from the frequent implication of the muscles supplied by the ulnar nerve in extensive paralyses and atrophies, especially in progressive muscular atrophy, ulnar paralysis arises chiefly from traumatic influences, pressure, wounds, fractures of the humerus, especially fractures of the internal condyle, dislocations of the shoulder-joint, etc. Primary neuritic paralyses or paralyses following acute diseases, such as typhoid fever, are much more rare.

Flexion of the hand, and especially its lateral movement to the ulnar side, is disturbed (flexor carpi ulnaris). Flexion of the last three fingers is imperfect from partial paresis of the flexor profundus digitorum, and the little finger can not be moved at all (hypothenar muscles). Paralysis of the interossei and the last two lumbricales is most striking, by which flexion of the proximal phalanges and extension of the terminal phalanges of the fingers becomes impossible. The defective extension of the terminal phalanges is most plainly seen when we hold the proximal phalanges firmly and ask the patient to extend the fingers fully. The last two fingers remain less extended than the index and middle fingers, whose lumbricales are innervated by the median nerve. Spreading the fingers, and still more bringing them together again, is also much impaired (interossei, lumbricales). The thumb can not be adducted against the metacarpal bone of the index-finger (adductor pollicis) or held firmly in this position.

In almost all old cases of ulnar paralysis a very characteristic position of the hand is developed, besides the muscular atrophy which is especially noticeable in the interosseal furrows of the back of the hand. By the contraction of the muscles antagonistic to the paralyzed interossei (extensor and flexor communis digitorum), the first phalanges are put in marked dorsal extension, but the terminal phalanges are flexed, so that the hand assumes an actual clawing position—"claw-like hand," *main en griffe* (see Fig. 109). A similar position of the fingers occurs as a congenital anomaly (in small children), and is probably due to defective development or complete absence of the interossei.

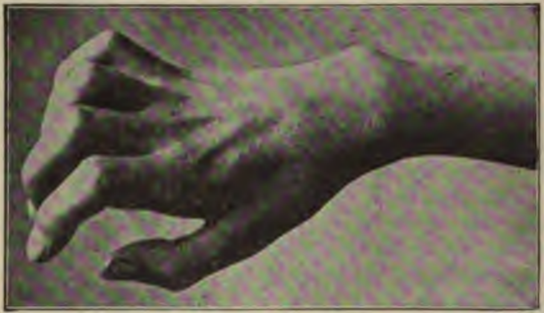

Fig. 109.—Paralysis of the ulnar nerve. Atrophy of the interossei. The terminal phalanges can not be extended. (From the Erlangen Medical Clinique.)

The disturbance of sensibility, if it be present at all, affects the volar surface of the last two fingers, the dorsal surface of the last three fingers, and a portion of the back of the hand (see Figs. 87, 88, and 89). Trophic disturbances of the skin of the fingers are not infrequently seen.

Median Paralysis.—Isolated peripheral median paralysis is seen chiefly as a traumatic paralysis, much more rarely as neuritic paralysis. Disturbances in the muscles innervated by the median are comparatively common as a complication of more extensive paralyses, in cerebral and spinal diseases (progressive muscular atrophy).

The disturbances of motion are very striking. Pronation of the forearm is almost wholly abolished (pronator radii teres and quadratus). The hand can be flexed only toward the ulnar side by the flexor carpi ulnaris (paralysis of the flexor carpi radialis). The terminal phalanges of the fingers can no longer be flexed (flexor sublimis digitorum and a part of the profundus), but flexion of the primary phalanges is normal by means of the interossei. The patient can grasp an object only by the last three fingers, which can still be partly flexed by the flexor profundus digitorum (ulnar nerve). The terminal phalanx of the thumb can no longer be flexed (flexor longus pollicis), and the first phalanx can be flexed only when the thumb is also adducted by the adductor and the inner head of the flexor brevis (ulnar nerve). The thumb can not be opposed at all (touching the terminal phalanx of the little finger with the thumb), since the muscles that act for opposition (opponens, abductor brevis, and outer head of the flexor brevis) are all innervated by the median nerve. The thumb therefore lies with the metacarpus turned backward in extension as a result of the unopposed action of the extensor longus ("ape position").

If there is any disturbance of sensibility, it is found on the volar surface of the thumb and the two adjacent fingers, and also on the dorsal surface of the terminal and middle phalanges of the index and middle fingers, and the radial side of the ring-finger (see Figs. 88 and 89). In severe cases trophic disturbances, vesicles on the fingers, a shining atrophic skin, and changes in the nails are often present.

Combined Paralyses of the Arm Muscles.—Combined paralyses, in which the affected muscles belong to the distribution of several nerves, occur in various forms, especially as a result of injuries which affect the brachial plexus in the neck or shoulder—plexus paralyses. There is either direct injury of the nerves by a blow or wound, or indirect injury from luxation of the humerus, fracture of the head of the humerus and the clavicle, tumors in the supra-clavicular fossa, etc. Plexus paralyses from primary neuritis also occur.

Among the many possible combinations of plexus paralyses, one form, first described by Erb, and since then repeatedly observed, deserves special mention. In this the deltoid, biceps, brachialis anticus, and supinator longus (muscles whose nerves all rise from the roots of the fifth and sixth cervical nerves) are paralyzed at the same time. The same combination of paralysis therefore occurs as if the motor roots of the fifth and sixth cervical nerves were themselves affected. The arm hangs down relaxed, and can not be raised at all; the forearm can not be flexed at all, but the hand and fingers have their normal mobility. The cause of the paralysis must have its seat at the point where the nerve-fibers for the muscles mentioned lie near one another (see Fig. 99). The supinator brevis and infra-spinatus are also not very infrequently paralyzed at the same time, so that the forearm, when pronated and hanging down, can not be supinated, and the humerus, when rotated inward, can not be turned outward. The supraspinatus and subscapularis may also be involved in the paralysis. Sensory disturbances are usually slight, but they may sometimes be found on the outer side of the upper arm and forearm.

This same combination of paralyzed muscles is found in a part of the delivery paralyses first described by Duchenne. These are sometimes seen in infants after hard labor, and are the result of injuries of the brachial plexus in turning, in bringing down the raised arm, in the Prague method, in extracting the child by the shoulders, in forceps delivery, etc. Such obstetrical paralyses may get entirely well in a few weeks or months, but not infrequently permanent atrophic paralyses remain.

Another rare form of plexus paralysis occurs from injury of the fibers coming from the eighth cervical and first dorsal nerves. This leads to paralysis of the smaller muscles of the hand (thenar, interossei) and the flexor muscles on the palmar side of the forearm. Slight disturbances of sensibility are usually present at the same time in the ulnar and median region.

In some cases of complicated paralyses of the brachial plexus, which are usually traumatic (Seeligmüller and others), co-existing symptoms on the part of the sympathetic have been observed, consisting of contraction of the pupil, narrowing of the opening of the lids, and a retraction of the eyeball on the paralyzed side. These symptoms, pointing to a paralysis of sympathetic nerves (*vide infra*, page 905), are probably always due to a lesion of the ramus communicans of the first dorsal nerve, as follows from clinical and experimental investigations (Klumpke). Vaso-motor symptoms in the face are usually absent, but we sometimes find a peculiar flattening of the cheeks, which has not yet been correctly explained.

Occupation Paralyses.—In certain occupations in which certain muscles or groups of muscles or nerves are over-excited or exposed to persistent pressure, paralyses may occur which merit a brief mention. Thus we see not infrequently atrophic paralysis of the muscles of the ball of the thumb in file-cutters, linen-ironers, basket-weavers, locksmiths, joiners, etc., and atrophy of the interossei in cigar-rollers. Bruns has described an isolated paralysis of the flexor pollicis longus as sometimes occurring in drummers as a result of over-exertion. The prognosis of these paralyses, which are often associated with paræsthesiæ and slight sensory disturbances, is not unfavorable, provided the patient can give up his harmful calling.

General Prognosis and Treatment of the Peripheral Paralyses of the Upper Extremity.—In the prognosis of the peripheral paralyses of the arm the same points hold good that have been spoken of in the prognosis of facial paralysis. In these, too, there are mild and severe cases, the latter having complete reaction of degeneration, and a course that lasts at least several months before recovery. A number of traumatic and neuritic paralyses can be cured only up to a certain point, or they may be even entirely incurable.

The treatment can fulfill a causal indication only in comparatively rare cases, when we can succeed in removing by operation any compressing tumors, cicatrices, splinters of bone, formations of callus, etc. When nerves have been cut, suture of the nerve is an extremely important measure, which promotes recovery and which may be the only possible means to secure recovery. In fresh neuritic paralyses the well-known anti-rheumatics (antipyrine, salicylate of soda) are sometimes used, especially if there is also pain.

In other cases the electrical treatment of paralyses promises the best success. We use the galvanic current chiefly, although we generally employ the faradic current at the same time. In regard to the method of application, we may employ the stabile action of the constant current on the very point of the lesion, especially in fresh cases, but the chief method is the electrical irritation of the paralyzed nerves and muscles. We treat the nerve above the point of the lesion in order to act in some degree against the hindrance to conduction from above and to overcome it. The muscles are irritated by galvanism by stroking the cathode over the individual paralyzed muscles. If there is reaction of degeneration, with anodic contractions predominating or exclusively present, we use the anode for the exciting pole. The other pole is placed on the back of the neck or on the seat of the lesion. Faradization of the muscles may also be of service, especially if the muscles react to faradism; but, even if this is not the case, the sensory faradic irritation has perhaps a favorable influence, since it produces a reflex irritation of the motor nerves. The single sittings should last five or ten minutes, and should take place daily or three or four times a week. The fresher the paralysis, the more favorable in general is the prognosis. Of course in such cases it is usually impossible to decide how much of the improvement is to be referred to the treatment and how much to spontaneous recovery, but we can not deny that we sometimes obtain noteworthy success even in old and severe cases by great patience and perseverance. The treatment must in such cases be kept up for months, or years, with occasional interruptions.

Embrocations with spirits and with similar substances must often be prescribed in practice, but they act favorably only when associated with methodical massage of the paralyzed muscles. We sometimes see a certain advantage, too, from local warm bathing, or from the use of the baths in Teplitz, Wiesbaden, Wildbad, etc.

7. Paralysis of the Diaphragm

Isolated paralysis of the diaphragm occurs but rarely, from wounds of the phrenic nerve in the neck, also as primary or secondary neuritic (post-diphtheritic) paralysis, and finally in hysteria. Muscular paresis of the diaphragm seems to develop sometimes as a result of inflammation of the serous layer of the diaphragm. The paralysis of the diaphragm which comes on as one symptom in more extensive paralyses, is more frequent and practically more important. In diseases of the upper cervical cord, in ascending myelitis, or in compression of the cervical cord by vertebral caries or meningeal tumors, in progressive muscular atrophy, in multiple neuritis, etc., the development of paralysis of the diaphragm is the cause of the rapidly fatal termination which follows the appearance of disturbance of respiration. The roots of origin of the phrenic nerve are the third and fourth cervical.

The symptoms of paralysis of the diaphragm are readily recognized, especially in the ordinary bilateral affection. We detect the modification of the respiratory movements at the first glance. While we are struck by the marked upper thoracic respiration, which becomes very labored on the slightest cause, while the visible and palpable protrusion of the epigastrium on inspiration is entirely absent. Instead of this there is usually an inspiratory retraction in the epigastric region. The respiration is but little accelerated in simple paralysis of the diaphragm as long as the patient is perfectly quiet; but in other cases the development of a severe bronchitis, from the defective respiration in the lower lobes of the lungs, causes constant dyspnœa. The explanation of the bronchitis may be found in the fact that the action of abdominal pressure is very much diminished by the constant high position of the diaphragm, which may be made out by percussion, and consequently the cough and the expectoration of secretion are very imperfect.

The prognosis is favorable only in hysterical and rheumatic paralyses; otherwise it is usually very unfavorable. In regard to treatment, the only thing that can be tried is to stimulate the diaphragm from the phrenic in the neck by faradism or galvanism, while the other pole is placed on the region of the insertion of the diaphragm in the thorax. A transverse conduction of the constant current through the diaphragm, associated with changes of the current, may also have a favorable influence.

8. Paralyses in the Region of the Lower Extremity

Paralysis of the Crural Nerve.—Crural paralysis is but rarely isolated. It is seen after injuries, after compression of the nerve by tumors of the pelvis or thigh, in disease of the vertebræ, psoas abscess, etc. There is also a primary neuritis of the crural nerve, and finally paralysis of the quadriceps sometimes follows acute articular rheumatism and other affections involving the knee-joint.

The most important muscles innervated by the crural nerve are the ilio-psoas and the extensor cruris quadriceps. The paralysis of these muscles is readily recognized. The thigh can not be flexed on the trunk, and the trunk can be raised from the recumbent position (ilio-psoas muscle) only with difficulty (by the help of the abdominal muscles, *vide supra*). If the thigh be flexed passively in bed the dependent lower leg can not be extended or raised in the air (extensor cruris quadriceps). In peripheral paralysis of the quadriceps the patellar reflex of course is absent. Walking and standing are very difficult or almost impossible, since the ilio-psoas is necessary for the stride forward and the quadriceps for fixation of the knee-joint. Paralysis of the sartorius and pectineus causes no special symptoms, but it can be recognized by the failure of these muscles to contract when their movements are opposed. If there is any disturbance of sensibility it is found in the lower half of the anterior surface of the thigh and on the inner side of the leg down to the great toe (saphenous nerve, see Figs. 90 and 91).

Paralysis of the Obturator Nerve is very rarely seen as an isolated phenomenon. The chief symptoms are the defective adduction of the thigh (the adductor magnus, longus, and brevis, and the gracilis), and the impossibility of crossing one leg over the other. Rotation of the thigh outward is also disturbed (obturator externus). Some disturbance of sensibility may be found on the inner side of the thigh.

Isolated Paralyses in the Distribution of the Gluteal Nerves are not common, but the muscles supplied by these nerves are often very markedly implicated in more extensive paralytic conditions, especially in muscular dystrophy and multiple neuritis. Paralysis of the glutæus maximus is rendered noticeable by the fact that this muscle extends the thigh on the pelvis. It is therefore especially called into play in going up-stairs, climbing mountains, and rising from a sitting or recumbent position. All these movements are rendered nearly impossible when the muscles are paralyzed. While walking on level ground is only slightly affected, the patient can not step up upon any step, he can get up from a chair only with effort, etc. On rising from a sitting position from the floor the trunk is raised by supporting the arms on the thighs (see the illustration in the chapter on juvenile muscular atrophy). When the patient is in bed we may test the function of the glutæus maximus by holding the flexed thigh tightly with the hand and making the patient extend it. The glutæus medius and glutæus minimus are abductors of the thigh, and also fix the thigh on the pelvis. If they are paralyzed the patient has a very characteristic waddling gait. On account of the predominance of the adductors the feet are then advanced near each other or even one over the other. The glutæus medius is also a rotator of the thigh inward, so that when it is paralyzed the outward rotators (pyriformis, obturator internus and externus, gemelli, and quadratus femoris) exert a predominant action.

Paralyses in the Region of the Sciatic come from traumatic lesions, from continuous pressure on the nerve from working in a squatting position (field work, etc.), from pelvic tumors, and in hard labors (pressure of the forceps, or, with a narrow pelvis, pressure of the presenting head on the nerves). Isolated primary neuritic paralysis in the sciatic region is rare, but we quite frequently see peroneus paralysis as a symptom of more extensive multiple neuritis (alcoholic polyneuritis, neurotic muscular atrophy, etc.). Diabetic neuritis may also be localized in the peroneal nerve. Very marked paralytic symptoms in the region of the sciatic are seen in diseases of the conus terminalis and the cauda equina (lesions of the lumbar vertebræ and sacrum).

Peroneus Paralysis.—Of the two main branches of the sciatic, the peroneus and the tibial, the former is far more frequently affected than the latter. Even in injuries of the sciatic trunk, the peroneus paralysis is usually much more evident than the injury of the tibialis fibers. We can readily understand that the peroneal nerve itself is quite often subject to external injury from its superficial position. The symptoms of peroneus paralysis are very readily recognized. The flaccid drooping of the toe strikes the observer at once. On walking, this becomes very marked, and the tip of the foot often sticks to the floor. The patient, therefore, has to raise the thigh higher, and to put the foot down awkwardly, toe first ("high action," "*steppage*"). Dorsal extension of the foot (tibialis anticus) and of the proximal phalanges of the toes (extensor communis digitorum longus and brevis and extensor hallucis longus), and also abduction of the foot and raising its outer border (the peronei), are almost impossible. In old cases a permanent toe-drop (talipes equinus or varo-equinus) develops, usually as a result of secondary contracture of the muscles of the calf, and this is often associated with a permanent flexed position of the toes from secondary contracture of the interossei.

Paralysis of the Tibial Nerve makes plantar flexion of the foot impossible (gastrocnemius and soleus). The patient can no longer rise on his toes. Adduction of the foot (tibialis posticus) and plantar flexion of the toes (flexor communis digitorum and flexor hallucis longus) are also abolished. As a result of secondary contractures, talipes calcaneus sometimes develops, and also a claw-like position of the toes, with dorsal extension of the first and plantar flexion of the last phalanges from paralysis of the interossei. If the popliteus be also paralyzed, the flexed lower leg can no longer be rotated inward.

In Paralysis of the Trunk of the Sciatic there is sometimes added to the symptoms mentioned the inability to flex the leg backward on the thigh (to be tested when the patient is lying on his side or standing), which is due to a paralysis of the biceps, semi-membranosus, and semi-tendinosus. In unilateral paralysis of the sciatic, walking is still possible, since the leg is fixed at the knee by the quadriceps extensor and is rigid like a wooden leg.

The distribution of the disturbance of sensibility on the posterior surface of the whole leg is given in Fig. 91. Vaso-motor and trophic disturbances, cyanosis, coldness and atrophy of the skin, and changes in the nails are often present. Atrophy and the electrical reactions of the paralyzed muscles occur under the same conditions as in all other peripheral paralyses.

Paralyses from diseases of the cauda equina will be described later in the chapters on the pathology of the spinal cord.

Treatment of all the paralyses last described follows the same rules as are given for the management of peripheral paralyses in the upper extremity.

9. Toxic Paralyses

Lead Paralysis.—Of all the toxic paralyses, that from lead poisoning is practically the most important. It is a common symptom of chronic lead poisoning, and is seen chiefly in those persons whose occupation gives rise to a long-continued absorption of small amounts of lead, especially, therefore, in type-setters, type-cutters, and type-founders; in artists and house-painters, from lead colors; in potters, from lead glaze; in tinkers, file-cutters, etc. Lead paralysis may occur as the only symptom of chronic lead poisoning, or it may be associated in various ways with other symptoms (lead colic, lead encephalopathy, etc.).

The investigations of Leyden, Zunker, Schultze, and others agree in their main conclusions that the anatomical cause of lead paralysis is certainly a degenerative disease and ultimate atrophy of the peripheral motor nerve-fibers belonging to the paralyzed muscles caused by the toxic influence of lead. The degenerative atrophy of the muscles is therefore to be regarded in part as a purely secondary disease, although it is possible that the muscles may also be injured by the lead at the same time with the nerves. The spinal cord, and especially the motor ganglion-cells in the anterior horns of the gray matter, remain, as a rule, unaltered, but undoubtedly, in severe, incurable cases, there may also finally develop a toxic atrophy of these ganglion-cells.

Lead paralysis shows an extremely typical localization in the great majority of cases, and it affects by far the most frequently a part of the radial distribution.

A paralysis of the extensor communis digitorum comes on rapidly or slowly. Extension of the proximal phalanx of the middle and ring, and later of the index and little fingers, becomes impossible, but the extension of the terminal phalanges by the interossei remains normal. There often follows later a paralysis of the extensor and abductor longus pollicis and the extensors of the wrist, and in severe cases a paralysis of the interossei and thenar muscles, while the supinator longus and the triceps almost always remain free in a remarkable way. In much rarer cases lead paralysis affects the deltoid, biceps, brachialis anticus, and supinators. Paralysis of the lower extremities is also very rare. We have seen a very characteristic case in which, besides extensive paralysis of both arms, walking was for some time almost impossible on account of bilateral paralysis of the ilio-psoas and extensor cruris (crural nerve). The patient recovered completely. Other observers have seen peroneus paralysis from lead poisoning [sometimes without noticeable involvement of the arm muscles.—K.]. Paralysis of the laryngeal muscles has also been described.

Lead paralysis is usually bilateral. Ordinarily the right forearm is first affected and the left arm some days or weeks later. In all severe cases a pronounced atrophy and electrical reaction of degeneration develop in the paralyzed muscles. It is an interesting point that changes in the electrical excitability (slow galvanic contractions, etc.) may sometimes be made out in muscles which can be perfectly well moved by the will (see page 850). The sensibility is almost invariably normal, or at most only very slightly altered, so that the sensory nerves are manifestly unaffected by the lead.

Lead paralysis permits a favorable prognosis in the cases where the patient can be removed from the injurious influence of the poison. Recovery takes place after several weeks, or in severe cases after some months. Relapses of paralysis and complications with other morbid results of chronic lead poisoning, however, are, of course, frequent.

The treatment is the same as in all other peripheral paralyses. Electricity is first to be considered. Local sulphur baths and the internal use of iodide of potassium are also recommended.

[In the severer forms of chronic lead poisoning the central nervous system may be affected as well as the peripheral nerves. In the chapter on poisons (*q. v.*) brief reference has been made to some of the other conditions due to lead poisoning. Besides lead colic and lead arthralgia various forms of anæsthesia and paræsthesia may occur. In the forms of lead encephalopathy, besides the general symptoms of headache, mental unrest, and convulsions referred to above, we sometimes meet with symptoms suggestive of a localized lesion, monoplegia or hemiplegia, hemianopsia, amaurosis, etc. In other cases the existence of progressive dementia with tremors, convulsions, etc., may lead to a mistaken diagnosis of general paralysis. In other cases the symptoms are suggestive of combined sclerosis of the spinal cord—ataxic paraplegia (*q. v.*). In cases of doubtful diagnosis it may be of assistance to examine the urine for lead, after iodide of potassium has been given in five- to ten-grain doses thrice daily for a week. The method of making such an examination will be found in the treatises on urinary analysis. If large doses of iodide be given there is some danger that the lead will be liberated too rapidly and that more acute symptoms may ensue. J. J. Putnam has shown that lead is sometimes found in the urine of healthy persons. The pathology of chronic lead poisoning is still undetermined. The cerebral symptoms have been attributed to disseminated foci of encephalitis or to more chronic degenerative processes. Since lead poisoning may cause contracted kidney the possibility of uræmia is also to be considered. Lead has been found in the urine in various cases of chronic degeneration of the spinal cord, and it may act as a cause of such affections, especially of combined scleroses. It is still uncertain whether such degeneration is due directly to the toxic influence of the lead, or to the severe anæmia produced by lead (compare the chapter on pernicious anæmia). In all severe cases of chronic lead poisoning iodide of potassium should be given for a long time to insure the elimination of the lead.—K.]

Arsenical Paralysis.—Arsenical paralysis is rare in chronic arsenical poisoning (colors containing arsenic, hangings, etc.), but it is much commoner after acute poisoning. After the first violent gastro-intestinal symptoms are over, a severe form of nervous disease manifests itself either immediately or two or three weeks later. The chief symptom of this affection is an extensive atrophic paralysis of the arms and legs. The legs are often affected alone, or at least the trouble is much more pronounced in them. The muscles of the trunk may also be involved in the paralysis. In general the rule holds in arsenical paralysis also that the extensor muscles are more affected than the flexors. Electrical reaction of degeneration is found in the atrophied muscles. The accompanying disturbances of sensibility are also very characteristic, either anæsthesia, or especially paræsthesia and severe pains in the sacrum, arms, and legs. The nerve-trunks are sometimes very sensitive to direct pressure. Ataxic disturbances, similar to those in alcoholic neuritis, also occur as well as paralysis. Trophic disturbances in the nails, hair, etc., have been repeatedly observed.

There are only a few investigations in man as to the anatomical cause of arsenical paralysis, but there can hardly be a doubt that the chief disturbance is a severe neuritic change. All the morbid symptoms are in harmony with this (see the chapter on multiple neuritis) as well as the general course of the disease, which in most cases ends favorably. Of course we must not exclude the fact that both in lead and in arsenical paralysis the cells in the anterior horns may also be affected in some severe cases.

Of the other less frequent toxic paralyses we may mention here phosphorus paralysis, copper and zinc paralysis, and mercurial paralysis, which in a few cases has been seen after the therapeutic use of mercury. Polyneuritis must also be considered to be the chief cause of all these paralyses. Of organic compounds, carbonic oxide and bisulphide of carbon may lead to toxic paralyses. Details as to the important alcoholic paralysis will be found in the chapter on multiple neuritis.

CHAPTER III

THE DIFFERENT FORMS OF LOCALIZED SPASM

1. SPASM IN THE MOTOR DISTRIBUTION OF THE TRIGEMINUS

TONIC spasm of the muscles of mastication is called trismus. As an independent disease it is very rare, but it often occurs as one symptom in complicated forms of spasm and other nervous affections, as in tetanus, in the epileptic attack, in hysteria, meningitis, etc. Both jaws are pressed firmly together, and we can feel through the cheeks the hard and tense masseters. In unilateral spasm of the pterygoids the lower jaw is pushed laterally in the opposite direction.

Clonic spasm of the muscles of mastication—masticatory facial spasm—consists of a paroxysmal and constant movement of the lower jaw, almost always in a vertical, but rarely in a horizontal direction. The single movements follow one another usually in a regular, rapid rhythm, and produce an audible chattering of the teeth. The mucous membrane of the mouth and the tongue is often injured.

The cause of these spasms can not always be established. Sometimes they seem to be of reflex origin, as in affections of the lower jaw, the teeth, or even of distant parts. We once saw a case which lasted for a year which was said to have arisen from a severe fright, and which therefore must be regarded as "hysterical," and also a case of clonic spasm in a ten-year-old boy in the masseters and mylo-hyoids of hysterical origin.

The treatment, apart from the treatment of the primary affection, must aim at removing the cause of the disease, if there be one, such as decayed teeth. In other respects electricity is of value in many cases, applied either by passing the constant current through the muscles, by faradizing them, or by using the wire brush. Of internal remedies we may try narcotics, bromide of potassium, atropine, etc.

Artificial feeding is of great importance, if the patient can not take food voluntarily from a persistent trismus. It is best to introduce a small œsophageal tube through the nose into the œsophagus. Rectal feeding is insufficient for a permanent method, but still it is sometimes of service. In some cases a successful attempt has been made to overcome the closure of the jaws gradually by pushing wooden wedges between the teeth.

2. Clonic Facial Spasm

(Mimetic Facial Spasm. Facial Tic. Convulsive Tic)

We know little that is definite as to the ætiology of facial spasm, the most frequent and practically the most important form of isolated spasm. In some cases, perhaps, the disease is to be referred to a lesion of the trunk of the facial, from exposure to cold, aural affections, or disturbances at the base of the skull, (such as aneurism of the vertebral artery pressing on the trunk of the facial, as was seen in one case), or else to a reflex irritation of the nerve as in trigeminal neuralgia, etc. It may be that many cases are not of peripheral but of central origin, from the facial center in the cerebral cortex. The disease may also appear after violent mental excitement. Finally, imitation and the habit of grimacing play a part in many cases, especially in children, that is not to be underestimated (hysterical facial spasm). At any rate we may see from what has been said that we should not judge all cases of mimetic facial spasm in the same way, but it is the most characteristic cases, due apparently to organic causes, which usually we are wholly unable to explain. Repeated observations have established the fact that the disposition to the disease is heightened by a general hereditary neuropathic taint. The tendency to twitch the facial muscles is very often noticed in nervous and irritable persons, but there is an essential difference between this grimacing and the true facial spasm with its lightning-like muscular contractions.

The symptoms of true convulsive tic consist of alternating, short, lightning-like contractions in almost all the muscles supplied by the facial. The disease is usually unilateral, often extending over the whole distribution of the facial, but sometimes confined only to individual parts—partial facial spasm. Sometimes the contractions are almost constant, though varying in intensity, so that the patient involuntarily makes the strangest faces; but they often appear in separate attacks, which last but a short time, and are interrupted by completely free intervals. The attacks arise without any special cause, or they may be excited by talking, voluntary movements, sensory and mental impressions, etc. In some very severe cases the contractions also invade the neighboring territory—the muscles of mastication, the tongue, the muscles of the neck, and even the upper arm. Voluntary motion in the muscles is perfectly normal, except for the disturbing influence of the spasmodic movements. All sensory disturbances are also lacking; there is neither anæsthesia nor pain.

A common partial form of facial spasm, which is entirely or almost entirely isolated, deserves special mention—blepharospasm, or spasm of the eyelids—that is, a tonic or clonic spasm in the orbicularis palpebrarum. The tonic form may be of reflex origin in affections of the eye, but sometimes it is excited from other regions of the trigeminus. It is, as a rule, bilateral, and it may last for days or weeks, sometimes with occasional interruptions. Graefe first noticed that the spasm could sometimes be instantly checked by pressure on the points of exit of the branches of the trigeminus, and often by pressure upon certain parts of the vertebral column or other parts of the body, so that the eyelids "fly up as if by a spring." The author has no doubt that in such cases we usually have to do with conditions of hysterical spasm. Clonic spasm of the lids, nictitating spasm, consists of a spasmodic winking and contraction of the eyes, which is sometimes almost constant. Here, too, we can often make out a reflex origin for the spasm, but frequently we can not find any cause.

Facial spasm, in its severe forms, and especially blepharospasm, is always a troublesome disease for the patient, and causes very much disturbance. The course is often very tedious. Sometimes there are long intervals, as we have seen during pregnancy, and then the spasm begins anew. In not very rare cases the affection becomes habitual and lasts for life.

Treatment is, therefore, usually a difficult and thankless task. The best results may be obtained where we can succeed in removing any reflex cause of the spasm, as in extracting decayed teeth, treating affections of the eye, or, in some cases, resecting the supra-orbital nerve. In applying electricity, our chief attention must be directed to any pressure points, to which we make a stabile application of the anode of the constant current. If there are no pressure points, we put the anode on the trunk of the facial and the different branches of the pes anserinus. In cases of reflex origin Berger obtained good results by applying the anode to the occiput, just under the protuberance, while the cathode was held in the hand—galvanization of the medulla. The single sitting should last five or ten minutes. The faradic current—a slowly "swelling current" *—has also been recommended. Of internal remedies we should first try bromide of potassium, and then arsenic, atropine, curare, or oxide of zinc. The action of these drugs is always very uncertain. A favorable result, however, has been obtained by nerve-stretching, in a part of the cases operated on, at least so far that the ensuing paralysis troubles the patient less than the constant twitching. When the paralysis ceases the twitchings usually begin again, but in some cases the benefit has been permanent. Finally, we must mention that the use of the red-hot iron has sometimes been followed by a decided improvement of the spasm in old cases. We employ cauterization along the cervical vertebræ, on the trunk of the nerve, or on any existing pressure points, by means of Paquelin's thermo-cautery.

In the cases which are not to be regarded as a form of organic spasm, but are to be classed as hysterical, due to bad habits, etc., a methodical training of the will by gymnastic exercises may be attended with the best results.

3. SPASM IN THE REGION OF THE HYPOGLOSSAL NERVE. LINGUAL SPASM

Although the tongue is often implicated in complicated forms of spasm, such as hysterical or epileptic spasms, isolated spasms in it are very rarely seen. They are relatively most common in hysteria. They occur, however, either in a clonic or a tonic form, and then they cause a marked disturbance of speech, or even of respiration, if the spasm draw the tongue backward. In the latter case it may be necessary to use inhalations of chloroform, and to draw the tongue forward by force.

[* A current that begins weak, and is gradually made stronger and then weaker.—K.]

4. Spasms in the Muscles of the Neck

Tonic and clonic spasms in the region of the muscles of the neck are not very frequent, but they appear in very various forms, and are at times a very severe and persistent affection. We can usually discover nothing definite as to the ætiology of this condition. Only in a few cases can we make out coarse anatomical disease of the nervous system or of the cervical vertebræ, rheumatic or other evil factors, reflex influences, etc.; but a general neuropathic constitution certainly plays a great part in the development of these forms of spasm. Although the spasms in the different muscular regions are often combined with one another, we can still distinguish the chief separate forms.

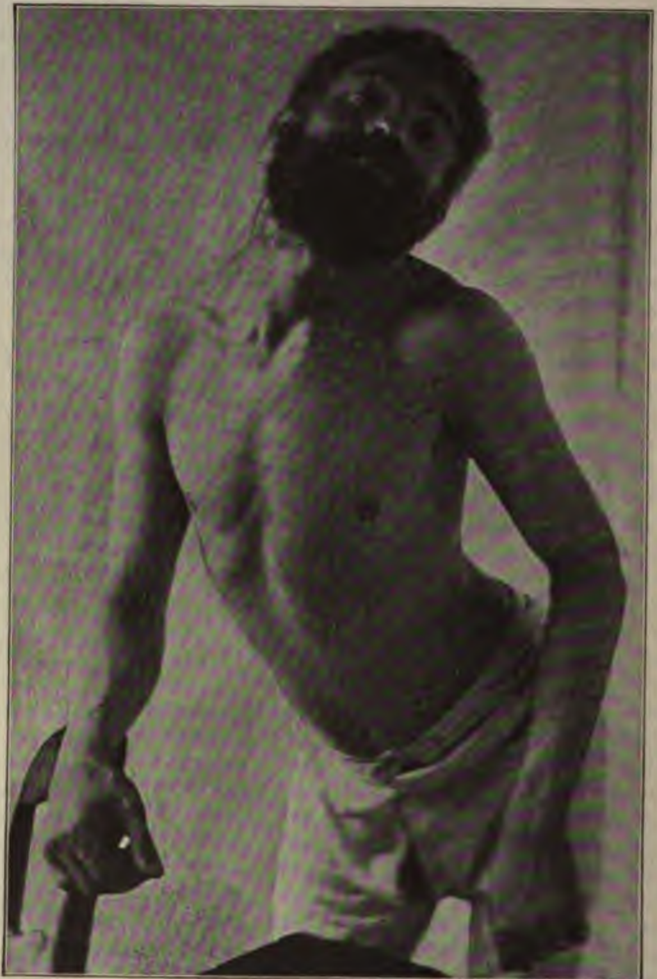

Fig. 110.—Severe case of spasm of the accessory nerve (trapezius and sterno-mastoid muscles). (From the Erlangen Medical Clinique.)

Spasms in the Distribution of the Accessory Nerve.—In clonic spasm of the accessory there are brief or more persistent twitchings of the head, which may attain great severity. Where there is a predominating unilateral spasm of the sterno-mastoid, the head is turned to the opposite side at every contraction of this muscle, and the chin is also somewhat raised. In unilateral spasm of the trapezius, when only the upper portion of the muscle is usually affected, the head is drawn backward toward the affected side against the shoulder. In bilateral and combined spasm of these muscles we see severe shaking and nodding movements of the head—the so-called nodding spasms, "salaam convulsions"—which have been observed chiefly in children. They may also be excited in like manner by contractions of other muscles of the neck (rectus capitis, longus colli). In tonic spasm of the accessory the head is constantly fixed in the abnormal position described above, and it can not be brought back passively to its normal position at all, or only incompletely. The oblique position of the head in unilateral tonic spasm of the sterno-mastoid is called spastic torticollis (*caput obstipum spasticum*). The affection called rheumatic torticollis hardly belongs here, since it is probably due to a rheumatic myositis of the sterno-mastoid.

Tonic and clonic spasm of the splenius (Fig. 111) is also isolated, or combined with spasm of the accessory. In this the head is drawn backward and toward the affected side, and thus we can feel the muscular swelling protruding to the outside of the cervical portion of the trapezius. Spasm of the obliquus capitis is probably the cause of the so-called rotatory tic, in which there are genuine spasmodic rotatory movements of the head.

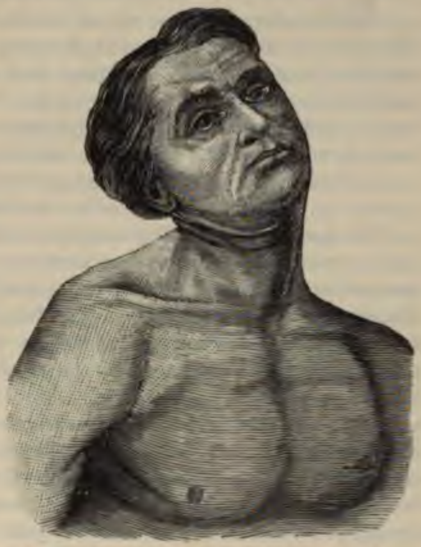
FIG. 111.—Spasm of the right splenius capitis. (From DUCHENNE.)

The course of the forms of spasm described is usually very tedious. There are mild cases which recover in a short time, but, on the other hand, many of these forms of spasm develop into a very distressing, habitual, chronic affection. In these cases any steady occupation, such as reading or writing, is rendered almost impossible by the constantly recurring spasmodic lateral and rotatory movements of the head. Any mental excitement, a feeling that he is noticed, etc., increase the patient's spasms, while they become milder when he is completely at ease. In some cases the twitching ceases entirely when the body is wholly at rest, but it returns at once on any voluntary movement. Combinations of the different forms of spasm occur in the most varied ways. Facial tic may be associated with spasm of the muscles of the neck. In the severest cases the spasm may extend even to the muscles of the upper arm. The occurrence of mental disturbances (melancholia, confusional insanity) has been repeatedly seen. All this favors the theory of the central nature of the disease. The patient's general condition may finally become very poor. Many fall into the morphine habit.

Treatment.—In some cases electricity has brought about recovery, or at least improvement. The method of treatment consists of the application of the anode to the affected nerves and muscles, or of the use of a swelling faradic current, or of the faradic brush to the skin over the affected muscles. Very often we have to change the method, and we must try to find out, by testing, the most efficient way to employ electricity. We may try cautious massage of the affected muscles. We have greater confidence in methodical gymnastic exercises carried out persistently for a long time. Of other remedies, narcotics, such as subcutaneous injections of morphine, are indispensable in severe cases, but we must be very careful in their use. We may also try bromide of potassium, antipyrine, arsenic [belladonna], and other nervines. In severe cases we resort to the use of the red-hot iron to the back of the neck. Other observers, and we ourselves, have seen good results from it, but of course benefit does not always follow. We may also advise nerve-stretching, but it is of doubtful advantage. [Resection of the affected nerves has also been performed in many cases with some alleged benefit. Brissaud and other French observers claim that many of the cases of torticollis are of psychical origin (hysterical?), and that they are cured by appropriate mental treatment.—K.] Finally, it must be mentioned, that we may give great relief to many patients by suitably applied mechanical supports.

5. Spasms in the Muscles of the Shoulder and Arm

Clonic spasms in the upper extremity are probably usually of central origin. They are rarely isolated, as in the pectoralis major, but are more frequently combined with other forms of spasm and other nervous symptoms. They sometimes seem to be of reflex origin, as in the clonic spasms associated with brachial neuralgia, and also the spasms sometimes seen in amputation-stumps. For many of these forms of spasm the cause is still wholly unknown.

Isolated tonic spasms, in single muscles, or groups of muscles, in the upper extremity, have been repeatedly observed. Tonic spasm of the rhomboidei causes an oblique position of the scapula, so that its inner border runs obliquely upward and outward from below and inward. This makes it hard to raise the arm above the horizontal line, as in serratus paralysis, but the separation of the scapula from the chest-wall, which is so very characteristic of the latter, is wanting. Tonic spasm in the levator anguli scapulæ is rare, except in connection with spasm of the rhomboidei or trapezius. In it the shoulder is raised and the head is bent a little to the affected side. Isolated tonic spasms in the pectoralis major, latissimus dorsi, deltoid, etc., are, on the whole, easy to recognize, but they are of very rare occurrence. Tonic flexor spasms of the hand and fingers are more common. We have ourselves seen several such cases, which lasted for months. In one case the spasm could at once be relieved by placing the anode of a moderately strong galvanic current on the median nerve. In another case the flexor spasm of the fingers had followed a mild acute inflammation of the wrist-joint. Brief tonic spasms of the flexors of the fingers and of the thumb muscles are of quite common occurrence. They are seen especially in persons who have to do some severe and persistent bodily work, also in alcoholic subjects, patients with gastric disease or chronic nephritis, in hysteria, etc. Some of these forms of spasm should perhaps be classed as tetany, a special form of spasm which will be fully described later.

We can scarcely give any general rules for prognosis or treatment. It is always important to remove any trouble which may cause the spasm. We may also try, first of all, electricity (a stabile galvanic current, applying the anode to the affected muscles and their nerves) and methodical gymnastics, the latter especially in all cases having any relation to hysteria (*q. v.*).

6. Spasms in the Muscles of the Lower Extremity

Clonic spasms in the muscles of the lower extremity are always, with rare exceptions, a symptom of spinal or cerebral disease. Of the tonic spasms the most frequent and the best known are the painful spasms in the calves, or cramps, which are apt to come on after great muscular exertion, mountain-climbing, or dancing. Many persons have an especially marked predisposition to such cramps, which readily come on, especially after making certain movements or holding the in a certain position. Similar painful cramps sometimes come in other les besides the calves, as in the abductor hallucis, etc. Constantly recurring, sive, and painful spasms in the leg muscles may, as we have seen, form a listressing affection which is hard to remove. In most of these cases there robably conditions of irritation occurring in the muscles themselves. Other spasms in the muscles of the lower extremity are rare, but individual cases lated tonic spasm in the abductors, in the ilio-psoas, in the muscles of the , etc., have been observed. More extensive tonic contractures of the muscles leg are not very rare in hysteria, especially in the hysteria of children.

ltatory Reflex Spasm.—In this place we may mention a peculiar form of , to which Bamberger has given the name of "saltatory reflex spasm." shows itself in the muscles of the lower extremities, never when the patient et in bed, but only when he tries to stand or to walk. As soon as the soles feet touch the floor, such vigorous contractions are set up in the muscles of gs as to force the patient to keep up a constant hopping, jumping, or trip- The heels are also usually raised spasmodically, and in many cases the nt would certainly fall if he were not supported. In the pure forms of ory spasm, on physical examination we can usually make out nothing but traordinary increase of the reflexes, especially of the tendon reflexes, but in cases there may also be other nervous symptoms. According to our present ies there can scarcely be a doubt that the cases described as "saltatory re- pasm" are without exception hysterical.

ertain similar affections exist as endemic neuroses. In the "jumpers" of e the spasms may be produced by any excitement. In other forms the ns are compelled to imitate everything they see or hear. The curious ion observed in Siberia, Java, and elsewhere, known as latah or myriachit, ied to this trouble. Alcoholism, cold, and privation seem to be important ogical factors, and these affections probably have some relation to hys- —K.]

rthrogryposis.—As an appendix we would here consider briefly a remark- lisease, the so-called arthrogryposis, which occurs chiefly in children in the ears of life, and consists of persistent tonic spasms and contractures of one en of all four extremities. The disease usually develops quite acutely, and run its course with fever and rather severe general symptoms. The legs are l either in a position of rigid extension, or they are drawn up spasmodically e body, and can not be extended passively even with violence. The arms exed, and the hands and fingers are also fixed in some position of contracture. nilder cases may recover after a few weeks, but we have seen two cases which l fatally, in which the autopsy gave an absolutely negative result. The e of this quite rare affection is still wholly unknown to us. Several authors of late tried to identify arthrogryposis with tetany (*q. v.*), which we can not t, at least not in all cases. In contrast to tetany we have in arthrogryposis broken tonic rigidity of the extremities lasting for days or even weeks. In d to treatment, prolonged warm baths are especially to be recommended. autious use of galvanism, massage, the bromide salts internally, etc., may e tried.

7. SPASMS IN THE RESPIRATORY MUSCLES

onic spasm of the diaphragm has been seen in a few rare cases. The lower of the thorax is much expanded, the epigastrium is protruded, and the ration, which shows intense dyspnœa, can be performed only by the upper of the thorax. The depression and immobility of the diaphragm can be made n percussion. Many patients have a severe pain in the region of the dia- gm. The condition is not without danger and demands instant interference:

inhalations of chloroform, subcutaneous injections of morphine, a warm bath with a cool shower-bath after it, faradization of the skin in the region of the diaphragm, galvanization of the phrenics, etc.

Clonic Spasm of the Diaphragm.—Singultus.—The well-known "hiccough" or "sob," due to a sudden spasmodic contraction of the diaphragm, associated with a brief inspiratory sound, is in its mild forms a very frequent condition, which soon passes off; but it sometimes increases to a persistent, obstinate, and very troublesome affection, which may last for weeks or months. Hysterical hiccough is the most frequent. This comes on especially after mental excitement, and it may sometimes last, with short interruptions, for weeks or even months. Persistent hiccough may also be excited reflexly in affections of the stomach, intestines, peritoneum, etc. In some cases the hiccough depends upon a direct lesion of the phrenic nerve, as we have seen in one case of tubercular mediastino-pericarditis. We have also seen hiccough lasting for hours in cerebral apoplexy, and in chronic myelitis extending to the cervical cord.

In the milder cases hiccough soon passes off without special treatment. Holding the breath, pressure on the closed glottis, blows on the back, etc., are procedures generally known by the laity, and they are often used to suppress hiccough. In hysterical hiccough intelligent and at the same time energetic mental treatment will often rapidly lead to the voluntary suppression of the spasmodic movements and thus to complete recovery. Such treatment is often aided by the influence (in part merely suggestive) of some internal remedy, such as bromide of potassium, or the use of electricity. In severe hiccough due to organic disease we must sometimes resort to narcotics (large doses of bromide of potassium, opium, morphine, inhalations of chloroform). The constant current to the phrenic nerve or faradization of the region of the diaphragm sometimes acts beneficially.

Complicated respiratory spasms, either in the form of spasmodically accelerated and forced breathing, or spasms combined with all sorts of other symptoms, with many gurgling noises, eructations, etc., are almost exclusively confined to hysteria. We have ourselves counted in one such case over two hundred respirations a minute! The best remedy for most of these forms of spasm, and one which often acts instantly, is a cool bath with energetic cold shower-baths. The yawning spasm (*chasmus, oscedo*), sneezing spasm (*sternutatio convulsiva, ptarmus*), laughing and weeping spasms, coughing spasm, etc., also belong to the respiratory spasms. We once saw an instructive example of the latter in a boy ten years old, undoubtedly due to hysteria. A peculiar, hollow, barking cough came on as a "reflex" (that is, due to association of ideas) either spontaneously or on pinching any part of the skin. The affection lasted for some weeks, and then disappeared quite suddenly.

CHAPTER IV

WRITER'S CRAMP AND ALLIED PROFESSIONAL NEUROSES

WRITER'S cramp (*graphospasm, mogigraphia*) is the commonest form of a whole class of peculiar disturbances of motion, to which Benedikt gave the appropriate name of "professional neuroses of co-ordination." Their characteristic feature is that the disturbance in a certain group of muscles appears only when these muscles come into harmonious action, in some definite and usually some delicate and complicated occupation. Although the persons who suffer from writer's cramp can move and use the muscles of their right arm and hand for ordinary purposes in a perfectly normal manner, these same muscles at once refuse their service when the patient begins to write. In this case the disturbance can not lie in the innervation of the individual muscles themselves, but it must be referred to the form of their associated action—that is, it must be a disturbance of co-ordination. It therefore becomes very probable that the cause of the spasm is to be sought, not in the peripheral parts, but in the centers. We are convinced that writer's cramp and the allied occupation neuroses are due to disturbances of the central (cortical) innervation. Writer's cramp is a morbid condition in writing similar to stuttering in speaking. Over-exertion in writing plays the most important part as an ætiological factor. Writer's cramp is therefore seen chiefly, but of course not exclusively, in those persons whose occupation entails continuous writing, especially in secretaries, merchants, bureau officials, etc. A general nervous predisposition seems also very notably to increase the tendency to writer's cramp. Most cases are associated with pronounced neurasthenic symptoms. Mental factors (fright, anxiety) sometimes play an unmistakable part. Attention has also been called to the fact that bad pens, such as hard steel pens, improper methods of holding the pen in writing, etc., may favor the development of writer's cramp.

Symptoms.—The essential symptom of writer's cramp is the appearance of certain disturbances at every attempt to write, which render writing very difficult or entirely impossible. The affection usually begins gradually, but increases quite rapidly. For the precise characterization of the disturbance Benedikt has distinguished three forms of writer's cramp, but they run into one another in various ways. The spastic form is the most frequent. The patient scarcely begins to write when contractions or tonic spasms come on in the different fingers. The pen is either pressed spasmodically deep into the paper, or, when it is moved, it makes very faulty irregular movements. We very often see a tonic spasm of the pronators of the forearm set in on each attempt to write. Writing is under such circumstances wholly impossible, or it is done only with the greatest exertion, and the characters are also utterly distorted, unequal, and mingled with false strokes and blots. Finally, in the tremulous form of writer's cramp there is such a marked tremor in the right hand at every attempt to write that the letters are completely illegible. We have seen several such cases in children, which we have regarded as distinctly hysterical.

In the paralytic form the disturbance is chiefly a tired feeling in the right arm of rapid onset, like paralysis, and it is often associated with pain. This form of disturbance in writing ought not properly to be classed with writer's cramp, since it is absurd to speak of a " paralytic " form of " cramp."

As has already been said, the motility of the right arm in every other respect is usually perfectly normal, but sometimes analogous symptoms also appear in the same patient in many other fine employments, such as sewing, piano-playing, etc. The sensibility is usually unaffected, except for the muscular pains already mentioned, and a frequent feeling of numbness in the forearm and fingers. A few painful pressure-points have sometimes been found on the cervical and dorsal vertebræ. We should also examine the peripheral nerves, since painful thickenings are sometimes said to be found in them, which possibly stand in a causal relation to the disease. If we are dealing with people who are generally neurotic, as is almost always the case, they often complain at the same time of headaches, mental uneasiness, general weakness, etc. In such cases the degree of the disturbance, as we have said, is always very much influenced by mental conditions (excitement, anxiety), just as is the case in stuttering. We knew a patient who could not write a word, in spite of the greatest effort, if any one were looking at him, but otherwise he wrote a beautiful flowing hand.

The diagnosis of writer's cramp is almost always easy. We must guard against confusing it with other nervous diseases, such as chorea, paralysis agitans, multiple sclerosis, beginning muscular atrophy, or agraphia, which, of course, under some circumstances may lead to disturbances in writing.

The prognosis is always to be given with reserve. Complete recoveries without doubt do occur, but many cases are extremely obstinate and others are incurable. Relapses are also very common even after improvement has set in. Many patients are obliged to choose another calling in consequence of their trouble.

The treatment begins first with the command to give up writing entirely for several weeks. If this command can be obeyed, the mere rest may be of service in mild incipient cases. Certain contrivances for writing, which the patient can best test himself, are often of advantage, such as sticking the pen-holder through a cork, using a large pen-holder, a change in the way of holding the pen and in the position of the arm, etc. Nussbaum has invented a kind of bracelet to which the pen-holder is fastened, and which is held firmly by the outspread fingers. Learning to write with the left hand, which is often tried by patients, usually leads to no good result, since the cramp very soon appears in the left hand also.

Of the special methods of treating writer's cramp the application of galvanism deserves the first mention. Avoiding all strong currents and variations of the current, we apply the stabile anode to the brachial plexus, and also to the different nerves (especially if they be sensitive to pressure) and the affected muscles, for five or ten minutes. The cathode is placed in the vicinity of the cervical vertebræ. If painful points are found, they receive special treatment. We may also use galvanism, as an experiment, through the head. Of late, massage and especially methodical gymnastics have shown still more favorable results than electrical treatment, but the use of them demands special technical skill, and therefore they have so far obtained excellent results chiefly in the hands of certain specialists; but such a methodical re-education in the movements employed in writing should always be tried. We can very rarely promise success from internal remedies such as subcutaneous injections of strychnine, atropine, etc., but those methods of treatment often act favorably which aid the general strength of the nervous system, such as cold-water cures, sea-bathing, and residence at a mountain resort. In writer's cramp, just as in stuttering, we see very plainly the influence of mental factors (rest, increasing or diminishing self-confidence, excitement, etc.) upon the trouble.

As an appendix we will mention here some other professional neuroses that are occasionally seen. They are the piano-player's cramp, which is seen especially in young conservatory pupils, violin- and 'cello-player's cramp, telegrapher's cramp, tailor's cramp, milker's cramp, the peculiar disturbances of innervation in the hands which often occur in cigar-rollers, etc. In the lower extremities there seems to be an analogous affection in ballet-dancers, and also in sewing-machine girls, turners, etc. We have seen professional cramp in the tongue in a clarionet-player. The special points in the symptomatology and treatment of all these forms of cramp are in large part analogous to the conditions described in writer's cramp. In piano-players the neurosis appears chiefly in the paretic form—mild fatigue—and is usually associated with quite severe pains, that come on during playing, in certain parts of the arm. In regard to treatment, the best results are obtained by energetic massage, but general treatment (iron, cold baths, a country residence) is never to be neglected. Finally, it may here be noted that in certain laborious occupations that are persistently practiced, a severe group of nervous symptoms may also arise. For example, Hirt has lately described an affection which occurs in sewing-machine girls, and is characterized by disturbances of sensibility, pain, paræsthesia, and in some cases anæsthesia, ataxia, absence of the tendon reflexes, and swaying with the eyes shut. The disease thus recalls very closely the picture of locomotor ataxia, but it is curable with proper treatment. Therefore Hirt suspected an affection of the peripheral nerves. Similar symptoms are also seen in other classes of laborers, as in farm-hands after laborious toil in the fields.

CHAPTER V

SIMPLE AND MULTIPLE DEGENERATIVE NEURITIS

1. Simple Neuritis

1. **Primary Simple Neuritis.**—In the previous chapters we have become acquainted with a series of morbid conditions which most probably are to be referred to primary inflammatory changes of a particular peripheral nerve. We have seen that many cases of sciatica and of neuralgia in other nerve regions, and probably, too, all the so-called rheumatic peripheral paralyses (rheumatic facial paralysis, deltoid paralysis, etc.), are due to such a neuritis. We have very little information as to the special causes of such forms of neuritis; we may only suspect that in such cases there are definite infectious or toxic substances which act on the affected peripheral nerves. In many cases, especially in most neuralgic affections, it seems rather to be slight inflammatory changes in the nerve sheath and the interstitial connective tissue of the nerve, but in all forms of neuritis leading to motor paralysis it is chiefly a destruction of the nerve-fiber itself caused by the action of the injurious substance ("parenchymatous inflammation," or, better, "degenerative neuritis"). In severe, and especially in acute, cases the parenchymatous and the interstitial changes may develop simultaneously. We will go more fully into the description of the anatomical details in the section on "multiple neuritis" below.

We may merely mention here, as of practical importance, those cases of primary simple neuritis which, after the analogy of facial paralysis which has been fully described, lead to acute peripheral paralysis in other nerve regions. To this class belong, first of all, neuritic axillary paralysis (deltoid), neuritic paralysis in the ulnar, long thoracic (serratus anticus major), crural, peroneus, etc.; neuritic plexus paralyses also occur, especially in the brachial plexus. In many, although of course not in all of these cases, a marked exposure to cold is to be looked upon as the cause of the neuritis ("rheumatic neuritis"). The disease itself usually begins with more or less severe pain and paræsthesia in the neighborhood of the affected nerve, a symptom which is often of decided significance in diagnosis and is usually referred to the involvement of the nerve sheath and interstitial tissue (*nervi nervorum!*), especially when motor nerves are attacked. At the same time with the pain, or soon after it, appear signs of motor weakness in the muscles supplied by the affected nerve. The paralysis may vary extremely in intensity. In mild cases the symptoms soon disappear and recovery ensues. In severer forms electrical reaction of degeneration and atrophy of the paralyzed muscles develop; but even here, as has already been said, we may usually expect a favorable issue. The affected nerves are often very sensitive to pressure. Slight disturbances of cutaneous sensibility are often to be made out on careful examination when there is neuritis of a mixed nerve trunk; but, as a rule, the motor disturbance is almost always much more apparent than the sensory. The treatment follows the methods in vogue in all peripheral paralyses.

2. **Secondary Simple Neuritis.**—There can scarcely be a doubt that secondary neuritis may occur in consequence of disease of neighboring organs. Neuritis

sometimes develops in open wounds which permit the direct entrance of inflammatory agents to the nerve; the inflammation is distributed chiefly in the sheath and the connective tissue of the nerve. It is, however, of rare occurrence—much rarer than we were formerly disposed to assume. Simple mechanical injuries and lacerations of peripheral nerves are much commoner, occurring in all sorts of affections in their neighborhood—fractures, luxations, caries of the bone, tumors, etc. In such cases we should not speak, without qualification, of "inflammation," since we usually have to do, as has been said, with purely mechanical consequences. Apart from the ordinary secondary descending degeneration (*vide supra*, page 849), such mechanical lesions of the nerves have in themselves not the slightest tendency to extend upward in the form of a "*neuritis ascendens*," or "*migrans*," as was formerly believed, and the assumption of an "ascending neuritis," following diseases of internal organs (sexual organs, kidneys, intestines, etc.), and finally leading not infrequently to secondary myelitis, so-called "reflex paralysis," etc., is warranted only in the rarest cases. It is certain, however, that inflammatory processes may sometimes attack neighboring nerve trunks and that an acute inflammation may sometimes extend immediately up the neurilemma of the outgoing nerve to the cerebral and spinal meninges. So far as these processes deserve clinical significance they will receive special mention in the appropriate places.

[It has long been known that, when a nerve has been cut, the portion below the injury rapidly degenerates—Waller's law of degeneration. The portions that degenerate are the axis-cylinder processes that have been separated from the cell-body of the neurone. As a result of more delicate methods of research, it has been found that the rest of the neurone—the cell-body and the axis-cylinder stump—also undergoes slighter changes. There may be, therefore, really an ascending degeneration in the nerve, but it is very slight and has little or no clinical significance.—K.]

Those cases of secondary neuritis which follow other and quite different diseases (usually acute infectious diseases) have quite a different origin. Since in such cases the degeneration often develops in several nerve regions at once, we will return more in detail to this form of secondary neuritis in the next section (see the following pages).

2. Multiple Degenerative Neuritis

Ætiology.—Although the occurrence of extensive primary disease in the peripheral nerves was practically unknown not very long ago, many investigations in the last few decades (Duménil, Joffroy, Leyden, and many others) have taught us that no part of the entire nervous system is so much exposed to all possible harmful influences as the peripheral nerve-fiber. We are at present acquainted with a long list of types of nervous disease which are due exclusively, or at least chiefly, to more or less extensive degeneration of the peripheral nerves. The more we investigate the causes of these diseases, the more we become convinced that it is almost always, in the last instance, chemical poisons which enter the body or are formed in the body itself and exert a destructive influence on the peripheral nerve-fiber. It is indeed possible that all such poisons act from the outset upon the whole peripheral neurone—that is, the ganglion-cell and peripheral nerve-fiber—but the visible injury (degeneration) is often manifested exclusively in the peripheral nerve-fiber, and probably first in the branches most remote from the trophic center, the nerve-cell. Only when the action of the poison is severe and long-continued can the whole neurone—that is, the motor ganglion-cell in the anterior horn of the cord—be visibly injured or even completely degenerated. [Changes may also occur in the peripheral sensory neurones, not only in the peripheral portions of their nerve-fibers, but also in their ganglion-cells in the spinal ganglia and in their axis-cylinder processes in the spinal cord.—K.] The toxic origin of peripheral nerve degeneration is most clearly seen in those morbid conditions which we can refer with certainty to a definite, well-recognized poison. These are the special "toxic neuritides," in the strict sense of the word, with which we have already become acquainted in the description of lead and arsenical paralyses. We have seen that, by the action of these poisons, extensive destruction of the peripheral nerves may ensue, which, of course, is followed by serious clinical symptoms. Disregarding several other rare forms of poisoning (copper, zinc, silver, phosphorus, mercury, etc.), there still remains one extremely important and comparatively common form of poisoning which leads to multiple neuritis, and that is chronic alcoholism. Alcoholic neuritis is by far the commonest form of multiple neuritis, and it will therefore require a special description later.

Besides these neuritides, directly of toxic origin in the strict sense of the word, we find a second large group of cases which come on secondarily in the course of or as a sequel to other diseases. To this class belong, in the first place, most of the cases already mentioned (page 832) of "paralysis after acute disease." Since it is usually an acute infectious disease (diphtheria, typhoid fever, scarlet fever, septic infection, puerperal disease [pneumonia], etc.), we may reasonably assume that it is the toxines formed under the influence of the corresponding agents of infection which lead to the death of the peripheral nerve-fiber; but similar conditions also occur in chronic infectious diseases. Tuberculosis in particular seems to stand in close relation to multiple neuritis, although, of course, it is often hard to exclude complication with some septic infection or the simultaneous action of other intoxications (especially alcohol, *vide infra*). [A more or less extensive neuritis is not uncommon in leprosy. The distribution of the neuritis is less symmetrical than in other forms of neuritis, and the morbid changes in the peripheral nerves, which involve the perineurium and interstitial tissue more than the nerve-fiber itself, are thought to be due to the direct invasion of the nerves by the bacteria rather than to the general toxic effect of the disease. The chief symptoms are marked anæsthesia and muscular wasting. The anæsthesia in the anæsthetic form of leprosy is due to these lesions of the peripheral nerves.—K.] It is certain that post-syphilitic nervous diseases (tabes, *q. v.*) are due, at least in part, to degenerations of the peripheral nerves; but in these cases there is usually extensive disease at the same time in the central nervous system, so that we do not class these conditions as true neuritis. [Syphilitic processes may, of course, involve individual nerves, somewhat in analogy with leprous neuritis, but it is doubtful whether true multiple neuritis is ever of syphilitic origin. Malaria is also said to cause multiple neuritis. Neuritis is more apt to follow the pernicious forms of malaria occurring in the tropics—Jamaica, India, Madagascar, etc. It may come on suddenly during the comatose period of a pernicious attack. In some of the acute cases there have been well-marked changes in the nerve-sheaths, in other cases there have been ordinary degenerative changes. The neuritis has been referred to the toxines produced by the plasmodium. Trophic and vaso-motor symptoms (cyanosis) seem more marked than in other forms of neuritis. There is a possible relation between malarial neuritis and beri-beri (*q. v.*).—K.] Of non-infectious diseases, diabetes mellitus, first of all, is sometimes most closely related to the onset of multiple neuritis. Here, again, we can scarcely help thinking that there is again some abnormal chemical substance which has caused the neuritis, but the question of diabetic neuritis is still in great need of further investigation. It is a fact that neuritic affections of various sorts occur in severe and also in mild forms of diabetes, but of course we may often think, on the other hand, that the glycosuria is only a sequel of the neuritis, or that it has come on at the same time with it (from the same cause, especially previous alcoholism). In a few cases polyneuritis has been seen in severe (usually ulcerative) carcinomata, but in these cases secondary septic processes may play the chief part, as in tuberculosis (*vide supra*).

The third group, finally, is composed of those cases of multiple neuritis which must be termed primary multiple degenerative neuritis. Of course, alcoholic or diabetic neuritis, for example, may come on apparently as a primary disease, but the true primary form of the disease includes only those cases where no other special preceding cause can be discovered, and where the neuritis apparently comes on as an independent disease, due to a special specific cause. The whole course of the disease, which often has an acute febrile character (*vide infra*), shows that we must in such cases also consider toxic-infectious influences, but we know nothing as yet as to the precise form of this infection. We can sometimes find no special cause. There has often been previous exposure to severe cold. Such cases are called "rheumatic multiple neuritis," without, of course, implying that they have any definite connection with articular rheumatism. It is worthy of mention that primary polyneuritis has sometimes been observed to occur in epidemic form. [The peculiar form of epidemic neuritis known as beri-beri will be spoken of later.—K.]

We must add in conclusion that we have very often seen cases of multiple neuritis where we could discover several of the above-mentioned causes at the same time, especially when alcoholism and tuberculosis or alcoholism and diabetes mellitus could unite their noxious effects. We have also seen alcoholic polyneuritis come on directly after some acute febrile disease (pneumonia, etc.), which distinctly reminds us of the appearance of delirium tremens in acute diseases.

Pathological Anatomy.—The anatomical changes of neuritis consist, as has been said, mainly in a degenerative destruction of the nerve-fibers. We do not know whether the toxic agents act directly on the peripheral nerve-fibers or on the peripheral motor neurones in their entirety. The visible coarse anatomical destruction is usually found, at all events, only in the peripheral offshoots of the neurone—that is, in the "peripheral nerves," and apparently it is most marked in the most distant terminal arborizations of the nerves. In the larger nerve-trunks and plexuses the changes are usually much less pronounced than in the smaller nerve-branches.

Only in a few acute cases do the interstitial connective tissue and the nerve-sheath show the changes of true acute inflammation. In such cases we find the affected nerves swollen and decidedly reddened from the great vascular injection; with the naked eye we can sometimes see several or many small hæmorrhages. The microscope shows the nerve-sheath and the interstitial tissue infiltrated with many round cells, but as a rule the interstitial tissue is little or not at all involved in the disease. The pathological anatomical process consists exclusively in a destruction of the axis cylinder and medullary sheath (see Fig. 112). If we stain the diseased nerve-fibers with osmic acid we see the sheath of Schwann filled with many large and small flakes and globules of the medullary substance of the nerve which are stained black. The fatty globules formed by this degeneration are taken up by individual leucocytes (perhaps also by the endothelial cells?), and thus the so-called "fatty granular cells" are formed. If the process has lasted some time and the acute symptoms have ceased, either the regeneration of the nerve-fiber begins, or, when the special nerve-tissue is permanently destroyed, there is a new formation of an abundant interstitial connective tissue. If we now examine a transverse section of a diseased nerve we find the individual nerve-fibers no longer close together, as under normal conditions, but separated from one another in many places, where some fibers have wholly disappeared, by a large amount of interstitial connective tissue. If the new formation of connective tissue attains a higher degree, the nerve then becomes firmer and denser than normal, and, when there is a very large amount of connective tissue (after the analogy of callus formation) it also becomes thicker, so that in some places we may sometimes speak of a neuritis nodosa. If, however, the polyneuritis recovers, it is due to regeneration—that is, to a complete new growth of the destroyed nerve-fiber. The remarkably great power of regeneration of the peripheral nerves is one of the most important facts in the pathology of the nervous system. It is explained in a simple manner by the persistence of the cell of origin (the motor ganglion-cell in the anterior horn) from which the destroyed terminal branches grow out again like the severed branches of a tree. Therefore the severest peripheral paralyses and ataxias may sometimes completely recover.

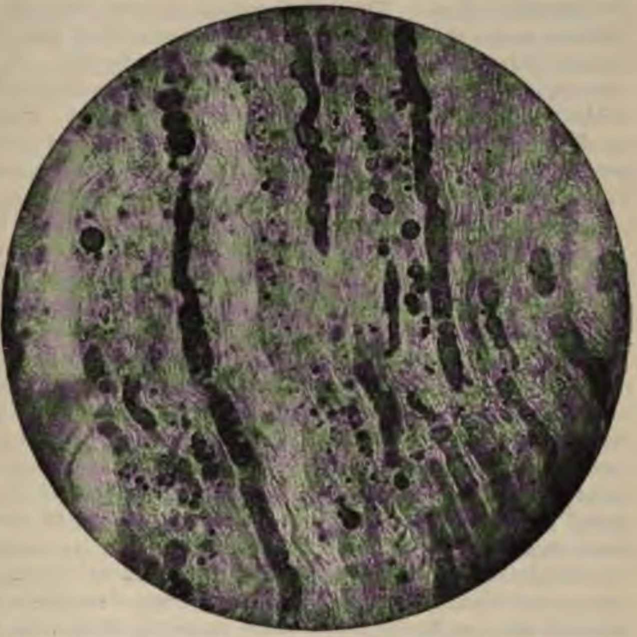

FIG. 112.—Radial nerve in alcoholic polyneuritis. Osmic-acid preparation. (Personal observation.)

In the cases of "chronic multiple neuritis" the disease succeeds an acute neuritis or develops in an insidious fashion from the start. In the latter case the first acute stage of hyperæmia and cell infiltration is wholly wanting and the disappearance of nerve-fibers and the secondary new growth of connective tissue is a chronic process from the first. Such conditions perhaps should not properly be called "neuritis," but "primary chronic degenerative atrophy of the nerves"; but the name is of less consequence than a clear idea of the nature and development of the morbid process. There is also nothing strange in finding in some cases slight changes in the motor ganglion-cells themselves. When the action of the toxic substance is severe or long continued the whole motor neurone, including its cell of origin, suffers. The paralyzed muscles in every peripheral neuritis soon become very much atrophied. Microscopic examination shows all stages of atrophy of the fibers up to complete disappearance. The transverse striation is for the most part preserved, and the number of the muscular nuclei is much increased. In individual cases we also find degenerative destruction of the muscular fibers, hypertrophy, swelling, waxy degeneration, etc. A genuine myositis—that is, the formation of small inflammatory foci in the interstitial tissue—is sometimes joined to the neuritis. In such cases of "neuro-myositis" the poison seems to act from the start simultaneously on the nerves and muscles.

Besides its action on the peripheral motor neurones and the muscles, the action of the morbific influence may extend still farther. Thus we find co-existing changes in the columns of Goll, and even in the brain, which under some circumstances may excite special clinical symptoms.

Symptoms and Course of the Different Forms of Multiple Neuritis.—In order to attain a correct understanding of the symptomatology of multiple neuritis, we must first remember that the disease is not ætiologically one absolutely single affection, and that therefore we must expect *a priori* certain differences in the clinical picture. These differences are due not only to the degeneration of different nerve-trunks, but also, in part, to the fact that the same specific variety of nerve-fiber is not always the one most affected. In general, of course, the rule undoubtedly holds good that the motor nerve-fibers afford the least resistance to all the injurious influences previously mentioned. Therefore peripheral paralyses are the chief and the most common symptom of polyneuritis. The sensory nerves are usually less affected; at least pronounced anæsthesia is exceptional. The involvement of the sensory nerves is shown rather by symptoms of irritation (pain, paræsthesia), which probably are to be referred less to a direct invasion of the sensory fibers than to an irritation of them as a result of the interstitial changes. There is, however, one frequent, important, and very interesting symptom in multiple neuritis, occurring alone or with the paralysis: a genuine motor ataxia. Its origin is not positively explained, but we regard it as most probable that the ataxia is due to disease of the centripetal fibers which also exist in the peripheral nerves, whose function is to regulate (in a certain sense reflexly) and to co-ordinate complicated voluntary movements. It is itself an extremely interesting fact that ataxia of movement without co-existing paralysis may occur in apparently purely peripheral diseases without any discoverable changes in the spinal cord; but ataxia may also be due to disease of the spinal processes of the spinal ganglion-cells—that is, to degeneration in the columns of Goll. From a symptomatological standpoint we must therefore distinguish two forms of neuritis —a paralytic form (the usual form) and an ataxic form (so-called acute curable ataxia, and also, especially, the pseudo-tabes of alcoholic subjects); but combinations of the two forms occur, since paralytic conditions and disturbances of co-ordination may be united.

Passing now to the description of the general aspect and course of polyneuritis, it seems best to keep separate the different forms which may be distinguished either clinically or ætiologically.

1. Primary Acute and Chronic Multiple Neuritis

The disease usually begins acutely, sometimes almost in an apoplectiform manner, and without any definite occasion, precisely like an acute infectious disease. Febrile symptoms, with temperatures from 102°–104° (39°–40° C.), come on in persons previously in good health, usually in adults before or during middle life, with severe general disturbance, loss of appetite, dullness, headache, and sometimes even mild delirium. In these acute cases albuminuria and a slight enlargement of the spleen have sometimes been observed, which symptoms also point toward the infectious nature of the disease. In other cases the initial general symptoms are much less severe, or they may be entirely wanting. The pains, which are hardly ever absent, are very characteristic. They are described as pulling and tearing, are felt chiefly in the loins and the extremities, and sometimes follow approximately the course of the large nerve-trunks. Since in some cases a number of the joints are swollen, the disease at first may be mistaken for acute articular rheumatism. The first symptoms of paralysis, usually in the lower extremities, appear very soon after these initial symptoms, or at the same time with them. The patient notices that he can not readily move one leg, and soon after he notices the same of the other. The paralysis may remain confined to the legs, but more frequently it extends to one or both arms. In regard to its distribution, the forearms and lower legs are generally more affected than the upper arms and thighs, and the extensors (radial and peroneal region) are also more affected than the flexors; but other varieties of distribution may occur, for instance, affection of the glutæi and thigh muscles, etc. If we examine the paralyzed parts more carefully we find a perfectly atonic and more or less extensive paralysis. The reflexes are almost always diminished, the tendon reflexes are usually entirely absent, and the cutaneous reflexes are weak, or they also have almost wholly disappeared. In only a few cases are the reflexes increased—a symptom which is to be regarded as analogous to cutaneous hyperæsthesia. We can usually make out, after a few days, a decided diminution of electrical excitability in the affected nerves and muscles, which soon becomes a pronounced reaction of degeneration. If the paralysis is of longer duration, there is a decided atrophy of the muscles. In these cases the severe initial symptoms of sensory irritation, as a rule, rapidly disappear, although slight pains, paræsthesia, and especially a considerable sensitiveness of the paralyzed parts to pressure and to passive motion, often last for a long time. In many acute cases the hyperæsthesia of the skin and of the deeper parts reaches a very high degree, but it is remarkable that the objective disturbances of sensibility are very slight in the great majority of cases; although on careful testing we can often find slight diminution of sensibility for touch or temperature. Other observers and we ourselves have also repeatedly seen the symptom of so-called delayed conduction—that is, delayed sensation of pain (*vide supra*, page 793). We usually find no disturbances in the distribution of the cerebral and bulbar nerves. An affection of the optic nerve has been mentioned in only a few cases. Facial paralysis and, much less frequently, paralysis of the tongue or of the ocular muscles may also occur. We have ourselves repeatedly seen total bilateral facial paralysis. We may mention as a rare complication acute mental disturbances (confusion, anxiety, weakness of memory, *vide infra*). Details of this very peculiar "polyneuritic psychosis" will be given below in the description of alcoholic polyneuritis. The marked increase in the frequency of the pulse, which is usually present, is important, and probably depends upon a disturbance of the vagus. Trophic disturbances in the skin, hair, and nails are not very rare. Profuse sweating sometimes occurs. Œdematous swelling of the affected extremities has also been repeatedly observed. The functions of the bladder and rectum, however, almost always remain unaltered, but in some cases slight disturbances of the bladder may of course be discovered.

In regard to the course of the disease, in the severest cases it may soon terminate fatally, almost always because the paralysis extends to the muscles of respiration. The inspirations are labored, and are performed with the upper part of the thorax only, while the epigastrium is motionless, or sinks in on inspiration from the paralysis of the diaphragm. There is also paralysis of the other muscles of respiration, the abdominal muscles, etc., so that, after the disease has lasted a week or ten days, death ensues with all the signs of respiratory insufficiency. Such cases have been repeatedly described in the past under the title of acute ascending paralysis (Landry's paralysis, *q. v.*). A second class of cases also begins quite acutely, but then takes a chronic course. The initial acute febrile symptoms cease after a few days, although the paralysis continues to develop to some extent. Then the affection seems to come to a stand-still, and the first signs of improvement gradually begin to appear. Since there is a more or less pronounced atrophy of the muscles in these cases, it always takes considerable time—usually several months—for recovery; but it is often astonishing to see how the most extensive paralysis may finally get entirely well. A third class of cases follows a chronic course from the outset, although even in these cases there may be more acute exacerbations of the disease. In these cases quite extensive atrophic paralysis gradually develops in the lower extremities, and usually in the upper extremities also. The reflexes disappear; the sensibility is as a rule somewhat, but very rarely much diminished. Pains are always present at first, but later on in the disease they often become subordinate. The bladder and rectum usually remain normal in their functions or show only slight disturbances. If the disease advance gradually it may terminate fatally even at a late period, after a course of months, usually from a final paralysis of respiration; but, on the other hand, even after a protracted course, the disease may come to a stand-still, and even progress to a complete or at least to a partial recovery.

The ***diagnosis*** of multiple neuritis is, as a rule, easy for one who is acquainted with the disease and notes the different symptoms carefully. In regard to diagnosis, the chief importance should be placed on the generally acute beginning, with pronounced symptoms of sensory irritation, with frequently a very considerable sensitiveness of the nerves and muscles to pressure and general cutaneous hyperæsthesia; and also on the appearance of a rapidly extending paralysis, whose peripheral nature may be attested by the presence of reaction of degeneration, muscular atrophy, and the absence of the cutaneous and tendon reflexes. Such a paralysis can be produced by nothing but an affection of the peripheral nerves or poliomyelitis (*vide infra*). This latter disease was, in fact, formerly confused with multiple neuritis, but a careful attention to the initial symptoms, especially to the pain and disturbances of sensation, usually makes the differential diagnosis possible.

The prognosis of multiple neuritis is doubtful, as is shown by the description of the course of the disease, but it is by no means very unfavorable. If the patients gets through the first acute stage of the disease without accident, we may hope for recovery, or at least actual improvement, even with extensive paralysis. Such striking results in the way of recovery, after paralysis that has lasted for months, are also important in regard to diagnosis, since such extensive processes of regeneration are possible in affections of the peripheral nerves, but scarcely in spinal diseases; and hence they are sometimes an additional confirmation of the diagnosis of a neuritis.

Treatment.—In the first stage of the disease, especially if severe pains, swelling of the joints, or high fever be present, it is advisable to try the exhibition of salicylic acid, from which several observers have seen a favorable effect. We give ten grains (gramme 0.5) of the acid every hour, or a few larger doses of salicylate of sodium, a drachm to a drachm and a half (gramme 4–6). Instead of salicylic acid we have used antipyrine and phenacetine with good results. When the pain is very severe we must use narcotics, such as injections of morphine. Embrocations of chloroform, and sometimes protracted warm baths, have also a palliative effect. In the further course of the disease proper care, a suitable position for the limbs, and diet—nourishing food—are the main things for the patient. The regenerative processes of recovery begin spontaneously, if they begin at all, but we may hasten recovery and make it complete by a subsequent electrical treatment, especially galvanism. For the completion of the recovery, bathing (simple warm baths, salt baths, etc.) is serviceable, and also the baths at Teplitz, Wiesbaden, Nauheim, and Oeynhausen, and the cautious use of massage.

2. THE ATAXIC FORM OF MULTIPLE NEURITIS

("*Acute Curable Ataxia*")

We can not make a sharp division between the ataxic and the paralytic forms of multiple neuritis. Both conditions, paralysis and ataxia, may occur in the same patient. It is important, however, to know that almost pure ataxia (without paralysis) may develop acutely, and that it is undoubtedly to be referred to degeneration of the peripheral nerves.

Under the ataxic form of polyneuritis must be classed, first, the cases of ataxia repeatedly observed after various acute infectious diseases, especially ataxia after diphtheria (*q. v.*), and also after typhoid fever, small-pox, septic affections, dysentery, etc.; but there is also a primary acute ataxia which comes on without any discoverable cause or after exposure to severe cold in previously healthy persons. Severe general initial symptoms are, as a rule, not present, and the pain is not very great. The ataxia usually develops in the legs, more rarely in the arms. There may be slight disturbances of sensibility at the same time, but not necessarily. The patellar reflex is usually but not always absent. As a rule, with proper care and nursing the patient recovers completely in a few weeks. The diagnosis of this primary acute ataxic form of polyneuritis is usually not difficult. The disturbance of the patient's gait may at first arouse the suspicion of tabes, but the rapid onset, the perfectly normal pupillary reflexes, and usually also the normal micturition, oppose it. We often find in some muscular regions a true paresis besides the ataxia, which is not like the conditions in tabes. We once saw, for instance, a severe ataxia of the legs associated with double facial paralysis. In any doubtful case a favorable termination in recovery forms the final criterion in favor of polyneuritis.

3. The Acute and Chronic Neuritis of Alcoholic Subjects—Alcoholic Paralysis

(*Pseudo-tabes* [*Ataxia*] *of Alcoholic Subjects*)

It has long been known that peculiar nervous affections often occur in alcoholic subjects (M. Huss, Leudet, and others); but formerly a disease of the spinal cord was assumed to be the cause of the symptoms, and only of late have we obtained the knowledge that the greatest part of the cases of this class are to be classed as acute and chronic multiple neuritis (Lancereaux, Moeli, and others). The practical importance of this alcoholic neuritis is not slight; first, because it may easily be confused with other nervous diseases, especially with myelitis and tabes, and, second, because its proper and timely diagnosis is of great significance in regard to treatment. Alcoholic neuritis, furthermore, is by no means rare, being certainly by far the commonest of all forms of polyneuritis. The more we consider the significance of this factor the more all other causes are thrown into the background. Not infrequently, of course, other causes must be considered at the same time with alcoholism; we see, for example, polyneuritis very often in alcoholic subjects who are also tuberculous, and sometimes in the combination of alcoholism and diabetes, etc.; but in all such cases chronic alcohol poisoning probably plays the chief part. Alcoholic neuritis occurs in drinkers of spirits, and also, as we have often noticed, in habitually excessive beer drinkers. It is alleged to be less frequent in drinkers of wine.

Alcoholic polyneuritis, like the other varieties of multiple neuritis, occurs in different forms. According to its course we may distinguish acute and chronic cases, and according to its symptoms a paralytic and an ataxic form, but there are no strict boundaries between these forms.

Alcoholic paralysis begins quite suddenly or more gradually. Motor weakness develops, having its chief seat usually in the legs, and thus rendering walking almost wholly impossible. This weakness generally begins with quite severe tearing and drawing pains and considerable tenderness on pressure over the affected muscles. Most writers agree that the muscles on the anterior side of the lower leg (peronei, tibialis anticus) are very often affected. We can agree with this for many cases, but by no means for all. We have often seen at the onset the greatest trouble in the muscles of the thigh (crural region), and especially in the glutæi. Such patients can still walk with effort on level ground, but they can not go up a step or rise from a chair. With greater paralysis the patient is absolutely bedridden. The muscles of the legs are flabby, they become more or less atrophied, their electrical excitability diminishes, there is sometimes pronounced reaction of degeneration (especially the middle form), and the patellar reflex and other tendon reflexes are usually absent, although there are some remarkable exceptions to this rule. The bladder and rectum are entirely normal or show only slight disturbances. The sensibility of the legs is rarely perfectly normal on careful testing, but the sensory disturbances, except for pain and paræsthesia, are overshadowed by the paralysis. We often find a slight blunting of sensibility for touch and pain, and very often the so-called delayed sensation of pain (*vide supra*, page 793). The skin reflexes are diminished.

In many cases the paralysis extends to the arms also, and chiefly in the radial distribution, so that both hands hang lax in volar flexion, and they and the fingers can be extended but little. The combination of double radial and peroneal paralysis has been often mentioned as characteristic of alcoholic paralysis. Even in patients who can still extend their hands and fingers well we often find that the strength of the movement is diminished. In severe polyneuritis the other arm muscles may also be affected. Muscular atrophy, electrical reactions, sensibility, and the reflexes are the same as in the legs.

The facial muscles are rarely affected, but they have been in a few cases. In some very acute and severe cases of polyneuritis there is also paralysis of the ocular muscles, so that the eye may become completely immovable; but in the few cases that have come to autopsy there seems usually to have been no neuritis of the oculo-motor nerves themselves, but many small hæmorrhages in the neighborhood of the third ventricle—that is, in the region of the oculo-motor nuclei (so-called polio-encephalitis hæmorrhagica superior, *q. v.*).

Mental disturbances ("polyneuritic psychoses") have been frequently observed in alcoholic polyneuritis. They often differ little from delirium tremens, but in other cases a very peculiar type of disease develops. The patient is confused, mixes up persons and places, is very restless, especially at night, apparently has hallucinations, and is consequently excited and anxious, but in the intervals is happy and contented. The most striking disturbance is an extraordinary weakness of memory for all recent events. Half an hour after eating the patient no longer knows that he has eaten. If we show him an object or utter a word or a short sentence to him, he forgets it completely in a few minutes. The more carefully we test the patient in regard to this, the more frequently shall we discover this remarkable disturbance of memory; but the recollection of the distant past and of things learned before the trouble is not at all or very slightly affected. The patient's initial confusion is chiefly the direct result of this actual failure of memory. The severe mental disturbances sometimes last only a few days, but the disturbances of memory may often be noticed for weeks, when they gradually disappear.

The ataxic form of alcoholic polyneuritis may also come on acutely, so that the patient's sudden inability to walk is due to a genuine ataxia of the legs. The ataxia, of course, is generally associated with manifest paralysis, at least in some muscular territories (glutæi, peronei, radials). The ataxia may often be masked by a greater paralysis, so that we can discover it only by careful examination. The patellar reflex is usually, but not invariably, lost; the sensibility is only slightly affected, apart from the pain and paræsthesia. The functions of the bladder remain wholly, or almost wholly, normal.

The chronic form of alcoholic ataxia, the special pseudo-tabes of alcoholic subjects, is more important because it is often harder to diagnosticate. In this form a type of disease develops comparatively slowly, which may closely resemble genuine tabes: ataxic gait, lancinating pains, sometimes even a sort of girdle sensation, absence of patellar reflex, slight disturbance of the bladder, definite disturbances of sensibility, etc. The disease resembles true tabes still more closely if temporary double vision occurs. In such cases we can avoid mistaking it for true tabes only by careful examination. The most important mark of distinction is afforded by the pupils, which almost always preserve their normal reaction in pseudo-tabes. We may also note that in pseudo-tabes the disturbances of the bladder are usually slight, and they may be entirely absent; that the muscles and nerves are tender on direct pressure, and that from the first we can usually find besides the ataxia a slight genuine paresis, which in some muscles (*vide supra*) may be marked. [We rarely if ever find in pseudo-tabes the anæsthesia or analgesia over the lower part of the chest, on one or both sides, in the area of the third to the seventh dorsal roots, so characteristic of true tabes.—K.] Finally, of course, the causal factor, pronounced chronic alcoholism, is an essential consideration. It is worthy of note that in alcoholic pseudo-tabes the anatomical lesion is probably not often limited to the peripheral nerves, but also involves the spinal processes of the spinal ganglion cells—that is, the region of the columns of Goll.

The course of this pseudo-tabes may extend over several years, but the prognosis is far more favorable than that of true tabes; decided improvement and even, in mild cases, complete recovery may occur; but there are also severe incurable forms, since dangerous complications (tuberculosis, etc.) often set in.

All the more acute forms of alcoholic neuritis afford quite a good prognosis, provided the abuse of alcohol can be permanently checked. We may then often see complete recovery in a few weeks or months. On the other hand, relapses are common, and these usually have a tedious course.

The first task in the treatment of alcoholic neuritis is to insist upon the absolute abandonment of the use of alcohol. If this be done, the milder cases usually get well of themselves. The favorable course is aided by electrical (galvanic) treatment and tepid salt baths. Internally we prefer strychnine preparations (pills of nitrate of strychnine or extract of nux vomica).

[4. BERI-BERI—ENDEMIC MULTIPLE NEURITIS

The peculiar disease of endemic origin, long known in India and Japan as "*beri-beri*" or "*kak-ke*," has been found by Scheube to be in its clinical and anatomical relations a well-characterized multiple neuritis. Cases are occasionally seen in seaport towns in sailors arriving from Asiatic ports, and Putnam has described cases occurring in fishermen returning from the Newfoundland banks. Independent outbreaks of the disease have also occurred among the insane in asylums in Alabama, Arkansas, Dublin, and elsewhere. Men are more frequently affected than women, and colored races more than the white. The cause of the disease is still obscure. Pekelharing and Winkler attributed it to a specific staphylococcus, but their observations have not been confirmed by later observers. Insufficient or improper diet probably increases the susceptibility to the disease, but it is doubtful if an exclusive diet of rice or fish, as was once believed, can actually cause beri-beri. Bondurant in Alabama found a close connection between the outbreak of the disease and the use of a polluted water-supply. In other cases there seems some association with malarial poisoning. Overcrowding and foul air favor its development. The disease may begin suddenly or insidiously, and it is often ushered in by moderate fever and gastro-intestinal symptoms: anorexia, vomiting, abdominal pain, and diarrhœa. Two forms of the disease—a "wet" and a "dry"—are usually recognized. The symptoms in the "dry" form are similar to those of ordinary multiple neuritis: pain, paræsthesia, tenderness, loss of power with degenerative muscular atrophy, especially in the extensors of the foot, slight disturbances of sensation, and diminution or loss of patellar reflex. The pass in good condition ("neuro-fibroma"). Hence it happens, as we have seen ourselves in one case, that, in spite of an extensive formation of neuro-fibromata, there are no nervous symptoms at all, no pain, no anæsthesia, no paralysis; but other cases have also been reported in which the tumors caused manifest and even severe symptoms.

Of other new growths on the nerves we may mention sarcoma. This also is often multiple, and then it may lead to very complicated types of disease. The so-called *tubercula dolorosa* deserve special mention. By this term we mean little nodules which may be felt beneath the skin, usually readily movable and very sensitive to pressure. They are not very rare, and are usually associated with drawing pains, which are rarely decidedly neuralgic and are not very strictly localized. They are situated in the extremities, especially in the arms, and in the back, the neck, etc. It is worthy of note that the symptoms are only at times very prominent, and then they disappear again, and that with this disappearance is certainly sometimes associated a spontaneous disappearance of the nodule. The anatomical nature of the tubercula dolorosa is not always to be established with certainty. Many of them are true neuromata, but others belong to other kinds of new growths.

The only successful treatment of neuromata is extirpation, which is to be undertaken only when the symptoms are very severe. If extirpation be not practicable, or if we have to do with multiple neuromata, the patient's trouble can be alleviated by symptomatic means, narcotics, and electricity. If we can compress the nerve above the neuroma, we can often cause by this means a temporary cessation of the pain.

II.—VASO-MOTOR AND TROPHIC NEUROSES

CHAPTER I

GENERAL PRELIMINARY REMARKS

(Erythromelalgia. Acute Angioneurotic Œdema. Myxœdema. Acromegaly. Hydrops Articulorum Intermittens. Injuries of the Cervical Sympathetic)

General Consideration of Vaso-motor Disturbances.—Physiology, as is well known, distinguishes two varieties of vaso-motor nerves—the vaso-constrictors and the vaso-dilators; but since experiments have detected the latter variety in only a few places—for example, in the chorda tympani, the nervi erigentes, and the sciatic—they have not acquired a very great significance in human pathology. We are at present much more disposed to refer every abnormal constriction of the vessels to an excitation, and every abnormal dilatation of the vessels to a paralysis of the vaso-constrictor nerves, although perhaps pathological conditions of excitation of the vaso-dilators may not be at all rare. In regard to the precise anatomical course of the vaso-motor nerves, we must first state that vaso-motor excitations may certainly proceed from the cerebrum, as is shown by the well-known symptoms of blushing and pallor from mental emotions. In experiments on dogs, Eulenburg and Landois have succeeded in producing a fall of temperature on the opposite side by irritating certain portions of the cortex in the immediate vicinity of the motor centers, and by extirpation of the same parts they have produced a rise in temperature. Furthermore, we know with certainty that there is an important vaso-motor center in the medulla oblongata (in the region of the upper olivary body in rabbits), the excitation of which, directly or reflexly, is followed by an almost universal vascular constriction, and its destruction by an almost universal vascular dilatation. We must probably seek the further course of the vaso-motor nerves very largely (or exclusively?) in the lateral columns, from which they pass out chiefly by the anterior roots. It is not known with certainty whether there is any decussation of the vaso-motor fibers, or, if there is, where it occurs. The larger part of the vaso-motor nerves collect, at any rate, in the principal trunks of the sympathetic, from which, as is well known, the separate plexuses that surround the vessels arise. It is not improbable, however, that there is also in part a direct passage of vaso-motor fibers from the cord into the peripheral nerves. In conclusion, we must mention that, according to Goltz's experiments, there are reflex vaso-motor centers in the cord for the different parts of the body.

The clinical vaso-motor symptoms are chiefly to be observed in the external skin. We distinguish them as follows:

1. *Symptoms of Vaso-motor Paralysis.*—We conclude that there is a paralysis of the vaso-motors if there is an abnormal redness of the skin. Such a redness is almost always associated with an objective and often a subjective feeling of an increase of temperature. Such conditions are observed either in connection with other nervous symptoms—as in fresh spinal or cerebral paralyses, and also very often in certain functional neuroses, such as hysteria and neurasthenia—or in the form of independent affections—the pure vaso-motor neuroses, injuries of the cervical sympathetic, etc. Of course in these cases it is usually hard to de-

cide whether there is really a vaso-motor paralysis or irritation of the vaso-dilators. There are cases in which the only symptoms are a persistent or paroysmal diffuse redness of the skin, especially of the head, associated with a great feeling of heat, with palpitation of the heart, strong pulsation of the arteries, anxiety, ringing in the ears, and sweating.

2. *Symptoms of Vaso-motor Spasm.*—Spasm of the small vessels becomes apparent by a striking pallor and coolness of the skin. There is often, with this, a decided feeling of formication and stiffness in the affected parts, which may even increase to an actual feeling of pain. Such vaso-motor spasms affect the hands especially, and form a chronic trouble that is not very rare. They are usually seen in people who are generally nervous and irritable, and sometimes also in washerwomen. A vascular spasm is sometimes seen in the extremities as one symptom of complicated paroxysms, such as nervous angina pectoris (*q. v.*), especially at the beginning of the paroxysm. A persistent spasm of the small arteries may give rise to considerable subsequent trophic disturbance. At least, the rare cases of so-called "spontaneous symmetrical gangrene" (*q. v.*), and also certain forms of scleroderma and some similar affections, are referred by many observers to a primary spasm of the vessels.

General Consideration of Trophic Disturbances.—We have much less information concerning the trophic nerves than we have concerning the vaso-motor. As is well known, the controversy is still going on as to whether we have any right to assume the existence of special trophic nerves. The facts of clinical neurological observation frequently seem to compel the hypothesis of special trophic functions, although, of course, it is usually very hard to determine this point, because we can seldom wholly exclude the indirect influence of sensory and circulatory influences (*vide supra*, anæsthesia of the trigeminus).

Among those symptoms which chiefly support the theory of the existence of specific trophic nervous influences, we have already learned to recognize the degenerative atrophy of the muscles and nerves. Various other sorts of trophic disturbances in the skin and the deeper parts (bones, joints) are seen in nervous diseases. Especially after wounds of the peripheral nerves we often notice a peculiar shining, smooth, atrophic condition of the skin—the "glossy skin" or "glossy fingers" of English authors. In other cases anomalies in the pigmentation of the skin seem to be connected with the nervous disturbances; thus spots deprived of pigment (*vitiligo*) often develop as a result of severe neuralgias. We must also bring to mind here the appearance of changes in pigmentation from nervous causes, especially the ætiology of Addison's disease (*q. v.*) and the occurrence of the so-called nervous nævi. Among the severe neurotrophic disturbances of the skin many observers, especially Charcot, class the appearance of acute bed-sores in many spinal and cerebral paralyses, but we have never been able to convince ourselves of the occurrence of a "neurotrophic decubitus," and we believe that every bed-sore is due in the first instance to external injuries (pressure on the soft parts, uncleanliness, and the entrance of the bacteria of inflammation).

Besides the trophic disturbances in the skin, we often see analogous changes in the nails and hair in nervous patients. The nails become brittle and cracked, assume a darker color, and often show abnormal conditions of growth, abnormal curving, striation, thickening (*onychogryphosis*), etc. We also see at times a loss of the nails. A loss of hair is seen in frontal neuralgia, in certain forms of headache, and not infrequently as an apparently independent nervous disease (*alopecia*). A very rapid whitening of the hair after mental excitement is well known to have occurred in some cases.

Various disturbances of growth and nutrition also occur in the bones and joints, and are to be regarded as trophic disturbances.

The implication of the bones in atrophic processes is seen chiefly in progressive unilateral facial atrophy (*vide infra*). A retarded growth of bone in the affected extremities is also a symptom frequently seen in the spinal and even in the cerebral paralyses that develop in childhood. It is uncertain, however, how far this is due to the loss of direct trophic influences or to the indirect influence of the loss of motor functions. In conclusion, we not infrequently see very striking and apparently trophic disturbances in the bones and joints in tabes and syringomyelia (*q. v.*).

Erythromelalgia.—We should apparently class here this peculiar disease, which is seen most frequently in the feet, although it also occurs in the hands. Men seem to suffer from it more frequently than women. The symptoms are severe pain, especially in the toes and fingers, but sometimes in the ball of the foot, the heel, etc., and also swelling, redness, and marked pulsation of the vessels. In some cases the affected parts assume a very dark-red or bluish-red color. We not infrequently see excessive sweating. [In some cases the condition seems due to neuritis; in others Weir Mitchell, who first described the disease, has found arterio-sclerosis in the smaller vessels with no neuritis.—K.]

The condition described either comes on in paroxysms or is persistent, although varying in severity. On standing or walking, or when warm, the pain usually increases. Other nervous symptoms, such as headache, vertigo, weakness, etc., are sometimes associated with erythromelalgia. The course is almost always very tedious, and the prognosis therefore doubtful. The treatment consists in the use of electricity, cool baths, iodide of potassium, antipyrine, and similar remedies. [Mitchell suggests stretching or resection of the nerves.—K.] Compare the section on acroparæsthesia (page 822), which name has sometimes been given to apparently allied conditions.

Symmetrical Spontaneous Gangrene. Raynaud's Disease.—This disease, whose nature is still quite obscure, begins with pallor and coldness of the fingers ("regional ischæmia"), associated at first with sensations of crawling and numbness, and later with severe pain. After a short time the pallor changes to a bluish-red color ("regional cyanosis"), especially in the terminal phalanges, which sometimes progresses to the formation of gangrenous spots and vesicles. In severe cases a portion of the terminal phalanges is completely destroyed and thrown off by dry gangrene. The whole process may last for months. It runs its course without fever, but it is sometimes associated with quite severe general nervous disturbances. In rare cases spontaneous symmetrical gangrene has been observed in other parts of the body, for example, in the thighs, the buttocks, the ears, the nose, etc. Several attacks of the disease may sometimes follow one another, so that the whole duration of the trouble may be for years.

In regard to diagnosis, we must first be convinced that the disease comes on spontaneously. Extensive areas of gangrene of the skin have repeatedly been observed in hysteria, produced artificially by the patient by means of caustics (caustic potash, etc.). We must also think of diabetic gangrene, severe arteriosclerosis, syringomyelia, exophthalmic goître, etc.

The treatment can be merely symptomatic. Wet applications, baths, cautious galvanization, and narcotics (morphine, chloral) when the pain is severe, are most employed.

Acute Angioneurotic Œdema (*Œdema cutis circumscriptum*).—This name has been given by Quincke, Strübing, and others to a disease characterized by the sudden onset of œdematous swellings in various parts of the body. These swellings usually disappear in a few hours, but they are very often repeated. Dangerous symptoms may arise if the œdema affect the pharynx or the entrance to the larynx. The patient's health otherwise is sometimes perfectly good, but in other cases it is more or less affected. Gastric disturbances especially (attacks of vomiting and gastralgia) have been observed at the same time in such patients. Acute angioneurotic œdema is manifestly closely allied to urticaria and erythema exudativum.

Myxœdema.—We may here mention myxœdema (*cachexie pachydermique* of Charcot), which was first described in England by William Gull and Ord. The disease has taken its name from a peculiar thickening and swelling of the skin, which is most marked in the face, but which is sometimes seen in the extremities, the trunk, the tongue, and even in the internal organs. This swelling is not œdema, but it is due to the development of a sort of myxomatous new growth, rich in mucine, in the connective tissue. Other trophic disturbances usually coexist: atrophy of the teeth and nails, [spade-like hands, coarseness and] loss of hair, failure in the sweat secretion and consequent dryness of the skin, etc. Besides these there gradually develops a general physical and mental weakness, which may increase to great hebetude or even to complete dementia. Disturbances of the sensory functions may also occur. It is a fact of especial interest that we find quite commonly a diminution and even a complete atrophy of the thyroid gland. It is not improbable that all the symptoms of the disease may arise from the failure of function of the thyroid gland. This hypothesis is confirmed by the repeated experiments of Kocher and others, that after complete extirpation of the thyroid in man almost the same symptoms ensue as in myxœdema (*cachexia strumipriva*). From this we must suppose that certain injurious substances accumulate in the body which are ordinarily rendered innocuous by the thyroid gland.

This theory has been materially supported by recent extremely interesting experience in the treatment of myxœdema. The physiologist Schiff made the remarkable observation that the symptoms of cachexia strumipriva did not appear in animals if the thyroid gland of another animal were transplanted into the abdominal cavity. Horsley, Bircher, and others hereupon made the first experiments in the treatment of myxœdema in man with extract of the thyroid gland (from sheep, calves, etc.). At first subcutaneous injections of thyroid extract were usually employed, but it was found that the internal administration of the substance of the gland or an extract thereof had a far more favorable influence. There are already very many positive observations which show that all the symptoms of myxœdema in man may be made to disappear completely in the course of a few months by the continued internal administration of the thyroid gland. The most convenient and certain form of administration is in tablets. Of course we must always be prepared for relapses. [Myxœdema is sometimes associated with exophthalmic goître and rarely with acromegaly.—K.]

[The condition known as **cretinism** is also dependent upon loss of function of the thyroid gland. Endemic cretinism is common in countries where endemic goître prevails, especially in the central Alps, and also, but less frequently, in the Carpathians, the Pyrenees, the mountain districts of Central and South America, and the Himalayas. Sporadic cretinism may occur in all parts of the world, and Osler has collected sixty cases in America. The affection comes on in infancy, and in its severer forms the condition is unmistakable. The victims are short and squat of figure, with prominent belly, "blubber lips, *retroussé* nose, sunken at the root, wide-open mouth, lolling tongue, small eyes, half-closed, with swollen lids, stolid, expressionless face, and muddy, dry skin." They are idiotic and often unable to talk, walk, or stand alone. In the milder cases the lax, waxy skin, the prominent abdomen, and the failure of development may suggest the diagnosis. Marvelous results have been attained in some of these apparently hopeless cases by the early and persistent use of the thyroid gland. The striking cretinous aspect has disappeared and physical growth and mental development have followed.

Closely allied to these conditions is the **myxœdematous infantilism** described by French authors. The subject preserves the physical and mental characteristics of a child of ten up to the age of thirty or over. The sexual organs are undeveloped, and the secondary sexual characteristics fail to appear. A careful examination will often reveal slight indications of myxœdema—the diminution in size or absence of the thyroid, the thick lips, thick dry skin, coarse hair, etc.—thus distinguishing it from the anangioplasic form of infantilism due to defective development of the arterial system. In this condition the administration of thyroid gland produces equally striking results.

Adiposis Dolorosa.—Under this name Dercum has described a peculiar condition, occurring in women between forty and sixty years of age with a neuropathic predisposition. Most of the victims were addicted to alcohol. The trouble begins with a gradual enlargement of various portions of the body, associated with pain, paræsthesia, and some tenderness. These enlargements may increase so as to form huge pendulous masses. Muscular weakness is also present, and locomotion finally becomes very difficult if not impossible. Mental weakness sometimes supervenes. The masses have been found to consist of fat and connective tissue. Disease of the thyroid has been found on autopsy, but the true nature of the trouble is still obscure. The course is slow, but progressive. Treatment has thus far been of little avail. Every effort should be made to improve the nutrition, and thyroid gland should also be given.

Scleroderma.—Although ordinarily classed among the skin diseases, scleroderma merits a brief mention here, as it is generally considered to be a vaso-motor or trophic neurosis. It occurs chiefly in neuropathic women, and it may follow some infectious disease. It sometimes occurs with syringomyelia, Raynaud's disease, or other forms of nervous disease. It may be diffuse and symmetrical or circumscribed (*morphœa*). It begins gradually, often after exposure to cold, with a feeling of stiffness, most marked in the neck, shoulders, arms, face, or scalp. The skin gradually becomes thickened and hard, very white or mottled, and movements of the affected parts become slow and stiff, and finally are completely lost on account of the rigidity of the skin. Vaso-motor symptoms are rare, but pigmentation is occasionally seen. When the face is involved a characteristic, expressionless "mask" is produced. If the chest be affected, respiration may be considerably impaired on account of the immobility. Later on the skin and deeper parts may atrophy, although the hardness and stiffness may remain. Recovery may occur, but the disease is apt to be permanent, the patient succumbing to some intercurrent disease. The circumscribed form may occur in patches, bands, or streaks, and may be confined to the distribution of one or more nerves. The white spots are bordered by a pinkish zone of dilated vessels. Anatomically the disease is characterized by changes in the blood-vessels and proliferation of connective tissue in the skin. The general condition should be strengthened by nutritious diet, good hygiene, salt baths, arsenic, and cod-liver oil. Galvanism is of some benefit. Thyroid extract may also be tried.—K.]

Acromegaly.—A "trophic disturbance" chiefly of the bones, but also involving the soft parts, forms the basis of that peculiar and rare disease recently termed acromegaly (P. Marie, Erb, etc.). The affection develops slowly in women and men, usually in youth or middle age. In women cessation of menstruation almost invariably precedes the onset of the disease. Besides the general symptoms of dullness, fatigue, and quite severe neuralgic or drawing, deep-seated pains in the head and the extremities, there gradually develops a striking increase in the size of the hands and feet, and thickness and plumpness of the face, due chiefly to enlargement of the nose, chin, and lips, which become puffed out to a considerable size. The increase in the lower part of the face (especially the lower jaw) in contrast to the normal cranium gives a very characteristic aspect to acromegaly. The hyperplasia of the lower jaw may reach so high a degree that the individual teeth are separated. In the hands and feet the s and toes, and especially their terminal phalanges, become thicker and , so that we may speak of actual "paws." The hyperplasia not only affects ›nes, but it makes the skin thick and wrinkled. A manifest kyphosis of the usually develops. Other ·orthy symptoms some- occur. Thus we some- see marked glycosuria, increased secretion of , and particularly dis- nces of vision, either ınopsia or amblyopia atrophy of the optic s. These disturbances sion are connected with ıoteworthy fact that in cases of acromegaly a : of the hypophysis cere- ·velops, which presses di- on the nerves or the tract. A patient under ›servation (Fig. 113) died symptoms of a tumor at ase of the brain, and the sy showed a sarcoma- tumor of the hypophysis had grown through the ent bony parts.

ıe diagnosis of acromeg- not difficult. We must y guard against confus- t with other conditions leading to thickening ıe bones (partial giant h, arthritis and osteitis nans, diffuse and local- ıyperostoses, etc.). The led hypertrophic osteo- ›pathy is also to be dis- ished from acromegaly. affection is seen especial- patients with chronic hitis, bronchiectasis, ic tuberculosis, etc., t also leads to a paw- hickening of the fingers, he changes in the lower › very characteristic of acromegaly are absent, and also the other nervous oms, especially those of tumor of the hypophysis.

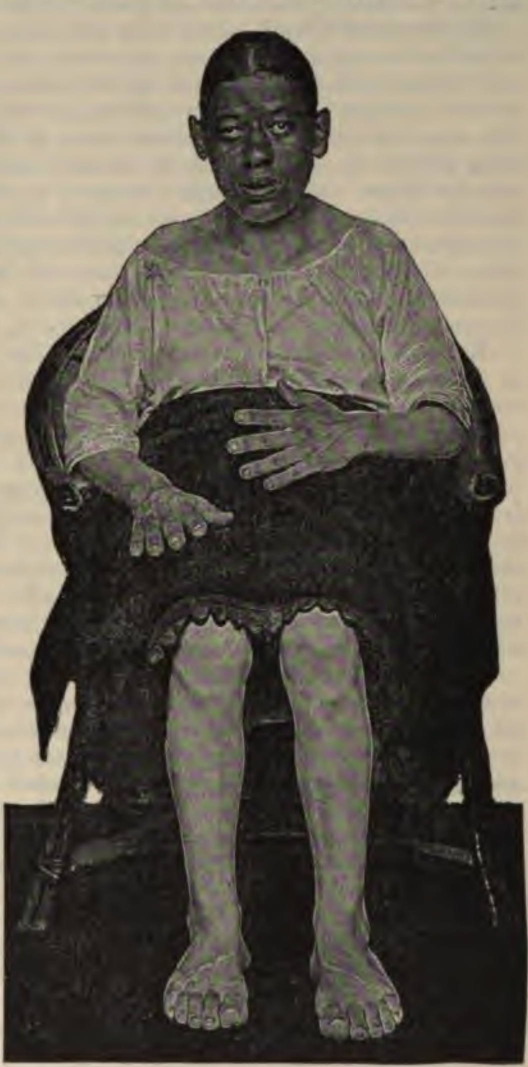

FIG. 113.—Acromegaly in a patient thirty years old. (Personal observation.)

ıe treatment of acromegaly is, unfortunately, almost wholly unpromising. dministration of thyroid tablets and also of tablets of the hypophysis has ried without much benefit.

igantism.—The "giants" who exhibit themselves in circuses and museums ctims of a disease closely akin to acromegaly if not identical with it. A of giant skeletons has shown a marked enlargement of the sella turcica, the site of the hypophysis, and an enlargement of the hypophysis has been found at autopsies. The giants themselves often have enormous hands and feet, they are feeble and short-lived. In some instances kyphosis has developed later in life, with more pronounced symptoms of acromegaly.

Hyperostosis Cranii.—This is an enlargement of the cranium, beginning in early life, involving the anterior part of the skull rather more than the posterior, and leading to enormous thickening of the bones, from one to three or more centimetres (one half to one and a half inch). Other bones are sometimes affected. The symptoms sometimes resemble those of cerebral tumor, the cranial nerves being affected by the bony growth. Headache is common and optic neuritis occasionally occurs. Marked exophthalmus is sometimes produced by the growth of the orbital bones which fills up the orbital cavities. The trouble is supposed to be of trophic origin, although the changes in the bones resemble those of inflammation. The involvement of the cranial vault and the absence of kyphosis and changes in the extremities render it easily distinguishable from acromegaly. Treatment has thus far proved unavailing.—K.]

Hydrops Articulorum Intermittens.—This is the name of a very rare disease, in which large swellings, usually of the knee-joint, but sometimes of the other large joints, develop at perfectly regular intervals of one to four weeks. These swellings of the joints run their course without fever, and usually without any great pain, and disappear again in a few days. Such attacks may be repeated at intervals of different lengths, during many years. Their nervous character is attested especially by the rapid onset and disappearance of the affection, and also by the combination of it with other nervous disturbances, such as angina pectoris, exophthalmic goître, vaso-motor symptoms, hysterical symptoms, etc., which combination has often been observed. In regard to treatment, salicylic acid, quinine, Fowler's solution, and subcutaneous injections of ergotine have been tried, but the trouble is usually very obstinate.

Disturbances of Secretion.—In addition to the vaso-motor and trophic disturbances we must consider the disturbances of secretion. These are not infrequent. We have already learned to recognize anomalies in the secretion of saliva in facial paralysis, and of the lachrymal secretion in trigeminal neuralgia. Analogous symptoms are occasionally noticed in other nervous diseases. Disturbances of the sweat secretion are the easiest to confirm. Our understanding of them comes substantially from the discovery of the "sweat nerves," arising mainly from the sympathetic, which was made by Luchsinger. In nervous patients we have seen quite frequently, on the one hand, an abnormal increase of the sweat secretion (*hyperidrosis, ephidrosis*), and, on the other, a diminution or a complete disappearance of it. The former is seen on the paralyzed side in many hemiplegias and in spinal paralyses, the latter in tabes dorsalis. Anomalies of the sweat secretion are quite frequent, and are usually combined with vaso-motor disturbances in certain general neuroses, such as hysteria and neurasthenia. In a few rare cases a genuine *hæmatidrosis* (bloody sweat) has been confirmed. [Colored sweating (*chromatidrosis*) has been reported in some very rare cases of hysteria.—K.] The condition known as unilateral hyperidrosis (unilateral sweating) is also especially interesting. In this there is an abnormal secretion of sweat, chiefly in one half of the face, more rarely in one arm or over the whole of one side. The affection has usually been observed in connection with tabes, syringomyelia, hemicrania, exophthalmic goître, hysteria, etc., and, in at least a number of cases, it seems to be due to direct lesions of the sympathetic. On the other hand, we have repeatedly seen persons, who were otherwise perfectly healthy, in whom the secretion of sweat, coming on under normal conditions from heat or physical exertion, remained limited to one half of the body, especially the face.

Injuries and Diseases of the Cervical Sympathetic.—In conclusion, we would briefly mention here the symptoms which have been observed in direct injuries of the cervical sympathetic, trauma, pressure of neighboring tumors, etc. If we have to do with a paralysis of the sympathetic, we see almost constantly on the affected side a contraction of the pupil from paralysis of the dilator pupillæ supplied from the sympathetic, in many cases associated with a slow reaction to light. We also frequently see a narrowing of the opening of the lids from paralysis of Müller's muscle, and in old cases a retraction of the bulbus oculi, flattening of the affected half of the face, and occasionally increased redness and warmth in the ear and cheeks from vaso-motor disturbance. In a few cases we see an increased sweat secretion. We may add that, according to Möbius, the normal reflex dilatation of the pupil, from a painful irritation of the skin of the face, is absent in paralysis of the sympathetic. The opposite symptoms are found in conditions of irritation of the sympathetic. The occurrence of sympathetic symptoms in certain injuries of the brachial plexus has already been mentioned above (page 870).

CHAPTER II

MIGRAINE

(*Hemicrania. Megrim. Sick Headache*)

Ætiology.—By migraine or hemicrania we mean a peculiar form of headache, coming on in separate attacks, usually unilateral, and associated with considerable disturbance of the general condition and with pronounced nervous gastric symptoms (complete loss of appetite, nausea, vomiting). The affection occurs in women and men, somewhat more rarely in the latter, and almost always begins in youth, generally at the period of puberty, although typical cases of migraine have been repeatedly observed in school-children. Quite frequently, but not always by any means, the disease affects persons who must be regarded as "generally nervous," who are anæmic, or who suffer from disturbances of menstruation. Heredity often plays a part, since hemicrania is both hereditary as such, and often appears in families which have suffered from other nervous diseases, such as epilepsy, hysteria, or the psychoses. We may mention as exciting causes, which may be made answerable both for the onset of the disease and especially for the individual attacks, physical and mental over-exertion, great mental excitement, disturbances of digestion, indulgence in alcohol, etc. [Eye-strain (*vide supra*) may rarely have some influence in causing migraine as well as other forms of headache, and a careful search for any errors of refraction should therefore be made.—K.] The special cause of the disease, however, is probably, as a rule, a congenital predisposition.

Many writers lay great stress upon certain vaso-motor symptoms which sometimes accompany migraine, and therefore believe that the disease is to be considered in the main an affection of the sympathetic; but we must agree with Möbius that this assumption is very improbable, and that the accompanying sympathetic symptoms are probably only secondary and of reflex origin, in consequence of the pain, or are merely a part of the whole attack of migraine. We are still utterly gnorant of the special nature of migraine—that is, of the kind or the place of the change which forms the basis of the clinical symptoms. The conjecture is interesting, but of course wholly unconfirmed, that there is in the attacks an "auto-ntoxication" of the body—that is, the action of some poison developing from time to time in the body itself; but we do not know upon what part of the nervous system this poison may act; we can merely conjecture that the place of irritation, upon which the pain and other migrainous symptoms depend, is in the brain itself (in the cortex or deeper parts). This conjecture is supported especially by the nature of many of the symptoms which accompany the pain of migraine—the sensitiveness of the sensory organs, the slight mental change, the scintillating scotoma, etc.

Symptomatology.—Migraine always comes on, as has been said, in separate attacks, which are repeated at intervals of varying lengths, although some cases often show a remarkably great regularity. The onset of the attack in women often has some relation to the menses; in other cases it is due to one of the exciting causes mentioned above.

The attack of migraine usually begins with certain prodromal symptoms, which the patient soon recognizes as sure signs of his approaching suffering. These prodromal symptoms consist of general uneasiness, discomfort, pressure in the head, vertigo, at times tinnitus, spots before the eyes, darkening of the visual field, chills, malaise, abnormal yawning, etc. In a short time the pain begins. It is felt most either in the anterior frontal region or in the temporal or parietal region; it generally shows a continuous character, not intermittent as in neuralgia, and it may increase to a very great intensity. Usually one side of the head (the left most commonly) is the chief seat of the pain, particularly the forehead and the neighborhood of the eyes; but it may also happen that the pain affects the right and left sides alternately, and it is sometimes not limited to one side, but embraces nearly the whole head. The scalp is usually hyperæsthetic on the affected side, and the points of exit of the nerves may be sensitive to pressure, but firm pressure on the head (with the hands or by tying a towel tight around it) sometimes gives relief.

The second chief symptom in migraine, besides the headache, is the complete loss of appetite and great nausea. Sometimes vomiting comes on only at the end of an attack, but often there is almost uncontrollable vomiting throughout the whole attack. The patient not infrequently notices that the vomitus has a very sour taste, and we have ourselves found a decided hypersecretion of gastric juice in several cases of migraine. The patients' general condition is almost always very much disturbed. They feel very wretched and dull, and are very sensitive to external impressions, any bright light, any noise, etc. They are incapable of any mental exertion. In many cases remarkable ocular symptoms occur (ophthalmic migraine): slight or marked ptosis of one eyelid, bright flashes before the eyes, scintillating scotoma, and not infrequently pronounced hemianopsia. These symptoms very often forebode the special attack of pain. Other severe nervous symptoms, such as paræsthesia in the hands and fingers, tinnitus, disturbances of speech, etc., have occasionally been observed.

The vaso-motor symptoms accompanying the headache may occur in two different forms. From them migraine was formerly divided into two subdivisions—*hemicrania sympathico-tonica* or *spastica*, and *hemicrania sympathico-paralytica* or *angio-paralytica*. In hemicrania spastica, first described by Du Bois-Reymond from observations on himself, the forehead and ear on the affected side are pale, the skin is cool, the temporal arteries contracted, the pupil is often decidedly dilated, the secretion of saliva increased—in short, there are a whole series of symptoms present which all agree in pointing to a condition of irritation of the sympathetic (*vide supra*). In hemicrania paralytica, however, which was first described by Möllendorff, also from observations on himself, the face is reddened on the affected side, it feels hot, the temporal arteries are dilated and pulsate strongly, there is sometimes unilateral sweating of the face, the pupil is contracted—all symptoms, therefore, which can depend only on a paralysis of the sympathetic.

As has already been said, the significance of all these symptoms is not absolutely certain. We must also add that this division is more theoretical than real, and that the cases which occur in practice can not by any means always be inserted into one or the other typical scheme without further ceremony. The vascular symptoms are often only slight, conditions of paralysis and of irritation of the sympathetic sometimes seem to alternate with each other in the same attack, and we may even frequently meet with apparently contradictory symptoms at the same time, such as pallor joined with contraction of the pupil. The vascular changes, when present, are almost never unilateral, but they are approximately alike in the two halves of the face. In severe attacks the whole face usually looks pale and sunken, but the initial pallor may give place to redness.

The duration of the attacks of migraine differs very much. It usually lasts several hours, or a whole day; then the pain gradually disappears, and there is often considerable vomiting and sometimes a profuse discharge of urine toward the end of the attack. In the intervals between the different attacks most patients are perfectly well and free from pain. There are also severe forms of migraine where the individual attacks may last for several days or more ("*état de mal*"). Without an accurate history these severe conditions of intense headache and persistent vomiting may readily be falsely interpreted.

The whole course of migraine is very chronic, and it may last for years and years. It is usually a trouble to which the patient finally becomes accustomed. We must generally be quite guarded in our prognosis, for many cases resist very obstinately all attempts at cure. We can give the patient only the consolation that the trouble generally disappears of itself in advanced life. It is not usually attended with any special danger. In only a few cases has it been noticed that attacks of hemicrania of years' duration have preceded a severe cerebral disease that developed later.

Diagnosis.—The diagnosis of migraine is not difficult, if we hold strictly to the definition of the disease. The laity, especially women, of course, delight in calling every headache and all sorts of other nervous and hysterical conditions "migraine." Especially characteristic of true migraine are the heredity, the beginning in youth, the occurrence in attacks, the accompanying vomiting, and the other attendant nervous symptoms. Errors in diagnosis may arise from the fact that attacks of symptomatic migraine may also occur in severe organic diseases (tabes, brain tumor, etc.). We have seen a patient who suffered for years with severe "migraine." He finally died, and the autopsy revealed a cysticercus in the fourth ventricle with secondary hydrocephalus.

Treatment.—Very many patients who suffer from migraine finally renounce any special treatment, after they have exhausted all possible remedies. They withdraw to their rooms when the attack comes on, darken the windows, take nothing but some tea, Seltzer-water, or cracked ice, put a cold compress about the head, perhaps try a foot-bath, and for the rest wait quietly until the attack is over. In fact, our remedies for cutting the attack short are quite uncertain. They sometimes aid, but they often leave us in the lurch, especially if used repeatedly. We must note especially that narcotics, such as morphine, are almost always ill borne in migraine, and do no good; but antipyrine, sodic salicylate (20–30 grains [grammes 1.5–2.0], in strong black coffee), antifebrine, phenacetine, salipyrine, etc., in many cases undoubtedly have a favorable action. Which remedy acts the best must usually be tested in the individual case. We have ourselves seen excellent results formerly from sodic salicylate, and lately from antipyrine, pure or mixed with the other remedies mentioned; the attacks of migraine, when the remedy was taken immediately upon the onset of the first symptoms, have become distinctly milder and have run their course more rapidly. Of course the action often ceases with time, and we must then try another of the remedies mentioned. Of the other remedies used we may mention guarana

(*Paullinia sorbilis*), half a drachm to a drachm (grammes 2–4) of the powder, and caffeine, or sodio-salicylate of caffeine, in three- to five-grain doses (gramme 0.2–0.3), which is sometimes very serviceable. Caffeine is also the chief constituent of "migrainine," which has lately been much praised. On theoretical grounds, inhalations of nitrite of amyl, three to five drops on a napkin, have been used in spastic hemicrania, and subcutaneous injections of ergotine in the paralytic form (aqueous extract of ergot, 2.5; dilute alcohol and glycerine, each 5; or one part of dialyzed ergotine in four parts of distilled water: injections of either, three to fifteen minims). The practical benefit, however, is very doubtful. Many other nervines, such as bromide of potassium and Fowler's solution, have been recommended for continued use, and also extract of cannabis indica; and, lately, nitrite of sodium, two parts in 120 of water, a teaspoonful one to three times a day, and nitro-glycerine, in troches containing $\frac{1}{120}$ to $\frac{1}{60}$ grain (gramme 0.0005–0.001). Their action is similar to that of nitrite of amyl. In ophthalmic migraine the treatment by large doses of bromide of potassium is praised, especially in France, as being very successful.

In many cases the general treatment is very important. Preparations of iron, sea-bathing, a mountain residence, and cold-water cures are often of decided service. We have seen remarkably good permanent results in several cases of severe migraine from the moderate use of Carlsbad water, either at Carlsbad itself or taken at home. We always strongly recommend a trial of this, especially when there are also symptoms of hypersecretion of the gastric juice (*vide supra*). With the moderate use of Carlsbad water we usually combine methodical hydrotherapy (cold sponging, half baths with douches, etc.) and an appropriate diet. The persistent application of electricity has also shown some good results, but we must not build very great hopes upon it. In the spastic form the action of the anode on the sympathetic is especially recommended, and in the paralytic form the action of the cathode, while the other electrode is to be placed on the cervical cord, or as high as possible on the occiput in the region of the medulla. Cautious galvanization of the head, and weak primary faradic currents, may also be used. Specialists in massage praise their mode of treatment in migraine; they massage either certain painful spots in the head, or the gastric region. Finally, we must mention that migraine often seems to be connected with diseases of the nose, especially with hyperplasia of the erectile tissue in the nose, and that in such cases treatment of the primary disease with the galvano-cautery may result in the cessation of the migraine.

CHAPTER III

PROGRESSIVE FACIAL HEMIATROPHY

(*Unilateral Progressive Facial Atrophy*)

UNILATERAL facial atrophy is an extremely rare disease, which consists of a very slow and gradual but usually constantly progressive atrophy of one half of the face, and affects the skin, and also the fatty tissue, the muscles, and the bones, either in a uniform or a very diverse manner. The affection usually begins in youth. The female sex seems to be more disposed to the disease than the male.

The atrophy, which has its seat much more frequently on the left side than on the right, begins usually in a circumscribed spot either on the cheeks or on the chin. The skin, as a rule, gradually assumes a whitish or brownish color. The affected part, and finally the whole half of the face, gradually sink in more and more, so that the disease can be recognized at the first glance. The atrophy shows a sharp limitation at the median line. In many cases the muscles apparently remain almost wholly intact, but in some cases they show a marked atrophy, especially the muscles of mastication. The corresponding half of the tongue and the soft palate has sometimes been found implicated. Exceptionally, the atrophy involves the neighboring region of the shoulder and the upper extremity. The bones also atrophy, especially in the cases which arise in early youth. The hair on the affected half of the head often falls out in great amount, and it becomes thin and atrophic. The sensibility remains perfectly intact. Marked vaso-motor and secretory disturbances have only rarely been observed. The accompanying illustration (Fig. 114) shows a patient who was described by Romberg many years ago, and who for a long time frequented the German cliniques in order to show himself.

Nothing definite is known in regard to the nature of the affection. Most observers at present agree that it is a trophic neurosis, an affection of trophic nerves or nerve-centers, but where we are to look for the special seat of the disease, whether in the trigeminus or in the sympathetic, we do not know. Mendel lately found in a case which came to autopsy a definite neuritis in the trigeminus.

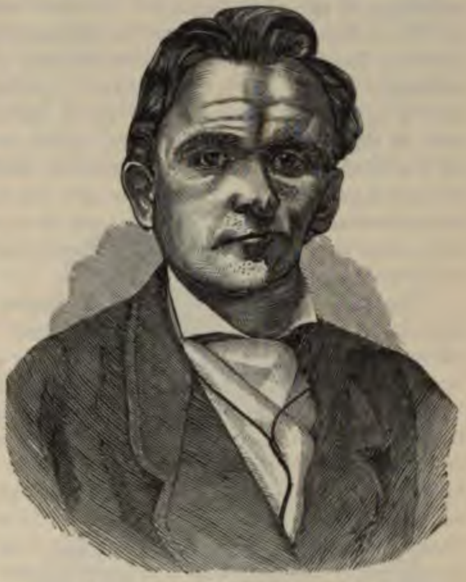

FIG. 114.—Left facial hemiatrophy.

The disease is not dangerous in itself, and usually causes no special subjective disturbance, but it seems to be incurable. In cases at their beginning we can at most make an attempt to bring the disease to a standstill by a long-continued application of electricity.

As an appendix it may be briefly mentioned here that there is a unilateral hypertrophy, which is also possibly connected with neurotrophic disturbances. We saw at Leipsic a ten-year-old boy, otherwise perfectly healthy, in whom a striking hypertrophy of the left side of his face and of the left arm had gradually developed.

CHAPTER IV

EXOPHTHALMIC GOÎTRE

(Parry's Disease. Morbo di Flajani. Graves's Disease. Basedow's Disease)

Ætiology.—The special group of symptoms to which the name of exophthalmic goître has been given, and whose three cardinal symptoms are acceleration of the pulse, goître, and exophthalmus, was first carefully described in Germany, in the year 1840, by the Merseburg physician Basedow, although similar but less precise observations had been published in England by Graves five years earlier. [Parry, in England, was the first accurately to describe the disease in 1786, and it was also recognized by Flajani in Italy in 1800.—K.]

In the ætiology of the disease all those factors play a part which are important in the ætiology of neuroses in general. In many cases a hereditary predisposition can certainly be discovered. The disease has been repeatedly seen in several members of the same family. We have ourselves seen severe exophthalmic goître in two sisters. Furthermore, exophthalmic goître is also quite frequent in those families in which there is a hereditary predisposition to neuroses in general—epilepsy, the psychoses, or hysteria. Among the exciting causes we must first mention great mental excitement—grief, terror, anger. Sometimes real injuries, as well as these "psychical injuries," seem to have an influence on the development of the disease—that is, great general bodily concussion, such as a fall. Many authors have laid considerable weight on diseases of the female sexual organs, but the importance of this factor seems to us to be exaggerated. It is certain, however, that the first symptoms of exophthalmic goître often develop at the period of pregnancy.

The influence of sex upon the origin of the disease is plain, since women, especially somewhat anæmic, "nervous" women, are much more frequently affected than men. Exophthalmic goître usually appears in middle life, while it is seen only exceptionally in children and old people.

Symptomatology.—Of the three cardinal symptoms of exophthalmic goître named above, of which, of course, one or another is often absent or only slightly developed, the acceleration of the pulse is the most constant and usually the earliest. The frequency of the pulse averages 100 to 120, sometimes only 80 or 90, but in other cases even 140 or 160. It is not alike at all times, but has many variations—the symptoms lasting for long periods and also coming on in single paroxysms. In sleep the frequency of the pulse, as we have ourselves confirmed, is sometimes almost normal, and sometimes also rapid, but usually less so then when the patient is awake. A very vigorous action of the heart, and as a rule the subjective feeling of palpitation, are usually associated with the acceleration of the pulse. There is a vigorous pulsation of the carotids, and sometimes of the smaller arteries. We do not discover any qualitative changes of the pulse. The pulse is usually quite regular, but arhythmia has been exceptionally observed. In some cases the patients suffer from pronounced angina pectoris.

Physical examination of the heart in many cases shows nothing especial, except an accelerated and violent action of the heart, but we sometimes find, as we can affirm from several cases in our own experience, a manifest hypertrophy of the left ventricle, and also dilatation of the heart, and even actual valvular disease. In the diagnosis of the latter some caution is necessary, because functional heart murmurs, especially at the base of the heart, are often present in exophthalmic goître.

The goître usually develops somewhat later than the first symptoms in the heart. In many cases it is entirely absent, or present only in a slight degree. The swelling of the thyroid gland is generally only exceptionally very marked. There are sometimes decided variations in it in the course of the same case. The comparative softness of the tumor, the frequent and strong pulsations in it, and the loud vascular murmurs, which are often but not always heard, and which arise in the dilated vessels of the thyroid gland, are characteristic of the goître in Basedow's disease. By laying the hand on it we can often feel the thrill and pulsation.

The exophthalmus, the protrusion of the eyeballs from their orbits, is always bilateral, although it is sometimes more marked on one side than on the other (see Fig. 115). In many cases it is entirely absent; in others it may attain so high a degree that an actual "dislocation of the eyeball" has been described. In the marked degrees of exophthalmus there is often a peculiar staring expression to the countenance. This is due chiefly to the unusual size of the palpebral fissure and to the less frequent winking than normal (Stellwag's sign). A peculiar symptom, first described by Graefe, is also worthy of mention. On raising and, still plainer, on lowering the eyes, the corresponding associated movements of the upper eyelid, which are always present under normal conditions, are absent. This "Graefe symptom" may sometimes be one of the earliest signs of the disease, and may therefore be of diagnostic value; but we must maintain, after much personal experience, that it is on the whole rare, and that it may often be wholly absent even in the severest cases of the disease. Disturbances of the pupils and of accommodation are unknown in exophthalmic goître, but paralyses of the external ocular muscles sometimes occur, and we have ourselves repeatedly observed anomalies in the movements of the eyeball, especially temporary strabismus. We might mention one symptom, which Möbius first noticed, and which we also have repeatedly but not constantly seen, especially in patients with rather marked exophthalmus. It consists in the fact that one eye very soon deviates outward if we have the patient converge the eyes strongly, as in fixation of a near object—"insufficiency of convergence." Inflammatory processes in the eye are sometimes seen, which are probably due to the fact that the upper lid can not so well protect the protruding eye.

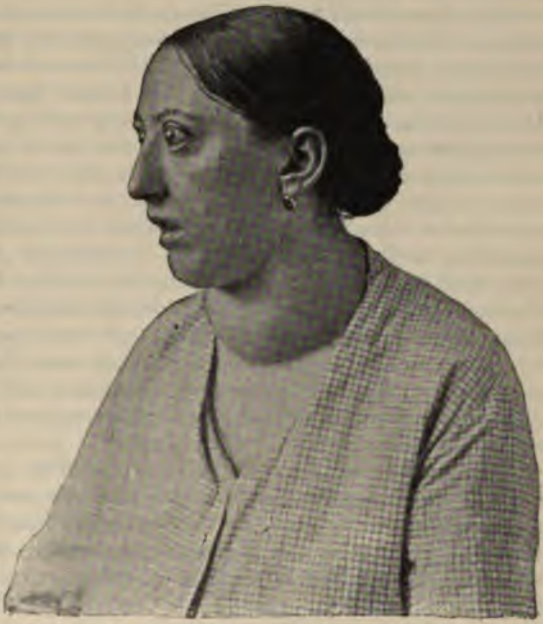

FIG. 115.—Patient with Basedow's disease (exophthalmus, goître). (Personal observation.)

Besides the chief symptoms of exophthalmic goître thus far described, we must also mention a list of other symptoms which come under our observation, both in the typical cases and still more often in anomalous cases—the so-called "*formes frustes*" of the French. Among them are some nervous symptoms, especially a peculiar tremor to which Marie first called attention. This tremor affects the whole body, or the extremities alone; it shows at times temporary remissions and exacerbations, and it may be so severe as to form the patient's chief complaint. A moderate tremor of the fingers and hands is one of the most constant symptoms of exophthalmic goître. In a case under our observation marked tremor was one of the first symptoms of the disease. It may become so violent at times that there are even spasmodic twitchings in the extremities and in the muscles of the face. We may also mention headache, vertigo, weakness of memory, and sleeplessness. The peculiar nervous anxiety and the irritable disposition of the patient are, however, very frequent, and in fact very characteristic, in many cases of the disease. The anxiety and the haste in all movements, in speaking, etc., often show themselves, even during the physician's examination, in so striking a way that they must be regarded as not unimportant factors in diagnosis. The easily occurring flushing of the face, the marked subjective feeling of heat, and the hot hands, from which many patients suffer, are probably due to vaso-motor disturbances. Objective elevations of temperature up to 100° or 101.5° (38°–38.8° C.) have also been repeatedly confirmed by others (Eulenburg) and by ourselves. A marked increase of the sweat production, which in rare cases is only unilateral, is often associated with the feeling of heat. On the other hand, one of our patients complained of a constant dryness in the mouth.

Of symptoms which are referred to other organs we must first consider some disturbances on the part of the respiration. The respiration is usually moderately accelerated, and many patients complain of dyspnœa and of a feeling of oppression in the chest. In one case we saw at times deep spasmodic inspirations; in other cases a peculiar dry, "nervous cough" appears, such as we not infrequently see in patients with goître. There are also symptoms on the part of the digestive organs. Paroxysmal vomiting is common, and in severe cases it may become so persistent, distressing, and uncontrollable as to constitute one of the chief dangers of the disease. In one of our female patients the disease began with an attack of almost uncontrollable vomiting lasting for several weeks. We less frequently see peculiar paroxysmal sero-mucous or even bloody diarrhœa. We have once seen severe jaundice. Finally we must mention certain disturbances in the skin: vitiligo has often been observed, and also diffuse brownish pigmentation of the skin or chloasma-like pigment-spots and urticaria. It is a noteworthy fact, first discovered by Vigouroux, that the galvanic resistance of the skin is remarkably small in patients with exophthalmic goître. This condition is probably due to the fact that the skin is made wet by the profuse sweating, and also, perhaps, to some form of atrophy of the skin. A very rare but dangerous occurrence, of which we ourselves have seen a striking example, is an apparently spontaneous gangrene of the extremities. In our case, which ended fatally, the gangrene affected the right leg. Not the slightest anomaly could be made out in the vessels at the autopsy.

The general nutrition of the patient suffers in most cases; a certain degree of anæmia and emaciation is often present. In severe cases, especially in those of rapid development, there is often in a very short time a high degree of emaciation associated with much general weakness. Muscular atrophy frequently develops, chiefly in certain regions—the arm or leg muscles. We may mention, in conclusion, that in many, though not in all, female patients disturbances of menstruation (especially amenorrhœa) occur. In a few cases peculiar atrophic conditions have also been found in the genitals and the breasts.

Special complications are not very common in exophthalmic goître. We may mention as most important that other neuroses (hysteria, epilepsy) and true psychoses may also occur with exophthalmic goître. The complication with diabetes, or, as a rule, more properly, with alimentary glycosuria, is also of interest. Tabes has also been found associated with exophthalmic goître. [Myxœdema and acromegaly may also be present.—K.]

Pathological Anatomy and Pathogenesis.—Although all the symptoms of exophthalmic goître point to an affection of the nervous system as a cause of the disease, as we see from the symptomatology, the results of pathological investigations are still very meager. There is a class of cases in which changes in the sympathetic, and especially in the lowest cervical ganglion, are said to have been present; but the pathological significance of the discovery is not placed beyond all doubt; and in other cases nothing abnormal at all could be found in the sympathetic. The theory that all the symptoms of exophthalmic goître are derived from a disturbance of the sympathetic also meets many difficulties and contradicıs. If we regard only the three cardinal symptoms, we can bring the accelera- ı of the pulse, and perhaps the exophthalmus, into harmony with the theory of tation of the sympathetic; but not the goître, which is due to a dilatation of vessels. The theory of a paralysis of the sympathetic explains the goître, and › the exophthalmus—if we assume as the cause of the latter a dilatation of the sels in the back of the orbit—but again the acceleration of the pulse remains xplained. The experiments by Filehne, in which symptoms said to be similar hose of exophthalmic goître were produced by dividing the restiform bodies in ng rabbits, have had up to the present time no bearing upon human pathology, e the restiform bodies have been found as a rule to be perfectly normal in opsies of patients with exophthalmic goître. The theory of the disease ad- ced by Möbius is by far the most probable and the most in accord with our sent scientific views. According to this, exophthalmic goître is due to a pri- y morbid change in the function of the thyroid gland, either from the abnor- ly great formation of certain irritating substances in the gland, or from the erfect destruction of such substances. At any rate, the peculiar contrast be- en the symptoms of cachexia strumipriva (*vide supra*, myxœdema) and those xophthalmic goître is striking. In cachexia strumipriva there are absence of thyroid, and then thickening of the skin, slow pulse, motor and mental slow- s, but in exophthalmic goître there are general emaciation and atrophy of the ı (diminished electrical resistance), tachycardia, mental irritability, tremor,

The results of thyroidectomy in exophthalmic goître (*vide infra*) also or the view that a change in the function of the thyroid gland is the main ture of the whole symptom-complex. [Overdoses of thyroid gland will give to certain symptoms like those of exophthalmic goître (nervousness, tachy- lia, etc.), while even small doses of the gland may aggravate the symptoms if n to a patient with exophthalmic goître.—K.]

Course and Diagnosis.—The course of the disease is in most cases very chronic, it may extend over years and years, but there are also more acute cases with a d development of all the symptoms. We may often see considerable variation he intensity of the symptoms. All the symptoms of the disease may often al- t wholly disappear, to recur after the lapse of years. In other cases, especially he milder form, there is not complete recovery, but there is a permanent arrest he disease. In general, the cases beginning in youth give a more unfavorable gnosis than those arising in later years. Complete recoveries have certainly ı observed, but they are not very common, although marked improvement in he symptoms is by no means rare. In some acute and apparently very severe s especially we have seen almost complete recovery; there was usually only a ain amount of exophthalmus after all the other symptoms had disappeared. disease sometimes terminates fatally with the signs of general weakness and lly paralysis of the heart, but more frequently from complications in the heart ungs.

In fully developed cases of exophthalmic goître a positive diagnosis can almost ays be made without difficulty. The peculiar facial expression, altered by ciation and exophthalmus and associated with goître, often permits the recog- on of the disease at the first glance; but the diagnosis is often very uncertain he milder, undeveloped cases. In many patients with slight or even marked re we find some "Basedow symptoms" without being able to call it actually asedow's disease." If there be no evident goître, we must be very slow in our gment, but absence of exophthalmus with other plainly developed symptoms ften seen. On the other hand, when there really is exophthalmus it is one of most unequivocal symptoms. In all doubtful cases we should consider care- y the mode of onset and the associated symptoms, especially emaciation, tre- , general nervous irritability, the feeling of heat, the tendency to sweating, etc.

Treatment.—In the first place we must consider the general treatment of the patient. Physical and mental rest, good food, and the avoidance of all stimulants —such as alcohol or strong coffee—and the cautious use of cold-water cures, especially sponging, salt baths, etc., may cause a decided improvement of the condition. For anæmic patients we prescribe iron, alone or in combination with small doses of arsenic. A course at the springs at Franzensbad, Schwalbach, Pyrmont, Elster, and Cudowa, is also sometimes attended with good results. Residence in mountainous regions or by the sea often seems to act still more favorably.

Of other remedies, electricity is first to be mentioned, especially the application of galvanism to the neck—the so-called galvanization of the sympathetic at the inner border of the sterno-mastoid. The slowing of the pulse, which sometimes comes on at once ("vagus irritation," or mental quieting!), is striking. Vigouroux praises faradization of the sympathetic and of the goître as the best method of treatment. Among internal remedies we may recommend atropine or tincture of belladonna, and ergot or ergotine. We believe we have repeatedly seen good results from the latter. Many physicians reject the use of preparations of iodine as without benefit, but we have ourselves at times seen decidedly good results from the long-continued use of small doses of iodide of sodium, etc. The administration of preparations of thyroid gland, however, are not to be advised, both for theoretical and practical reasons [*vide supra*]. Digitalis has often been prescribed for the palpitation, but usually without any good result. With great exophthalmus the eyes must be protected from external injuries. The occasional severe symptoms on the part of the digestive organs (vomiting, diarrhœa) must be treated symptomatically, by ice, opium, and champagne.

In view of the above-mentioned theories as to the nature of exophthalmic goître, partial extirpation of the goître has of late been frequently tried. Very favorable results have been reported by different physicians, but, on the other hand, there have of course been some sad experiences. In general we should advise the operation only in severe cases where all other methods of treatment had proved of no avail.

III.—THE DISEASES OF THE SPINAL CORD

CHAPTER I

DISEASES OF THE SPINAL MENINGES

1. ACUTE INFLAMMATIONS OF THE SPINAL MENINGES

Ætiology and Pathology.—Isolated acute inflammation of the spinal meninges is very rarely primary, so far as we know, but inflammatory processes in the neighborhood quite frequently involve the meninges, or a spinal meningitis occurs as one symptom of a general cerebro-spinal meningitis. This latter condition is seen chiefly in the idiopathic, generally epidemic, cerebro-spinal meningitis—a specific infectious disease, which has already been described in detail in a previous chapter. A tubercular spinal meningitis is also very often combined with tubercular inflammation of the cerebral meninges, but, since the symptoms of the latter are usually in the foreground of the picture, we will treat of tubercular cerebro-spinal meningitis in the section on diseases of the cerebral meninges. Secondary cerebro-spinal meningitis is sometimes seen in the course of certain other infectious diseases, and is then probably to be regarded as a special localization of the specific poison of the disease. This is the explanation of the occurrence of acute spinal and cerebral meningitis as a sequel of croupous pneumonia, and also of its occurrence in pyæmic and septic diseases, and, very rarely, in typhoid fever and the acute exanthemata. We must mention, finally, the occurrence of a purulent cerebro-spinal meningitis as a sequel of empyema, pulmonary gangrene, etc., which, although very rare, we have repeatedly noticed. In these cases the infection of the meninges also results from the primary foci of disease, but the channel of infection is not yet exactly known. Perhaps the intercostal nerves are the media of communication.

In all the cases so far mentioned we have chiefly an inflammation of the pia mater, a so-called *lepto-meningitis*; the dura mater is not implicated in the disease at all, or only to a slight degree. The condition is different in those inflammatory processes which gradually invade the meninges from the neighboring parts outside the cord. Thus we very often see circumscribed inflammations on the outer surface of the dura (pachymeningitis) in caries of the vertebræ, and these inflammations often invade the inner surface of the dura, or more rarely reach the pia mater. Acute purulent peripachymeningitis is a very rare disease; it is a purulent inflammation of the connective tissue between the dura mater and the vertebral column, which is always of secondary origin. We have seen a very characteristic case of this sort in the course of a puerperal pyæmia. The inflammation had spread from a purulent inflammation of the pelvic cellular tissue, through the foramina of the vertebral canal, and had finally set up a purulent inflammation on the outer surface of the dura, extending up to the cervical cord. We meet with an inflammation of the pia mater chiefly in diseases of the spinal cord, from an extension of the process, as the pia is involved to a greater or less extent in many cases of myelitis.

We do not know with certainty whether other influences, especially injuries and exposure to cold, can lead directly, as has often been claimed, to inflammation of the spinal meninges.

We need to say but little in regard to the pathological anatomy of acute spinal meningitis. The changes in purulent inflammation of the pia mater have been described in the chapter on epidemic meningitis. Precisely the same conditions are found in the other forms of acute leptomeningitis. The changes in pachymeningitis are completely analogous. The dura mater is traversed by dilated vessels, and therefore is reddened; it is also thickened, and on its internal or external surface (*pachymeningitis interna* or *externa*, or *peripachymeningitis*) there is usually found a purulent or a sero-purulent exudation.

Symptoms.—An accurate distinction between acute inflammations of the pia mater and those of the dura mater can not be made clinically. The symptoms of the disease include the symptoms of any primary disease present, the general symptoms, such as fever, etc., and in addition the necessary consequences which the presence of a disturbance of the meningeal circulation and of the meningeal exudation exerts on the cord and nerve-roots. These consequences are due both to a mechanical compression of the parts named, and often probably to an invasion of the substance of the cord itself by the inflammation. To these is added the frequent combination of spinal symptoms with the symptoms of a co-existing cerebral meningitis.

The symptoms of acute spinal meningitis are already known to us from the description of epidemic meningitis (see page 111). Recapitulating them briefly, we may mention chiefly the very severe pain in the back, the great sensitiveness of the vertebral column, and the stiffness of the back and neck. To these may be added usually symptoms of irritation on the part of the nerve-roots: eccentric pains in the trunk and the extremities, hyperæsthesia of the skin and of the deeper parts, symptoms of direct or reflex motor irritation, muscular tension, contractions, etc. The cutaneous and tendon reflexes are often, but not always, much diminished or abolished in consequence of the lesion of the nerve-roots. There are at times disturbances in the passage of urine and fæces. If, in the later course of the disease, there are actual paralysis and anæsthesia, they are usually a sign of a more marked implication of the cord itself.

Diagnosis.—From the symptoms named, we can in many cases make a diagnosis of spinal meningitis. Of course a meningitis is found often enough on the autopsy-table whose symptoms during life were completely obscured by other severe general symptoms, while, on the other hand, with severe constitutional symptoms the symptoms of meningitis may be illusory, as in typhoid fever or pyæmia. Fuller information as to the seat and the extent of the inflammation is afforded by considering the most painful parts of the vertebral column, the predominance of pain and cutaneous hyperæsthesia in the arms (cervical region) or legs (lumbar region), etc. When the meningitis involves the upper portion of the cord and the medulla there may also be disturbances of respiration, symptoms in the pupils, and anomalies in the innervation of the heart. We can decide as to the form of the meningitis, whether purulent or tubercular, only by a consideration of the history, the other morbid symptoms, and the course of the disease.

Prognosis.—We have seen an undoubted recovery, in severe cases, only in epidemic cerebro-spinal meningitis, and in the sporadic cases of idiopathic meningitis, which are probably identical in ætiology. In all other cases reported with a favorable termination the diagnosis may be doubted, for in general the rule is certain that, in extensive acute purulent leptomeningitis and pachymeningitis the prognosis is almost absolutely unfavorable, whether it be secondary to another infectious disease or arise from propagation from some neighboring focus of inflammation. We may, perhaps, make an exception of certain mild, circumscribed cases, which do not come to suppuration, but these are always uncertain in regard to diagnosis.

Treatment.—In regard to treatment we must refer entirely to what has been aid under epidemic and tubercular meningitis.

2. Chronic Spinal Leptomeningitis

Although chronic leptomeningitis (usually wrongly termed chronic spinal neningitis) once played quite a large part in the diagnosis and pathology of disases of the spinal cord, we must at present assert that its occurrence as an indeendent disease is exceedingly rare. Most of the reported cases of chronic menngitis were published at a time when the diagnosis of many diseases of the cord tself was still perfectly impossible, and when the thickenings and opacities of he meninges were much more striking at the autopsy than far more essential hanges in the substance of the cord itself, which could be made out only by a careful microscopic examination, and not by the naked eye. In recent years only very few cases have been published which can be regarded, at least with some eason, as primary chronic meningitis. It is especially hard to determine this ince syphilitic disease of the spinal meninges (*vide infra*) shows almost precisely he same anatomical appearance. Clinical experience is also entirely against the dea of the frequent occurrence of chronic spinal meningitis. Among many cases f spinal disease we shall scarcely be induced even to assume the probability of he existence of primary chronic meningitis.

The case is different with secondary chronic leptomeningitis. This, in the first place, is, in rare cases, the termination of an acute meningitis. Secondary leptomeningitis may certainly be detected, especially in epidemic meningitis. We also find chronic meningitis frequently as a secondary affection in primary iseases of the cord and the vertebræ. Thus, for example, in old cases of chronic pinal disease, associated with atrophy, such as tabes dorsalis, progressive muscuar atrophy, etc., the pia is almost always quite opaque, thickened, and often nited to the cord and the dura by very many firm adhesions, while a cloudy ero-gelatinous exudation is found in the meshes of the arachnoid. All these nomalies, however, are of a secondary nature, and have no clinical significance; or the same changes, though rarely so marked, are quite often found in old eople, where they are analogous to the equally frequent opacities of the cerebral neninges, the pleuritic adhesions, etc., and where, during life, they have not aused the slightest symptoms of spinal disease.

The symptoms which have been set down as characteristic of leptomeningitis orrespond precisely to those of acute meningitis, except, of course, that they are elatively less intense, and that the course of the disease is more protracted. Pain and stiffness in the back and neck, abnormal painful sensations and paræshesia in the extremities, a girdle sensation, and finally paresis, anæsthesia, and esical disturbances, are the leading features of the type of disease as constructed, n whose fabrication there have been, at any rate, many confusions with myelitis, pinal syphilis, spondylitis, beginning tabes, multiple neuritis, etc. In the few ases confirmed by autopsy the clinical picture resembled in some degree, from he intention tremor of the arms and the spastic symptoms in the legs, that of nultiple sclerosis. It is strange that often there were scarcely any shooting pains uring life in spite of marked meningeal changes.

It is clear that under such circumstances no special rules for the diagnosis or ven the treatment of chronic spinal meningitis can be given. Given a case, we would try local applications to the vertebral column; painting with iodine; dry, r, exceptionally, in strong patients, wet cups; also protracted tepid baths, 90° o 95° (26°–28° R.), or cautious cold-water treatment; and finally the use of the alvanic current. Of internal remedies, iodide of potassium would be most indiated. We may refer to the description of the treatment of myelitis in regard to ll further details.

3. Pachymeningitis Cervicalis Hypertrophica

Pachymeningitis cervicalis hypertrophica was first fully described, as a special yet very rare form of disease, by Charcot in 1871, and later by his pupil Joffroy. Ætiologically, it is still utterly obscure. Alcoholism, exposure to cold, and injury have been alleged to cause it. No small number of cases seem to have some connection with syphilis.

Anatomically, the disease is characterized by a chronic and often very considerable thickening of the dura, almost always, as it seems, in the cervical portion of the cord, while the pia takes but a comparatively small part in the affection. The dura may attain a thickness of six or seven millimetres, and usually appears composed of a number of concentric layers. Histologically, the hypertrophy consists of a new growth of dense connective tissue. The clinical symptoms of the disease arise from the fact that, first, the penetrating nerve-roots, and later on the cord itself, undergo a considerable mechanical compression. If this is of high degree and persistent, there are, as a necessary result, secondary degenerations of the motor nerves and muscles, and a secondary descending degeneration of the pyramidal tract in the cord.

The clinical symptoms are easily understood from this. The disease almost always begins with severe pain, which shoots from the neck into the occiput and the arms. Besides this, there are paræsthesia and a numb feeling in the arms and hands. Rarely there is an eruption of herpes. All these symptoms depend upon the irritation of the posterior roots.

After this first period of the disease (*période douloureuse* of Charcot) has lasted some two or three months, the second period begins—the period of paralysis. An atrophic paralysis in the upper extremities gradually develops, mainly as a result of the compression of the anterior motor roots. This affects, in a remarkable manner, chiefly the distribution of the ulnar and median nerves, while the distribution of the radial on both sides usually remains free. The hand, therefore, assumes a characteristic position (Fig. 116), as a result of the contracture of the antagonistic extensors. The paralyzed muscles rapidly become atrophic and show a marked reaction of degeneration. In this stage there may also be partial anæsthesia of the skin.

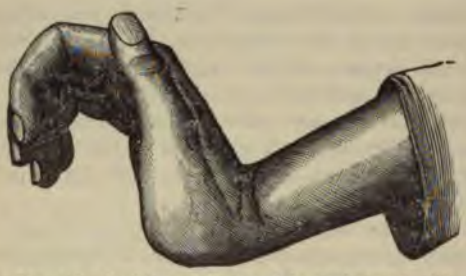

Fig. 116.—Position of the hand in pachymeningitis cervicalis hypertrophica. (From Charcot.)

If the compression of the cord advances, the motor fibers for the lower extremities, which pass through the cervical cord, must necessarily at last be involved sympathetically—the third period of the disease. The result of this is a spastic paralysis of the lower extremities—that is, a paresis or paralysis with increased tendon reflexes, but of course without muscular atrophy, because the trophic centers for the muscles of the legs, in the anterior cornua of the lumbar cord, remain perfectly intact. The compression of the cervical cord, however, may finally lead also to anæsthesia of the lower extremities, to paralysis of the bladder, and bed-sores, under which symptoms death ensues; but, on the other hand, it must be mentioned that probably cases of recovery, or at least of actual improvement in pachymeningitis cervicalis hypertrophica, may occur even after it has lasted for years.

The diagnosis of the disease is based first upon the fact that the affection begins with pains in the arms, and upon the later appearance of the characteristic paralyses. It may easily be confused with tumors in the cervical cord and with cervical spondylitis. Amyotrophic lateral sclerosis is distinguished, however, by the absence of disturbances of sensibility, by the bulbar symptoms, and by the fact that the functions of the bladder remain intact.

Treatment can do little directly, and it must be chiefly symptomatic. Baths, iodide of potassium, and electricity are most used. Joffroy recommends the application of the actual cautery to the neck.

4. HÆMORRHAGE INTO THE SPINAL MENINGES

(*Hæmatorrhachis. Meningeal Apoplexy. Pachymeningitis spinalis hæmorrhagica interna*)

Large hæmorrhages into and between the spinal meninges are of rare occurrence. They arise chiefly from traumatic influences, from concussion or fracture of the vertebral column, or from direct injuries of the meninges, such as stabs or gun-shot wounds. In a few cases great physical exertion may also lead to a meningeal apoplexy. Diseases of the vertebræ, caries, and carcinoma, may also lead to a hæmorrhage from the erosion of a vessel. The frequent little meningeal hæmorrhages which appear as a complication of meningitis, in hæmorrhagic diseases, in the course of severe general infectious diseases, septic infection, typhoid fever, and small-pox, and as a result of severe general convulsions, very rarely have any clinical significance. Finally, it may be mentioned that aneurism of the aorta or its branches may rupture into the vertebral canal.

The clinical symptoms of meningeal hæmorrhage are almost always sudden and "apoplectiform," but are unattended by any disturbance of consciousness. Their intensity depends entirely upon the degree of compression which the nerve-roots and the cord undergo from the effused blood. The symptoms of irritation usually predominate—severe pain in the back, paræsthesia, and neuralgic pains in the extremities; and also symptoms in the motor distribution, tension, tremors, and contractures of the muscles. With large hæmorrhages, symptoms of paralysis, partial anæsthesia, disturbances of the bladder, symptoms of unilateral lesion, etc., may ensue, but the different types of the disease of course vary with the seat of the hæmorrhage. On the whole, the diagnosis of meningeal hæmorrhage can but rarely be made with any certainty, unless suggestive ætiological factors precede and the symptoms and manner of beginning are especially characteristic.

The course is quite favorable in many cases if the blood be rapidly reabsorbed, but sometimes a permanent disturbance of function is left.

In regard to treatment, complete rest and the energetic local use of ice are chiefly to be recommended, and also local blood-letting—dry cups, or leeches, where there are severe initial symptoms of irritation. Under some circumstances we may try to remove a part of the blood by lumbar puncture, and thus relieve the pressure on the cord. [When the diagnosis of hæmorrhage into the meninges is reasonably certain, an attempt should be made to relieve the condition by opening the spinal canal and removing the clot.—K.] If permanent disturbances be left, they should be treated by the ordinary methods—iodide of potassium, baths, and electricity.

We must here speak of **pachymeningitis interna hæmorrhagica** as a special form of disease, which usually occurs at the same time with hæmatoma of the cerebral dura mater (*vide infra*), and is precisely analogous to it in its ætiology and pathological anatomy. Encapsulated collections of blood are found on the inner surface of the dura; these often have a considerable circumference, and contain blood already disintegrated, detritus, hæmatoidine crystals, etc., since they are usually of old standing. Besides this, there are also the signs of a fibrinous inflammation—as in the cerebral dura—which, according to the opinion of most observers, is the primary process, so that the hæmorrhages into the newly formed false membrane are secondary. The symptoms of the affection—which has been observed chiefly in the chronic insane (general paralytics) and drunkards—are rarely pronounced, and consist chiefly of pain in the back, stiffness of the vertebræ, and some signs of compression on the part of the nerve-roots and the cord; but we can very rarely make a definite diagnosis.

CHAPTER II

PRELIMINARY REMARKS ON THE LOCALIZATION AND TOPICAL DIAGNOSIS (SEGMENTAL DIAGNOSIS) OF DISEASES OF THE SPINAL CORD

THE pathological processes that take place in the spinal cord may be divided into two groups, according to their localization. In the first group the anatomical changes are limited with remarkable regularity to definite neurone territories which are associated both anatomically and physiologically. These diseases are called *system diseases*, since the entire group of functionally associated neurones is called a "system." We may, in all probability, conceive that these affections arise in one of two ways: either certain neurone systems in some individuals have from the outset a congenital (inherited) weak and morbid predisposition, and, in consequence thereof, they are not able to perform their functions, but atrophy prematurely; or, that certain external, usually toxic, influences exert their morbid action, not on the whole nervous system or fortuitous circumscribed parts thereof, but only on certain definite neurones (cells or fibers)—"elective injuries." The latter hypothesis finds a fitting and instructive example in the behavior of many well-known poisons (curare, strychnine, morphine, lead, etc.), which injure only certain very definite neurones and parts of nerves, while they leave all other neurones completely intact. According as the disease involves one or more neurone systems we speak of *simple* or *combined* system diseases. Spinal muscular atrophy, amyotrophic lateral sclerosis, etc., are due to disease of the motor neurones in the cortico-muscular tract; they are simple system diseases. Hereditary ataxia, on the other hand, is an example of a combined system disease, since in this usually not only certain peripheral sensory neurones but also motor neurones undergo simultaneous degeneration. Since the processes of many neurones extend, on the one hand, from the spinal cord or spinal ganglia into the peripheral nerves, and, on the other, from the cord to the brain, and *vice versa*, we can not, strictly speakng, class the systematic neurone diseases with diseases of the spinal cord in their limited sense. Only for practical and, in part, purely conventional reasons do we consider the system diseases in this section.

In distinction from the *system diseases* there is a second group of diseases of the spinal cord where either there is no such limitation of the anatomical process to definite neurone territories, or the limitation is only very slight. In these cases the disease extends a greater or less distance transversely and longitudinally in the cord, forming either one large focus of disease of irregular outline, or many smaller, irregularly disseminated foci. To this group, the *unsystemic, diffuse diseases* of the spinal cord, belong all the traumatic destructions, also the hæmorrhages, new growths, and inflammations of the spinal cord (myelitides), multiple sclerosis, etc.

e diagnosis of diseases of the spinal cord is usually, first of all, a topical osis. From the symptoms present in the individual case we try to deter- first that *place* in the spinal cord where an affection must be situated to in the given symptoms. By appraising all the existing symptoms of dis- and also all the functions that are still in normal activity, we can deter- whether the disease is limited to one definite neurone system or whether it spread diffusely over a larger portion of the spinal cord. Only after the l diagnosis of the disease is thus established do we try to form a judgment the *nature* of the disease by connecting it with well-known typical forms ease or by considering the whole course of the disease and any accompany- mptoms.

e topical diagnosis naturally presupposes an accurate knowledge of the ions of all the different parts of the spinal cord—a knowledge which at at we by no means possess, but we know at least the distribution of the and sensory functions in the different portions of the cord in their main es, and upon this basis we can often determine, at least with approximate nty, the localization of the disease, the so-called segmental diagnosis, in the verse lesions of the spinal cord.

In regard to the localization of *motor functions* we know definitely that the fibers of the anterior roots all arise from the ganglion-cells of the anterior a. To each individual muscle belongs a group of cells in the anterior of the cord, from which the corresponding motor nerves, which innervate uscle, arise. These different muscle nuclei, however, apparently do not lie y beside and above one another, but they encroach upon one another, so that ally their accurate definition becomes difficult. Furthermore, the fibers of uscle nucleus do not always pass out from a single anterior root, but often two or more roots. The following summary of the arrangement of the l muscle nuclei in the different segments of the spinal cord has been com- by the aid of the well-known tables of Allen Starr, Kocher, and others, but be regarded as only an approximate expression of the actual conditions. details will be changed and many gaps filled by later investigations. The les printed in italics derive the main part of their nerves from the corre- ing spinal segment.

NTS OR ANTERIOR ROOTS	MUSCLES
ervical Segment.	*Small muscles of the neck.* (Obliquus capitis superior et inferior, rectus capitis posticus major et minor.) *Sterno-hyoideus.* *Sterno-thyroideus.* } Ramus descendens hypoglossi. *Omo-hyoideus.* Thyro-hyoideus. Trapezius. Sterno-cleido-mastoideus.
rvical Segment.	Rectus capitis anticus major. Longus colli. Complexus major et minor (trachelo-mastoideus). Cervicalis ascendens. Splenius capitis et colli. *Sterno-cleido-mastoideus.* *Trapezius.*
rvical Segment.	Rectus anticus major. Longus colli. Complexus. Cervicalis ascendens. Splenius. Trapezius.

	Deltoideus (N. axillaris). *Biceps.* *Coraco-brachialis.* } N. musculo-cutaneus. *Brachialis internus.* *Supinator longus et brevis.* Diaphragma. Pars clavicularis pectoralis majoris. Serratus anticus major. Teres minor. Latissimus dorsi.
6th Cervical Segment.	*Serratus anticus major* (N. thoracicus longus). *Pectoralis major et minor.* *Latissimus dorsi.* *Subscapularis.* *Teres major.* *Triceps.* *Pronator teres et pronator quadratus.* Biceps, brachialis internus. Pars clavicularis pectoralis majoris. Extensors of the hand and fingers. Scaleni.
7th Cervical Segment.	*Extensors of the hand.* (Radiales externi, s. extensor carpi radialis brevis, ulnaris externus, s. extensor carpi uln *Flexors of the hand.* (Radialis internus, s. flexor carpi radialis, ulnari s. flexor carpi ulnaris.) Extensors of the fingers. Pronators of the forearm. Portio sterno-costalis pectoralis majoris. Latissimus dorsi. Subscapularis, teres major.
8th Cervical Segment.	*Long extensors of the fingers.* (Extensor communis digitorum, extensor pollici brevis, extensor indicis.) *Long flexors of the fingers.* (Flexor digitorum sublimis et profundus, flex longus.) Small muscles of the hand.
1st Dorsal Segment.	*Small muscles of the hand and fingers.* (thenar, hypothenar.) Long extensors of the thumb. *Dilatator pupillæ* (to N. sympatheticus, *vide* p.

SEGMENTS OR ANTERIOR ROOTS	MUSCLES
2d Lumbar Segment.	*Cremaster.* Ilio-psoas.
3d Lumbar Segment.	*Ilio-psoas* (psoas, iliacus internus). *Sartorius.* *Adductors* of the thigh. *Pectineus.*
4th Lumbar Segment.	*Extensor cruris quadriceps.* Obturator externus? Gracilis, adductores.
5th Lumbar Segment.	*Glutæus medius et minimus.* *Semimembranosus, semitendinosus.* *Biceps femoris.* Tensor fasciæ latæ. Dorsal extensors of the foot and toes.
1st Sacral Segment.	*Pyriformis.* *Obturator internus.* *Gemelli.* *Quadratus femoris.* } Outward rotators of the thigh. *Dorsal extensors of the foot and toes.* (Tibialis anticus, peronei, extensor digitorum communis.)
2d Sacral Segment.	*Flexors of the foot* (gastrocnemius, soleus). Flexor digitorum communis, flexor hallucis longus. Tibialis posticus. Small muscles of the foot. *Erection.*
3d Sacral Segment.	*Perineal muscles.* *Ejaculation* (ischio-cavernosus, bulbo-cavernosus).
4th Sacral Segment.	*Sphincter et detrusor vesicæ.*
5th Sacral Segment and Coccygeal Segment.	*Sphincter ani,* levator ani.

Considering the muscles with regard to their function, we obtain the following table of the motor functions of the spinal segments:

CERVICAL CORD

1st Cervical Segment: Turning the head. Bending the head backward.
2d and 3d Cervical Segments: Bending the head forward. Raising the shoulders.
4th Cervical Segment: Inspiration. Drawing the shoulders backward.
5th Cervical Segment: Raising and outward rotation of the upper arm. Flexion and supination of the forearm.
6th Cervical Segment: Adduction and inward rotation of the upper arm. Extension and pronation of the forearm.
7th Cervical Segment: Flexion and extension of the hand.
8th Cervical Segment: } Flexion and extension of the fingers. Movement of the thumb.
1st Dorsal Segment: }

LUMBAR CORD

2d and 3d Lumbar Segment: Flexion and adduction of the thigh.
4th Lumbar Segment: Extension of the lower leg.
5th Lumbar Segment: Abduction and inward rotation of the thigh. Flexion of the lower leg.
1st Sacral Segment: Extension and outward rotation of the thigh. Dorsal extension of the foot.
2d Sacral Segment: Plantar flexion of the foot. Toe muscles.—Erection.
3d Sacral Segment: Ejaculation.
4th Sacral Segment: Bladder.
5th Sacral Segment: Rectum.

If there is disease of the anterior horns of the gray matter (poliomyelitis, syringomyelia, hæmatomyelia) or compression of the anterior motor roots, the muscles paralyzed will correspond directly to the affected segments; but with more extensive transverse disease (myelitis, tumors, compression) of course those muscles will also be paralyzed whose nuclei lie below the seat of the lesion, since the fibers of the pyramidal tract leading to them will be interrupted. From what has already been said (page 851), we may readily distinguish the nuclear or root paralyses from the pyramidal (lateral) tract paralyses by the greater atrophy, and especially by the electrical reaction of degeneration.

2. The distribution of the *sensory* conducting path to the different segments of the spinal cord corresponds in general to the territories of distribution of the sensory fibers arising from each spinal ganglion (*vide* page 796 *et seq.*); but since the outgoing posterior root-fibers are mixed with one another in the plexuses, each region of the skin is supplied by sensory fibers from at least two and probably three spinal segments, or from two or three posterior spinal roots. The accompanying diagram, devised by Kocher (Table 1), gives, as far as our present knowledge admits, a good representation of the extent of the different root territories. We must be prepared for individual variations. We see that in the arms the radial region is innervated from the upper (fourth and fifth) roots of the brachial plexus and the ulnar region from the lowest roots (first, and probably second

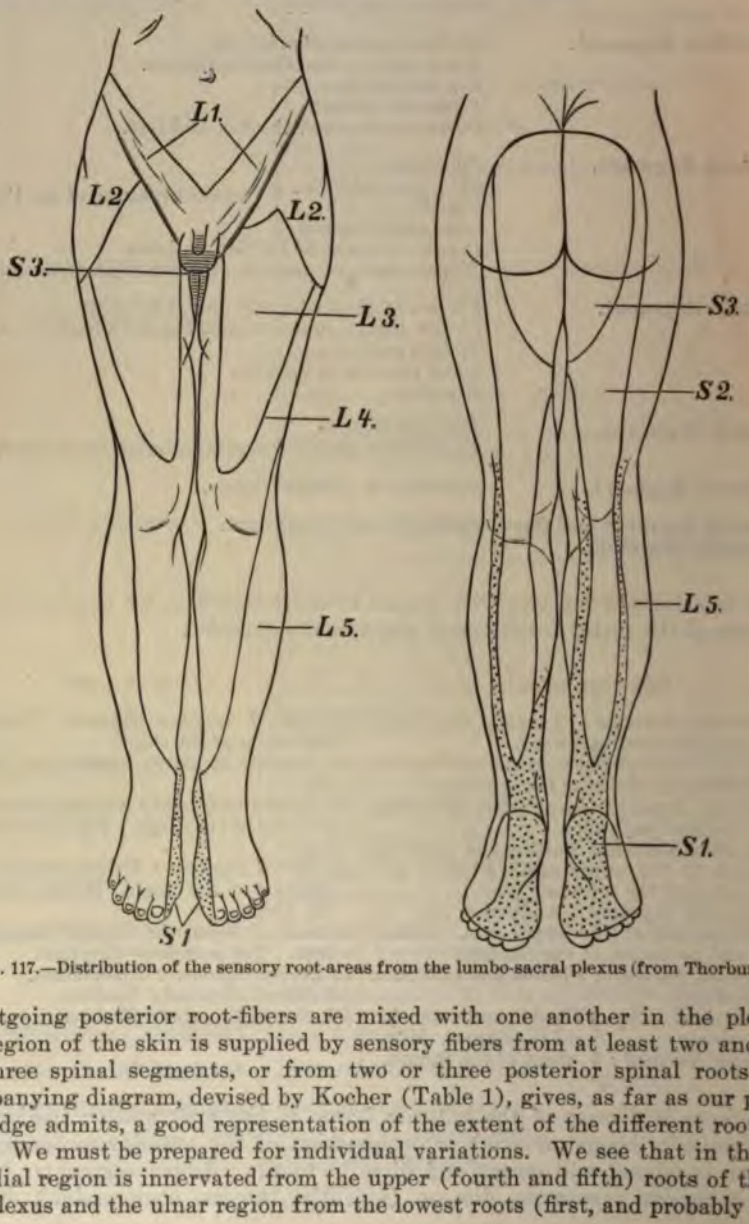

FIG. 117.—Distribution of the sensory root-areas from the lumbo-sacral plexus (from Thorburn).

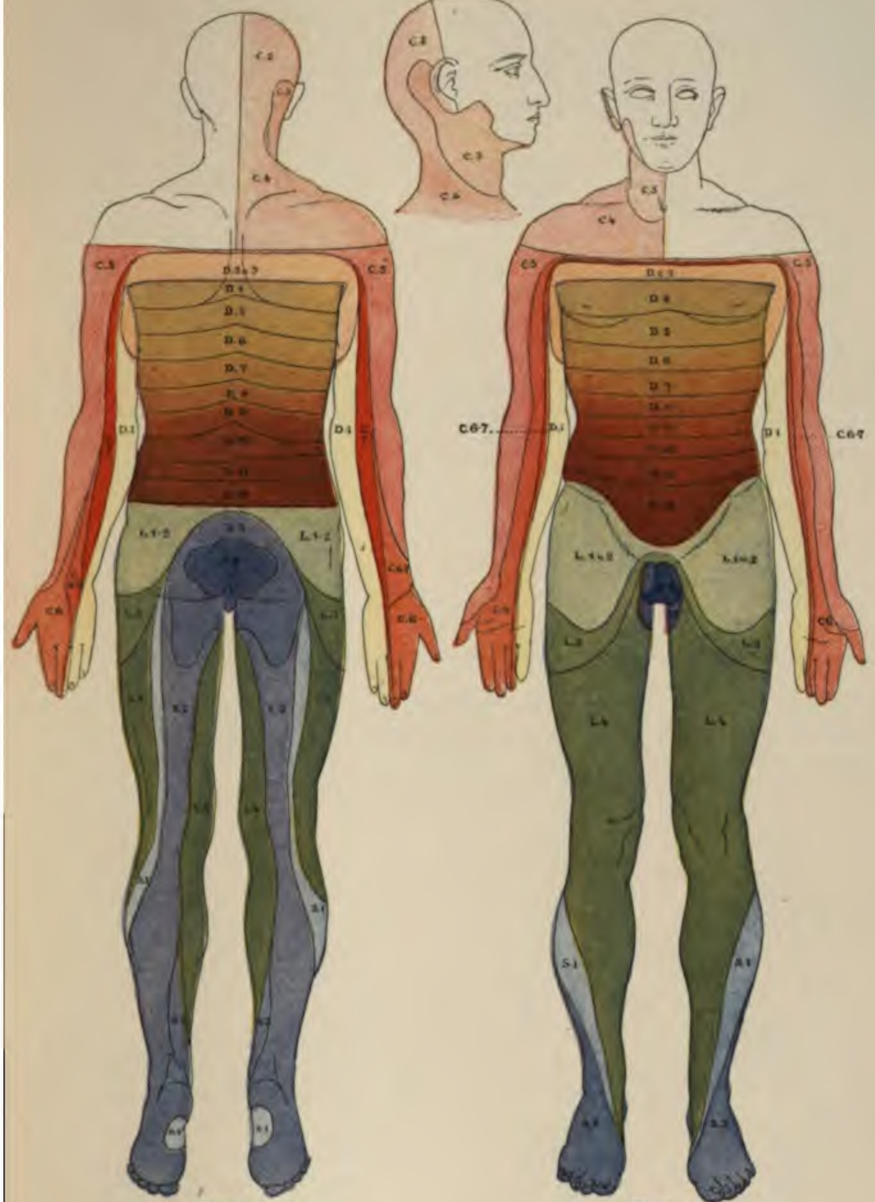

Fig. 2. *Fig. 1.*

Distribution of the Areas of the Sensory Roots upon the Surface of the Body (from Kocher).

Red: Area of the cervical roots (C. 2 to C. 7).
Yellow: Area of the dorsal roots (D. 1 to D. 12).
Green: Area of the lumbar roots (L. 1 to L. 4).
Blue: Area of the sacral roots (S. 1 to S. 4).

dorsal roots). If we conceive of the arm raised horizontally with the thumb directed upward, we can in a way imagine a horizontal segmental division of the arm according to the different sensory-root areas. On the trunk the segmental arrangement of the different sensory root-areas is very plain; although the segments do not run parallel to the ribs and intercostal nerves, but they are strictly horizontal. In the legs Kocher's diagram fails to give the area of distribution of the fifth sensory lumbar root. We therefore add for comparison the diagram given by Thorburn for the distribution of the sensory root-areas from the lumbosacral plexus (Fig. 117). The two diagrams differ considerably from each other, so that further investigations are very desirable. Figs. 118 to 124 (from work thor's clinique by L. R. Müller) show summarily the distribu-

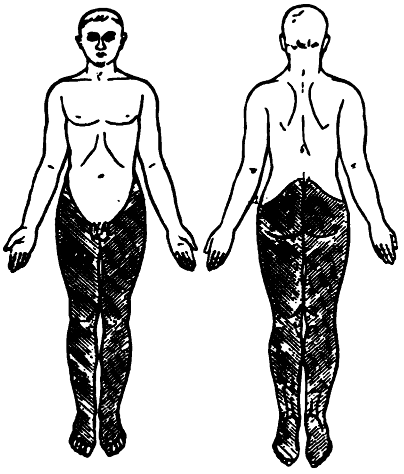

Fig. 118.
Lesion at the level of the second lumbar segment.

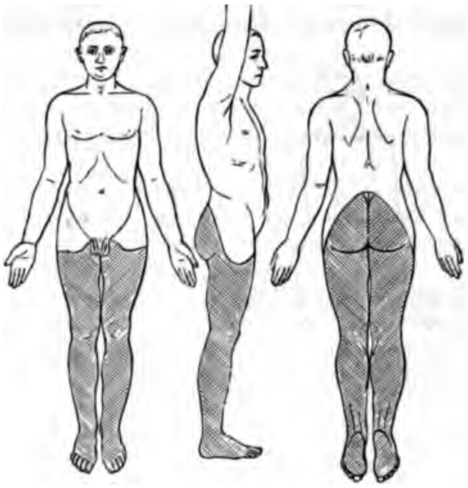

Fig. 119.
Lesion at the level of the third lumbar segment.

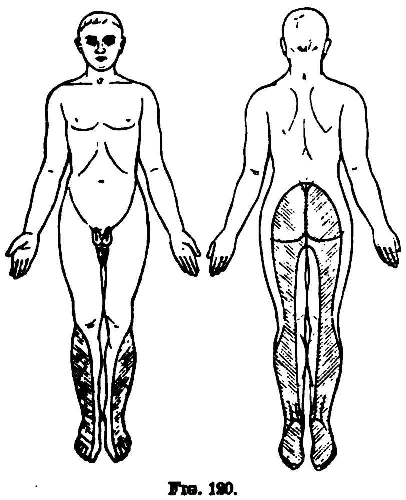

Fig. 120.
Lesion at the level of the fifth lumbar segment.

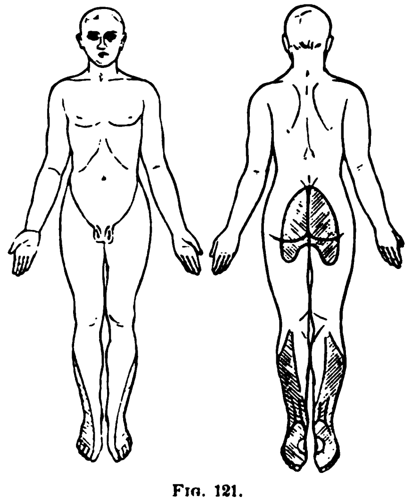

Fig. 121.
Lesion of the first sacral segment.

tion of the anæsthetic (shaded) areas of the skin in transverse lesions of the spinal cord at different levels of the lumbo-sacral cord.

In subsequent chapters we will return to many details in regard to the significance of disturbances of sensation in accurate segmental diagnosis. We can here speak of only two important points. Symptoms of sensory irritation are regarded in spinal diseases almost exclusively as root symptoms, due to an irritation of the affected posterior roots by pressure, laceration, etc. Such symptoms of irritation, therefore, are seen chiefly in diseases which arise in the coverings of the spinal cord (the vertebræ and the spinal meninges). Then we must also refer once more to the circumstance mentioned on page 799, that the tracts for the conduction of sensations of pain and temperature after their entrance into the cord probably go at once to the gray matter of the posterior horns, while the simple sensations of touch are conducted to the brain through the white posterior columns. If we find, therefore, in spinal diseases the so-called dissociated anæsthesia, especially anæsthesia for pain and temperature with retained sensibility for touch, we may be quite certain that there is a central lesion in the posterior gray cornua, such as is found especially in syringomyelia, injury of the cord, and central glioma.

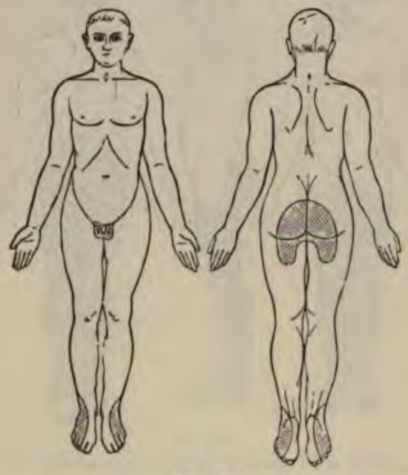

Fig. 122.
Lesion of the second sacral segment.

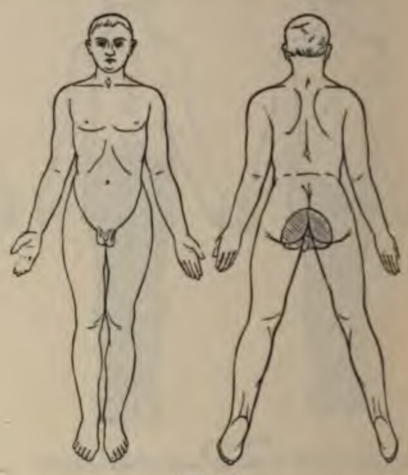

Fig. 123.
Lesion of the third sacral segment.

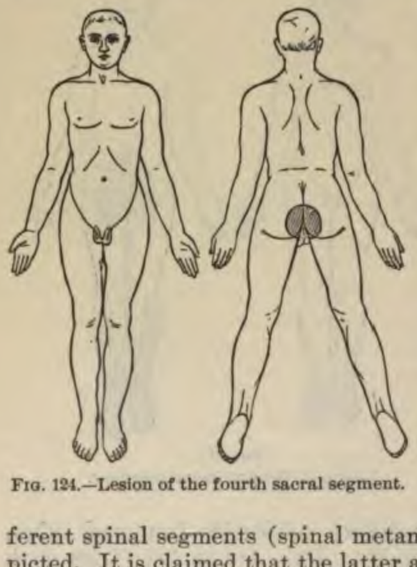

Fig. 124.—Lesion of the fourth sacral segment.

[Brissaud and other recent French writers lay much stress upon the difference between the sensory areas supplied by the spinal roots (radicular metamerism) and the sensory areas supplied by the different spinal segments (spinal metamerism). The former have already been depicted. It is claimed that the latter areas surround the limb, their boundaries being perpendicular to its long axis. They correspond, therefore, to areas covered by a glove, a sleeve, a stocking, etc. (geometrical anæsthesia). The determination of the areas of anæsthesia may therefore be of importance in deciding whether disease affects merely the spinal roots or the substance of the cord. Dejerine, however, denies any spinal metamerism, and various German observers claim that in syringomyelia, where the central gray matter is affected, the areas of anæsthesia correspond to the root distribution as given above, and are not arranged in bands about the limb; that there is no difference between radicular and spinal metamerism, and that lesions of any spinal segment cause practically the same distribution of anæsthesia whether the roots or the central gray matter be involved. The question is still unsettled, and further study is necessary.—K.]

3. The presence or absence of certain *reflexes* may also aid the segmental diagnosis. Of course the conditions here are often very ambiguous, since the reflex may be absent either from an interruption of its particular centripetal or centrifugal roots or from an interruption in any part of the reflex arc itself. The centripetal and centrifugal portions of the reflex arc are not always necessarily at the same level. Our knowledge is by no means absolutely definite as to the position of the reflex arc itself. The statements as to the relation of definite segments of the cord to certain reflexes therefore refer in general only to afferent (centripetal) posterior nerve-roots involved in such reflexes. The assumption that the transmission of the irritation to the appropriate motor ganglion-cells takes place in the same corresponding segment of the spinal cord, is warranted at most for certain simple mono-muscular reflexes (patellar reflex, abdominal reflex, etc.). For more extensive reflexes (general flexion reflex in the leg, etc.) we must assume from the outset that the reflex process spreads over many segments of the spinal cord.

We have set forth the relations of the spinal segments (or posterior roots) to the different reflexes in the following table, which represents our present knowledge of the subject, although it is still very uncertain:

Biceps tendon reflex.	5th cervical segment ?
Triceps tendon reflex.	6th cervical segment ?
Tendon reflexes in the forearm.	7th and 8th cervical segments ?
Upper abdominal reflex.	8th and 9th dorsal segments.
Middle and lower abdominal reflex.	10th to 12th dorsal segments.
Cremaster reflex.	1st to 3d lumbar segments.
Patellar reflex.	2d to 4th lumbar segments.
Glutæal reflex.	4th and 5th lumbar segments.
Plantar flexor reflex.	1st and 2d sacral segments.
Achilles tendon reflex.	2d sacral segment ?

Many details as to the behavior of the reflexes in different spinal diseases will be mentioned in later chapters. We may here state briefly that in general, the absence of a reflex points to a lesion of the corresponding segment of the spinal cord. Transverse diseases above any given reflex arc are usually (but not always, *vide infra*) associated with a pronounced increase of the reflex. In diseases of the lumbar cord the tendon reflexes in the lower extremities are therefore lost as a rule, while in diseases of the dorsal and cervical cord they are usually much increased.

4. At this point we must mention some important anatomical relations between the position of the different portions of the spinal cord and the surrounding vertebræ. It is well known that the lower end of the spinal cord extends only to the level of the second lumbar vertebra. The vertebral canal below this point is filled partly by the filum terminale, but chiefly by the bundles of roots of the cauda equina. The greatest breadth of the cervical enlargement is at the level of the fifth and sixth cervical vertebræ. The dorsal cord begins at the level of the

second dorsal vertebra, the lumbar cord at the level of the tenth dorsal vertebra. The lumbar enlargement attains its greatest circumference at the eleventh dorsal vertebra. The position of the conus terminalis corresponds to the upper half of the second lumbar vertebra.

The topical relations between the different spinal segments and spinal roots and the bodies of the vertebræ are also of great practical importance. Since the spinal segments are shorter in the vertical direction than the bodies of the vertebræ, the spinal roots, from above downward, must travel farther and farther downward to reach their corresponding intervertebral foramina. The accompanying diagram from Gowers (Fig. 125) makes this relation plain. A lesion of the eighth dorsal segment, for example, lies opposite the body of the seventh, and not of the eighth dorsal vertebra; but since the spinous process also has a descending direction, the lesion must be sought at the level where we can feel the seventh or even the sixth spinous process. These relations have great practical significance for any operative procedure, but we must also remember that there may be individual variations from the rule.

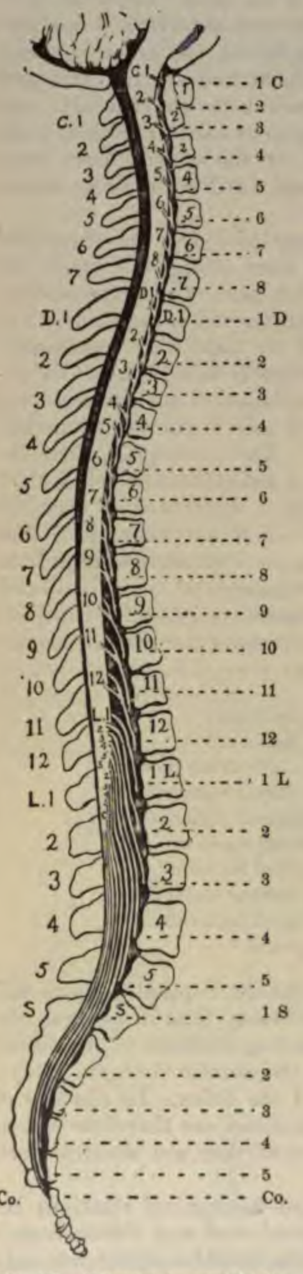

FIG. 125.—Diagram to explain the relation between the points where the different spinal roots are given off from the cord and the different vertebral bodies and spinous processes.

CHAPTER III

DISTURBANCES OF CIRCULATION, HÆMORRHAGES, TRAUMATIC LESIONS, AND FUNCTIONAL DISTURBANCES OF THE SPINAL CORD

1. **Disturbances of Circulation.**—Our knowledge as to the occurrence and as to the clinical significance of pure disturbances of circulation in the spinal cord is very slight. Most that is stated in regard to them in the older descriptions of the pathology of the spinal cord corresponds much more to theoretical hypotheses than to the actual objective facts.

It goes without saying that a complete anæmia of the spinal cord must destroy its function; this fact is best illustrated by the well-known experiment of Stenson: if we compress the abdominal aorta of an animal, and thus cut off the blood-supply to the lumbar cord almost completely, a paralysis of the posterior portion of the body very rapidly ensues. Embolic (or thrombotic) obstruction of the aorta also leads to flaccid paralysis of the legs in man, but this paraplegia is to be referred to anæmia of the peripheral nerves and muscles, and not to any change in the spinal cord, as we must maintain after a personal observation upon this point. The spinal cord is abundantly supplied with blood by the spinal arteries coming from the vertebral artery. Some precisely analogous observations have been made in man in the rare cases of obstruction of the aorta by emboli or thrombi. Pronounced spinal symptoms in general anæmia, which may be referred to a co-existing anæmia of the cord, are rare, and at any rate are of much less clinical prominence than the important results of a co-existing cerebral anæmia (*vide infra*). In only a few cases has paraplegia been seen after a great general loss of blood, as after metrorrhagia or intestinal hæmorrhage. The severe spinal symptoms sometimes occurring in pernicious anæmia are due to combined columnar degeneration in the cord (*vide infra*).

All statements as to the occurrence and clinical significance of hyperæmia of the spinal cord are still more dubious. We do not know whether there is active hyperæmia of the cord. The hyperæmia from stasis, in general disturbances of the circulation, in which certainly the spinal cord often takes part, causes no especially marked symptoms.

2. **Hæmorrhage into the Substance of the Spinal Cord—Spinal Apoplexy—Hæmatomyelia.**—Primary hæmorrhage into the spinal cord is as rare as hæmorrhage into the brain is common. It is due most frequently to traumatic influences (fall on the buttocks, blow on the back, etc.). We will return to this traumatic hæmatomyelia later. The sudden onset of spinal paralysis has also been seen after great physical exertion, and this is probably due to a spinal apoplexy. It is possible, but it is not yet absolutely certain, that primary vascular disease (small aneurisms) may favor the occurrence of hæmorrhage in such cases. Small capillary spinal hæmorrhages are not infrequently seen as a secondary complication in tumors of the cord, and in inflammatory affections such as myelitis, epidemic meningitis, etc., and in the general hæmorrhagic diathesis, as in scurvy or severe general infectious diseases, and after great congestion, convulsions, etc. F. Schultze has also found hæmorrhages into the gray matter of the posterior horns in new-born infants who have come into the world asphyxiated after difficult labor, and these hæmorrhages may perhaps be of some significance in later life.

Anatomical experience in regard to primary spinal apoplexy is still quite slight. The commonest situation for spinal hæmorrhage is the gray matter, either in the cervical or the lumbar enlargement. If the hæmorrhage be abundant, we find the substance of the cord destroyed to a great extent. The apoplectic center usually extends principally in the long axis of the cord. The blood is still fluid in fresh cases. Later on it undergoes all those changes which are fully described in the chapter on cerebral apoplexy. It is not improbable that some cases of syringomyelia may be referred to a primary hæmorrhage into the spinal cord.

The symptoms of spinal apoplexy must in the first place depend entirely upon the seat and extent of the hæmorrhage. The sudden, apoplectiform onset of the symptoms is always characteristic. It usually begins with severe pain in the back, followed very speedily by more or less widespread paralysis, anæsthesia, vesical disturbances, etc. Since the hæmorrhage is usually situated in the gray matter, the localization of the muscular paralyses and the form of sensory disturbance (dissociated anæsthesia!) often show the peculiarities attendant upon such a lesion. Hæmorrhage into one half of the cord sometimes shows very definite symptoms of unilateral lesion (*vide infra*). Thus in some cases of hæmorrhage into the cervical cord, in particular, Minor has observed the following symptom-complex: partial atrophic paralysis of one arm, spastic paralysis of the leg on the same side, partial anæsthesia (just as in syringomyelia) in the extremities of the opposite side.

The course of hæmorrhage of the cord may in many cases be comparatively favorable. If the blood be absorbed, and essential paths of conduction be not permanently destroyed, the symptoms of paralysis gradually pass away, and recovery or at least improvement and an arrest of the symptoms follow. In many cases, of course, the severe type of spinal paralysis develops, with bed-sores, cystitis, etc., and this, after a longer or shorter time, leads to death.

We must always be very guarded in making a diagnosis of spinal hæmorrhage. We can do so with some probability only when the symptoms begin in a pronounced apoplectiform manner, and when definite ætiological factors (trauma) are to be made out; but, as we shall see later, central softening without actual hæmorrhage may follow trauma and cause precisely the same symptoms as hæmatomyelia. We should also not forget that many forms of acute myelitis, and even chronic spinal affections (especially spinal syphilis), may also show a remarkably sudden onset or at least may suddenly become worse. The distinction between genuine spinal apoplexy and meningeal hæmorrhage is often difficult. Initial symptoms of marked sensory irritation favor meningeal hæmorrhage, while the occurrence of symptoms pointing to an affection of the gray matter, especially the combination of atrophic muscular paralysis with partial anæsthesia (thermoanæsthesia, analgesia), warrants the suspicion of central hæmatomyelia.

Treatment.—If we have the rare opportunity to be able to interfere at the beginning of the symptoms, we should prescribe a perfectly quiet position in bed, local use of ice, and eventually ergotine. Later on the treatment should be directed according to the methods generally in use in spinal paralysis.

3. **Traumatic Lesions of the Spinal Cord.**—In spite of the protected position of the spinal cord, it is often the seat of severe acute traumatic lesions. Fractures and dislocations of the vertebræ are the most frequent, and these may give rise to considerable injury of the spinal cord by the dislocation of the vertebræ, or by the projection of a fragment of bone. Dislocations occur most frequently in the cervical region (between the atlas and axis or very often between the fifth and the sixth cervical vertebræ). The upper vertebra in such cases is usually dislocated forward. The spinous process of the lower vertebra therefore projects backward, while the spinous process of the dislocated vertebra is pushed forward. The head is bent forward. Every movement of the head is painful, and is therefore anxiously avoided. Fractures are very often situated in the middle cervical vertebræ or at the twelfth dorsal and first lumbar vertebræ.

Besides the indirect lesions of the cord by displaced vertebræ, the action of the injury (fall on the back, etc.) may also extend immediately to the spinal cord. In many cases there may be at the moment of the injury a temporary distortion of the vertebral column associated with local laceration of the cord; but sometimes severe traumatic lesions of the cord occur without any vertebral injury. These consist of hæmorrhages, which are usually central (hæmatomyelia (*vide supra*), or traumatic softening, which is also usually central. Hæmorrhage and softening may also be associated. Some writers hold that an effusion of cerebrospinal fluid may sometimes cause laceration and injury of the tissues. The different actions of the traumatic force are, of course, very often associated; thus we not infrequently see, in particular, above and below the destruction of the cord caused by a vertebral lesion, central hæmorrhages, or softening in the anterior and posterior horns.

Gun-shot wounds of the cord are quite frequent, in which the bullet either penetrates the cord itself or produces injury of the vertebræ and hæmorrhage, which involve the cord indirectly. Stabs and incised wounds of the cord have been repeatedly seen. The point of a knife or sword may penetrate the spinal canal from behind through the soft parts and cause a partial section or at least a contusion of the cord. In such cases a secondary "traumatic inflammation" may be added to the direct injury.

The symptomatology of injuries of the spinal cord follows the general rules for the localization of disease of the cord laid down in the previous chapter. There is usually at first a pronounced and often complete motor paralysis of the lower extremities. There is also anæsthesia, and very often vesical and rectal paralysis. In many severe cases the secretion of urine seems at first much diminished or wholly wanting. If the spinal roots be affected there are severe shooting pains and paræsthesia. Above the limit of total cutaneous anæsthesia we sometimes find a zone with dissociated anæsthesia (analgesia and thermo-anæsthesia). This symptom points to a hæmatomyelia or a central softening in the corresponding posterior cornu of the gray matter above the seat of the special lesion (*vide supra*). The reflexes are usually diminished at first, but, later, if the injury be above the reflex arc, they are increased; but if the arc itself be broken, they are permanently absent. This rule, however, undergoes an interesting exception, since we often find after severe injury of the cervical cord, and especially after a complete transverse lesion thereof, a flaccid paralysis of the lower extremities and complete absence of the tendon, and more rarely of the cutaneous, reflexes (Bastian, Thorburn, Bruns, and others). To explain this striking fact we must suppose that the reflex path in the lumbar cord has also undergone some disturbance, either from the initial general concussion or from its separation from all the higher parts in the brain. With the loss of reflex is often associated paralysis of the bladder and rectum. We often see in men, especially after injury of the cervical cord, a more or less complete and persistent erection of the penis, which is probably due to a direct or reflex irritation of the nerves of erection. In injuries of the cervical cord particularly, we often see a great and general increase of temperature, up to 110° or 112° (43°–44° C.), especially in severe and rapidly fatal cases; this is of physiological interest, and agrees with the results of experiments. On the other hand, there are also, especially in injuries of the dorsal cord, as it seems, great falls of temperature, down to 90° or 86° (32°–30° C.).

The further course of the affection differs very much. In the worst cases death ensues in a few hours or days. In other cases the patients recover from the first "shock," but permanent paralysis remains, which may sooner or later lead to death from the ensuing sequelæ, cystitis and bed-sores; but we often see partial improvement and a cessation of all the symptoms. Although certain functional disturbances remain permanently, life is not further endangered. Finally, in comparatively mild cases, there may be a complete recovery.

The very characteristic injuries of the conus terminalis and the cauda equina yet demand special mention. The conus terminalis is sometimes crushed in fracture of the first lumbar vertebra. The symptoms consist of paralysis of the bladder, rectum, and sexual functions, with anæsthesia of the skin over the sacrum and about the anus, perineum, and genitals (cutaneous branches of the last sacral nerves, compare Fig. 117, page 924). If the injury extends higher there are also sensory disturbances on the posterior aspect of the lower extremities, and paralysis in the region of the sciatic (see Figs. 118–124, pages 925, 926). A precisely similar type of disease, however, also develops from injury of the cauda equina (for example, from fracture of the lowest end of the vertebral column or fracture of the sacrum), since injury of the root-fibers of the sacral and coccygeal plexuses must of course cause the same symptoms as injury of the corresponding spinal segments themselves. In such cases we also see the peculiar distribution of the sensory disturbances and the paralyses localized in the sciatic region, especially the double peroneal paralysis that is so very characteristic. The differential diagnosis between an affection of the cauda and a lesion of the lowest portion of the cord is not always easy, but it is of practical importance, especially in regard to the question of any surgical interference. Besides the consideration of the chief point of action of the injury, we must consider especially that symptoms of severe sensory irritation (shooting pains, paræsthesia) favor in general an affection of the cauda, while the existence of dissociated disturbance of sensation is of value in assuming a (central) disease of the spinal cord.

The treatment of the primary affections belongs to the domain of surgery; especially any attempt to lay open the vertebral canal, in order, if possible, to relieve the existing pressure on the cord by reducing the dislocation of the vertebræ or the splinters of bone. In most cases, particularly in all cases where the injury is confined to the cord, we have to confine ourselves to putting the patient in a proper position on a water-bed, and guarding as carefully as possible against bed-sores and cystitis. Locally, the constant application of ice is most to be recommended. We can expect but little from local blood-letting, inunctions with mercurial ointment, etc. If the first acute stage pass off favorably, the treatment of any paralytic symptoms remaining follows the ordinary rules (baths and electricity).

4. **Diseases of the Spinal Cord after a Sudden Reduction of the Atmospheric Pressure [Caisson Disease].**—In bridge-builders and others, who have worked for hours under water in the so-called "caissons," under an external pressure of two or three atmospheres, we sometimes observe peculiar nervous symptoms after they leave the caisson—that is, on the sudden reduction of the atmospheric pressure. These symptoms do not come on immediately after the change to the ordinary atmospheric pressure, but usually some minutes or even half an hour later. They progress very rapidly, so that in a few hours at the latest the disease is fully developed. Besides the frequent mild and transitory symptoms of pain in the ears and hæmorrhage from the ears, articular and muscular pains in the back and the extremities, slowing of the pulse and vomiting, vertigo, tinnitus, disturbances of vision and speech, clouding of consciousness, or even complete mental confusion, there are also more persistent and severe disturbances of motility and sensibility, which point unequivocally to an affection of the spinal cord. Usually the spinal symptoms affect the lower extremities chiefly; more rarely they also involve the arms. Paralysis of the diaphragm has also been observed. The type of disease differs much in its details; as a rule the symptoms of spastic paraplegia predominate, but in other cases there are marked disturbances of sensation or co-ordination. The bladder is often involved; there is usually retention of urine. The patient sometimes recovers in a few weeks, but in other cases the condition terminates fatally in a comparatively short time—in a few weeks or months. Anatomical investigations (Leyden, F. Schultze, von Schrötter, and others) show in such cases a disseminated but extensive affection in the dorsal cord, chiefly in the posterior columns and the posterior portions of the lateral columns. The nervous tissue in the diseased parts is completely destroyed, and instead of it is found detritus and a collection of large, round, fatty granular cells. Hæmorrhages into the cord, which might perhaps be expected, have not been seen. The cause of these small multiple foci of softening is to be found in the occurrence of many gas emboli. During the workman's stay in the compressed air the blood absorbs an abnormal amount of gas, especially nitrogen (Hoppe-Seyler, P. Bert, and others). With too sudden a return to the ordinary conditions of atmospheric pressure, bubbles of gas form in the blood, and, in case they can not be thrown off fast enough by the lungs, they lead to numerous emboli in the smaller vessels with their consequences. The anatomical arrangement of the vessels seems to be the reason why the embolic foci are situated chiefly in the dorsal cord.

The treatment is the same as in acute myelitis. The prophylactic measures to be employed in a given case are of course very important. The onset of morbid symptoms can be certainly avoided by the cautious and slow reduction of the atmospheric pressure. [No person suffering from any disease, especially of the heart or kidneys, no one addicted to alcohol, and no old or very stout person, should enter a caisson. It is probably safer not to enter a caisson fasting. Beginners should remain in the caisson a comparatively short time—not over an hour. For more experienced workmen a day's work should not exceed four hours, divided into two shifts with four hours' interval. Great precautions must also be taken against cold. The process of "unlocking" must be slow, at least five minutes for each atmosphere of pressure. It is said that when the symptoms first come on they may be averted by restoring the victim to the caisson or to a cabinet capable of affording increased atmospheric pressure. Fluid extract of ergot (a drachm [gramme 4.0] every hour for a few doses) is said to give great relief at the outset.—K.]

5. **Functional Disturbances (Spinal Irritation—Spinal Neurasthenia).**—In practice we very often see cases where the patient complains of a set of symptoms which are in all probability of spinal origin; but since all the objective signs of a severe spinal affection are entirely absent, and since the whole development and further course of these cases oppose the theory of a coarse anatomical disturbance in the cord, we have a right to regard them as mere "functional disturbances," and thus to express their relation to certain injurious ætiological influences, and their comparative freedom from danger. It is often very hard to judge as to the special origin of the symptoms. The hypothesis of "fine molecular changes" and also the hypothesis of "abnormal vaso-motor influences and disturbances of the circulation" are forms of speech without any actual basis. In very many cases—though of course not in all—the influence of a morbid imagination and of mental anxiety is very evident. The spinal symptoms are also very often associated with certain cerebral symptoms, so that the former are only a part of a general neurasthenia. We must therefore refer to the description of the latter.

If we look for the cause for the development of the disease, we find, as a rule, one or more of those injurious factors, which have an undoubted influence in the development of almost all the neuroses: severe and persistent emotional excitement, mental and physical over-exertion, improper methods of living, poisons, such as alcohol and nicotine, and sexual excesses, such as masturbation, etc. Besides these there is very often a hereditary predisposition, a congenitally weak resistance of the nervous system, which is often further enfeebled by a poor state of the general nutrition. Finally, a hypochondriacal disposition is of great significance, as it causes not only an abnormally increased attention to the symptoms, but also an abnormal hyperæsthesia to all subjective sensations, and even the development of new subjective sensations. The constant anxiety about the dreaded results of any excesses committed (especially with many patients the fear of the supposed effects of previous masturbation) is often much more injurious than the excesses themselves. Hypochondriacal dread, too, usually plays the largest part in the spinal neurasthenic conditions frequent among physicians (tabophobia).

The symptoms of the morbid conditions in question usually begin gradually. The patient begins to complain of weakness and fatigue in walking, and also very often of pains in the back and loins, and not infrequently in the extremities also. In spite of the vivid description which the patients give of their pain, they usually have to admit, if questioned about it closely, that the intensity of the pain is really not very great. Besides the pain there are usually many forms of paræsthesia—numbness, formication, cold feelings, etc. The more the patient knows, or at least believes he knows, of the symptomatology of spinal diseases from reading and from associating with other patients, the more detailed are his complaints. Disturbances of the bladder are usually present only in a slight degree, but still they do occur. They often depend merely upon the disturbance of the involuntary reflex mechanism, due to the added factor of increased voluntary attention. There are very often sexual disturbances (pollutions, sexual weakness, etc.), which are usually to be referred to former excesses, especially to masturbation or to the hypochondriacal condition of the patient.

If we make a physical examination of the patient we can not discover definite signs of a spinal affection. In some of the cases we find an increased sensitiveness of the vertebræ on pressure, which may be limited to a few definite spots, a symptom to which the name of "spinal irritation" is often given; but we often fail to find this tenderness of the vertebræ. Nothing abnormal is to be discovered in the pupils or the reflexes. The tendon reflexes are sometimes quite lively and sometimes weak. The sensibility is objectively perfectly normal; neither can we discover actual paresis or atrophy of the muscles. Vaso-motor disturbances, however, are often seen: abnormal coldness, pallor or redness of the hands, tendency to sweating, etc. The manifold cerebral symptoms, that are usually present at the same time, will be mentioned in the description of neurasthenia. The condition of the general nutrition in many patients remains unchanged, but, of course, some become pale, thin, and weak.

The diagnosis of functional disturbances of the spinal cord is usually not difficult, as we have said, and it may often be made from the history, from the patient's whole outward behavior, and from the manner of his complaints; but we can not lay too much stress upon the injunction that a careful physical examination should always be made, in order to avoid confusion with an incipient serious disease. We shall repeatedly call attention in what follows to the symptoms which must chiefly be observed for this purpose.

In regard to prognosis and treatment we will refer to what is said in the chapter on neurasthenia.

CHAPTER IV

THE PRESSURE PARALYSES OF THE SPINAL CORD

(Slow Compression of the Spinal Cord, especially in Caries and Carcinoma of the Vertebræ)

Ætiology.—Many pathological processes in the vicinity of the spinal cord exert a gradually increasing pressure upon it, and thus inhibit the conduction of nervous irritation, and even cause coarse organic changes in the substance of the cord. The seat of such affections is in the first place in the membranes of the spinal cord. In the chapter on meningitis we have already mentioned the compressing action of the masses of inflammatory exudation on the nerve-roots and the cord, and we have learned to recognize, especially in pachymeningitis cervicalis hypertrophica, a characteristic example of a gradually increasing compression of the cervical cord. Precisely similar conditions are found in the not very rare meningeal tumors, whose special pathology will be described in connection with the tumors of the cord itself.

By far the most frequent pressure paralyses of the spinal cord, and hence the most important practically, are caused by certain diseases of the vertebræ, and first of all by chronic caries of the vertebræ (spondylitis, Pott's disease, spondylarthrocace).[*] There is no longer any doubt that in almost all cases vertebral caries is of tubercular origin—a local tuberculosis of the vertebræ. Although these facts were formerly rendered very probable by the histological conditions of the process, and by its frequent relation to other unquestionable tubercular diseases, such as phthisis, miliary tuberculosis, and tubercular meningitis, they have of late been confirmed beyond a doubt by the almost invariable discovery of tubercle bacilli in the cheesy nodules of the vertebral caries. Tubercular spondylitis occurs at almost any age; it is rare only in old people. It often develops in children, but it is almost as frequent in adults. The ætiological significance of injuries, such as a fall or a blow, which are often mentioned by the patients themselves or their parents, is in most cases doubtful, but not wholly to be ignored. As a rule vertebral caries apparently develops quite independently as a primary disease. Of course we can very often succeed in finding ætiological factors rendering tubercular disease likely—the tubercular habit, hereditary tendency, or previous tubercular disease elsewhere, such as phthisis, pleurisy, other affections of the bones, etc.

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[*] In the kyphoscolioses of the vertebral column, not due to spondylitis, but caused by abnormal mechanical conditions or conditions of growth, there are practically never any symptoms of compression in the cord, even in very pronounced cases. In these cases the cord manifestly shows quite a great adaptability.

Cancer of the vertebræ, as well as caries, leads to pressure paralysis of the spinal cord; but it is relatively much rarer than caries, it develops chiefly in older persons, and it occurs only as a secondary new growth in cancer of other organs—the breast, the stomach, the œsophagus, etc. Sarcoma is the chief primary tumor of the vertebræ; but it may also involve the vertebræ secondarily by extension from growths in the vicinity.

We must mention here briefly, as very rare causes of compression of the spinal cord, aneurism of the aorta, which gradually erodes the vertebræ, echinococci in the vertebral canal, exostoses of the vertebræ, [myeloma,] and syphilitic new growths.

Pathological Anatomy.—Vertebral caries is by far most frequent in the dorsal portion of the vertebral column (dorsal spondylitis), less frequent in the cervical portion (cervical spondylitis), and rarest in the lumbar portion (lumbar spondylitis) and in the sacrum (sacral spondylitis). It usually extends over several adjacent vertebræ, or more rarely two separate foci of disease are seen. The process itself, the details of which can not be discussed here, probably always begins in the spongy substances of the bodies of the vertebræ. We see here, on section, in incipient cases, roundish, pale reddish, or yellowish nodules, which consist of newly formed fungous tissue—that is, tubercular granulation-tissue. The bony substances become more and more destroyed by the invasion of the new growth, which itself shows the characteristic tendency of all tubercular new growth to cheesy degeneration. Thus there is often an extensive destruction of the bodies of the vertebræ, which later on involves the vertebral processes also, the intervertebral disks, and the other articular connections between the different vertebræ.

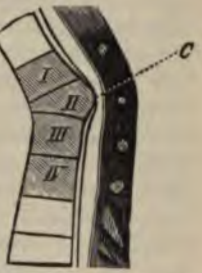

FIG. 126. — Schematic representation of vertebral displacement in spondylitis. The point of compression of the cord, at the level of the second dorsal vertebra, is at *C*.

There are essentially two factors to be considered in regard to the question which chiefly interests us here—that is, in regard to the occurrence of compression of the spinal cord. In the first place, it is clear that the complete or partial destruction of the bodies of one or even more vertebræ, and of their articular connections, can not remain without influence upon the position of the other adjacent vertebræ. In fact, we very often see dislocations of the vertebræ as a result of it, usually by the pushing backward of the partly destroyed vertebra by the movements of the vertebræ above and below the diseased portion on one another (see Fig. 126). There arises, on the one hand, a contraction of the vertebral canal, and with it often a very considerable limitation of the space for the cord; and, on the other hand, that characteristic projection of the spinous processes in the region of the diseased portion of the vertebral column which forms the so-called Pott's boss—the angular kyphosis (Fig. 127, page 938). In very slight degrees of the disease there is only a trifling projection of one or more spinous processes, but in other cases it gradually becomes an extensive deformity of the vertebral column, which strikes us at the first glance; but it must be especially borne in mind that in very many cases of vertebral caries, where the body of no vertebra is wholly or mainly destroyed, there is no knuckle formed at all. The line of the spinous processes in such cases preserves completely its normal external form.

The second factor, which is very often to be considered in the mechanism of compression of the cord, is the formation of tubercular granulation tissue or of foci of cheesy pus on the posterior surface of the bodies of the vertebræ. Since the inflammatory tubercular new growth involves the periosteum, there are often formed here large collections of cheesy pus, which are situated beneath the periosteum, raise it, and push it out far into the vertebral canal. Still more commonly, the tubercular new growth directly involves the outer surface of the dura, and forms here extensive cheesy masses which of course may also cause a compression of the cord. The inner surface of the dura at the corresponding parts is usually a little injected, but otherwise perfectly normal. A direct invasion of the inner surface of the dura or of the pia by the tubercular process through the dura is rare.

If now a greater or less contraction of the vertebral canal has arisen from dislocation of the vertebræ, or from the projection inward of the cheesy purulent masses into the canal, the consequences of pressure on the spinal cord itself are not long wanting. In cases where there has been much displacement of the vertebræ or an abundant formation of cheesy purulent masses on the outer surface of the dura with considerable narrowing of the vertebral canal, the direct and purely mechanical injury of the nervous elements certainly plays a part; but we not infrequently see during life marked paraplegia in vertebral caries, where the autopsy shows only a comparatively slight narrowing of the vertebral canal, usually from granulations on the outer surface of the dura mater. In such cases there are a number of other factors, for the most part also of a mechanical nature, which lead to destruction of the substance of the cord. In the first place the compression of the epispinal lymph-space causes a stasis of lymph (Schmaus) in the periadventitial and also in the periganglionic lymph-spaces and in the lymph-spaces between the axis cylinders and the medullary sheaths. The retained lymph has a mechanical and probably a toxic action on the nerve elements. We also see not infrequently thrombosis of the small afferent arteries, causing anæmic necrosis of their respective vascular territories. It is hard to decide how much effect may be ascribed to direct compression of the vessels; and equally hard to say whether toxines from the tubercular nodules may perhaps reach the substance of the cord, and there exert an injurious action. In no case, however, have we a right, in our opinion, based upon a comparatively large number of cases seen in hospital and private practice, to speak of a "compression myelitis." Myelitis—that is, inflammation—always implies agents of inflammation at the place of inflammation, and of that there is no suggestion in compression of the cord. The histological changes at the point of compression (*vide infra*) are not of an inflammatory nature, but they may all be explained by the above-mentioned mechanical factors.

If we find a greater narrowing of the vertebral canal, the spinal cord is also smaller at the point of compression. If the narrow part correspond to a bend in the vertebral column, we can very often see a marked angle of bending on the anterior surface of the cord. The whole configuration of the cord and the arrangement of the nervous elements may be considerably disturbed and distorted mechanically. The consistency of the cord at the part affected, the extent of which seldom exceeds a few centimetres, is usually diminished; the cord is soft and may be easily bent. In old cases only do we find the cord itself harder than normal and sclerosed (*vide infra*). It is very remarkable, however, that often, as we have repeatedly seen, marked symptoms of paralysis may be present during life without finding any coarse mechanical lesion of the cord in the cadaver, so that the cord may have an almost perfectly normal appearance. As in the peripheral nerves, so in the spinal cord, a moderate pressure is manifestly enough to excite a partial break in the conduction, without being at the same time necessarily associated with an actual mechanical destruction of nervous elements. On careful microscopic examination of the cord, we find in such cases, in spite of the existence of a complete paraplegia during life, that most of the nerve-fibers are still completely preserved, and that there are only here and there a few lacunæ, corresponding to circumscribed lymph-spaces (*vide supra*), and to single fibers which have been destroyed. This discovery is especially interesting because it makes us understand the possibility of recovery, even in apparently severe cases (*vide infra*).

If we take a portion from the soft place of compression for examination in the fresh state, we find usually many, sometimes (in old cases) only a few, granular cells, according to the amount of disintegrated nerve-medulla, the remains of which are taken up by the white blood-corpuscles (the wandering cells), and probably also by the glia cells. If we make stained cross-sections of the hardened cord, we see under the microscope no signs of hyperæmia, or accumulation of cells about the vessels, and only exceptionally a little hæmorrhage; but we do find, besides many still preserved nerve-fibers, other fibers, which are involved in the disintegration or are already destroyed. Very commonly the changes are distributed in the form of nodules. We find groups of greatly swollen axis-cylinders, or of axis-cylinders torn off and rolled up in a spiral. The medullary sheaths are everywhere involved in the disintegration, until they blend with the disintegrated axis-cylinders. The products of disintegration developing everywhere serve, as was said above, to form the granular cells. The ganglion-cells and the glia also show signs of disintegration. We not infrequently find the so-called corpora amylacea, whose origin is still somewhat obscure. If the destruction of the nervous tissue have advanced to a certain degree, there is in the later stages, as in all analogous processes, a secondary implication of the neuroglia. Now follows an increase of the interstitial connective tissue. Its proliferations, which take the place of the destroyed nervous tissue, seem broad and thick—at first loose, but later firm and fibrillary. Thus it happens that in old cases we find nothing at the point of compression but a loss of nerve-fibers in the cord, and instead a firm fibrous tissue. We can sometimes notice processes of regeneration in the nerve-fibers (*vide infra*). In general the changes are much more marked in the white matter of the cord than in the gray.

Finally, after every protracted compression of the cord we find an ascending and descending secondary degeneration of certain systems of fibers in the cord (*vide infra*).

We need not go more fully into the details of compression of the cord from other causes, since the results, as far as they are of a purely mechanical nature, are precisely the same. In cancer of the vertebræ there may also be dislocations of the vertebral column after the destruction of some of the vertebræ, but usually the compression depends upon the direct growth of the newly formed tissue into the dura. In these cases the compression of the nerve-roots in the intervertebral foramina is also of importance.

Clinical History.—1. **The Pressure Paralyses in Vertebral Caries.**—Many cases of spondylitis run their course without involving the cord, or at least they involve it only in quite a subordinate fashion. In other cases the symptoms of disease of the vertebræ exist for a long time alone, until at last, slowly or suddenly, the signs of compression of the cord are added to them. Finally, in a third group of cases, the vertebral disease is so latent that only the cord symptoms are prominent in the type of the disease, and the disease of the vertebræ is easily entirely overlooked.

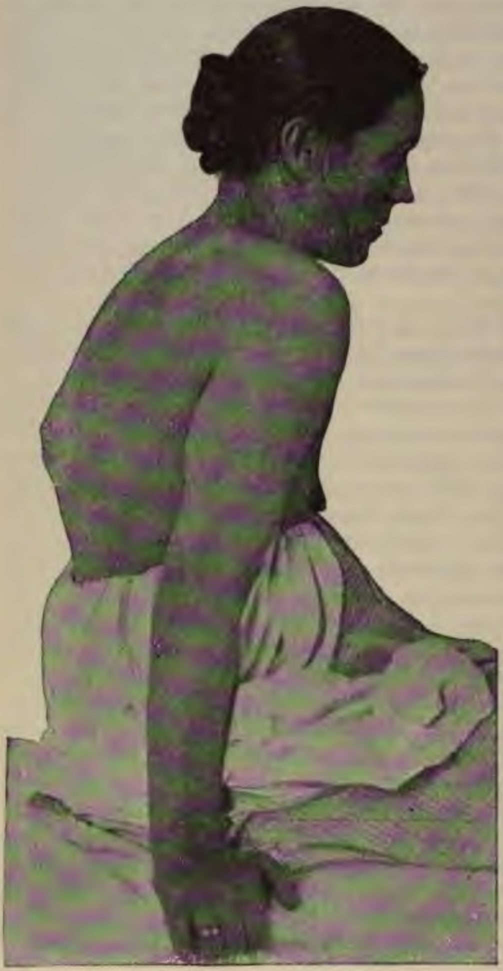

FIG. 127.—Angular kyphosis in vertebral caries. (Personal observation.)

Usually the symptoms of the developing primary disease, the affection of the vertebræ, precede the appearance of the first cord symptoms by some time. The patient feels a dull pain at a definite part of the spine, which is increased by movements of the trunk, by bending or straightening up. Many patients notice the stiffness of the vertebral column of themselves, and sometimes even a beginning deformity. In characteristic cases we notice at once, on examining the vertebral column, an angular kyphosis (gibbus, Fig. 127) of considerable extent or limited to a few vertebræ; but sometimes there is only a slight prominence of a single vertebra, and not infrequently there is externally no change in the formation of the vertebral column. The tenderness on pressure on the spinous processes is also not a constant symptom; it may be very pronounced, but it is not infrequently absent even in marked kyphosis. The first cord symptoms usually consist of painful sensations, which are not confined to the place of the disease, but spread out along the course of the nerve-paths. These pains are due to the compression and the consequent irritation of the nerve-roots, and therefore extend, according to the seat of the affection, into the shoulders and arms, into the lateral portions of the trunk, or into the lower extremities. The initial pains are most frequently felt in the region of the lower ribs or of the epigastrium, corresponding to the most frequent situation of vertebral caries in the lower dorsal vertebræ. They are sometimes very severe, and then they usually have a pronounced neuralgiform character, or they may be more dull, pressing, dragging, etc. Besides the special pains, there are also many forms of paræsthesia, such as formication, cold feelings, and burning feelings.

The disturbances of motility begin to appear at the same time with these symptoms or soon after them. A stiffness and weakness arise which impede the gait more and more, usually not in both legs at once, but first in one and then in the other, although, as a rule, both legs are soon involved. This paresis increases rapidly or slowly, and it may finally go on to a complete motor paralysis. If the seat of the affection be in the dorsal vertebræ, as it usually is, or if it be in the lumbar vertebræ, the paralysis affects the lower extremities only, and the arms, of course, remain intact; but in cervical spondylitis the arms are usually first and chiefly affected. Only on great compression of the cervical cord is the conduction of the fibers passing through it for the lower extremities impaired, and then there are also disturbances of function in these. Ataxia of the legs is not very common, but it certainly does occur in compression of the cord. The ataxia of course is usually concealed by the co-existing paresis of the muscles or the existing spastic conditions. We see ataxia most frequently in the stages of development or recovery of compression paralyses.

Disturbances of sensibility are quite often found, independently of the pains and paræsthesiæ above mentioned, but in many cases of pressure paralysis they are present in only a comparatively slight degree. It seems that the sensory nerves resist pressure more than the motor nerves, just as in the pressure paralyses of peripheral nerves; but possibly their position in the gray matter of the posterior cornua protects them better from the above-mentioned mechanical injuries than is the case, for example, with the motor fibers in the pyramidal tract. The fact is that often, even in complete motor paraplegia, there is little if any diminution of sensibility, and that marked anæsthesia is rare, and is seen only in the last stages of the disease. We find most frequently a slight blunting of sensibility equally to all forms of sensation, especially to the sense of pain, in other cases partial disturbances of the sense of temperature with the sensibility to touch and pain well preserved, sometimes partial anæsthesia of the sense of pressure, etc. The different parts of the skin not infrequently act differently, so that we find portions with quite normal sensibility, as well as very anæsthetic portions. There may also be hyperæsthetic areas of the skin (especially at the upper boundary of the disturbance of sensation), particularly with marked hyperæsthesia to pain (pin-pricks).

The condition of the reflexes is interesting. If the seat of the compression be above the reflex arc, which we must assume to be in the lumbar cord for the reflexes of the lower extremities, we should expect that the reflexes would persist, and in many cases even be increased, corresponding to the decline in the inhibitory influences coming from above. The latter takes place in most cases with the tendon reflexes. The increase of the tendon reflexes, and the coincident increase of muscle tonus, may reach so great a degree in many cases of compression of the dorsal and cervical cord as to show in the lower extremities the pronounced type of spastic rigidity and spastic paralysis. The legs are then found in a rigid tonic extension, with the knees pressed firmly together, or there is spastic contracture of the flexors and adductors. All passive movements of the legs can be performed only with the greatest difficulty, and the attempt often causes a general tremor of the leg. All the tendon reflexes (patellar reflex, adductor reflex, ankle clonus, etc.) are much increased, but we must remember that even in apparently flaccid paraplegia the reflexes are sometimes quite lively. In cervical spondylitis the tendon and periosteal reflexes in the arms are also increased sometimes, but in other cases, if the reflex arc be injured, they are absent. Where the seat of the compression is above the lumbar cord, the cutaneous reflexes sometimes show considerable vigor, but their increase is much less frequently decided than is the increase of the tendon reflexes. In severe pressure paralyses in the dorsal cord the cutaneous reflexes are sometimes diminished. They are rarely entirely absent, but one must understand testing them, and must employ long-continued cutaneous irritation, such as pinching and pricking, in different parts of the skin in order to provoke them (*vide supra*, page 839). [In spastic conditions the Babinski reflex is usually found.—K.] In many cases of total compression of the cervical cord we find a flaccid paraplegia with absence of the tendon reflexes in the legs, just as in traumatic total transverse lesion of the cervical cord. In these cases we shall usually be able to find secondary changes in the lumbar cord on careful examination (*vide* page 931).

Disturbances of the rectum and bladder occur in almost all severe cases of pressure paralysis. The difficulty of micturition is often an early symptom of the disease; later on there is complete retention of urine, and, in the more advanced stages of the disease, there is usually incontinence. With this the danger of the development of cystitis becomes very great. The bowels are usually constipated, but sometimes there is also incontinence of fæces.

Trophic disturbances are often found in the paralyzed parts. If there be severe symptoms of sensory irritation there may sometimes be eruptions of herpes, corresponding to the course of the nerves. More frequently there are chronic disturbances in the nutrition of the skin in severe and long-continued cases. It becomes dry, the epidermis scales off, and the nails become brittle. Bed-sores form very easily in severe cases on the sacrum, on the buttocks, on the inner side of the knees, and on the heels, especially when the patient has insufficient care. The muscles retain their normal volume and their normal electrical excitability in many cases as long as their trophic center remains uninjured; but sometimes, even when the point of compression is above the lumbar cord, there is a great flaccidity (*vide supra*) and atrophy of the muscles of the legs, although the electrical reaction of the nerves is normal, or at most a little reduced in quantity. In cervical spondylitis with lesion of the cervical cord or of the anterior cervical roots there may be atrophic paralysis in the arms. If there be compression of the lumbar cord there is atrophic paralysis of the leg muscles. We have already learned to recognize the peculiar type of nervous symptoms in disease of the lowest portion of the cord or of the cauda equina (page 931). We sometimes see the same type in caries of the upper lumbar vertebræ with compression of the conus, or in caries of the sacrum with compression of the cauda equina. The points to be noticed in distinguishing between affections of the conus and affections of the cauda have already been mentioned on page 931.

Thus we see, under some circumstances, in compression of the cord, the whole group of symptoms arising which are the necessary consequence of the break in conduction in the spinal cord, and which we shall likewise meet again in various other spinal affections, especially in myelitis and in tumors. The intensity and number of the symptoms must, of course, vary very much in the different cases. If the compression be quite slight, there are only mild symptoms of sensory irritation and slight paresis. One of the earliest and most constant signs of a compression of the cord, in the dorsal or cervical region, is a decided increase of the patellar reflex. We sometimes find it at a time when there is scarcely a single other cord symptom present. If the compression increases, the paresis becomes more marked, the disturbance of sensibility is greater, and vesical disturbances arise, until finally the complete type of an entire transverse interruption of conduction in the cord is developed. The precise limitation of the symptoms, especially the distribution of the sensory disturbance, follows the rules already given (page 920). There is only rarely, however, a complete transverse destruction of the spinal cord in vertebral caries. The conduction of sensory impressions, at least, is usually not wholly abolished. The time required for the development of the symptoms of spinal compression differs very much. They sometimes attain a considerable height in a short time, and sometimes they develop only after a course of months. Variations in the intensity of the symptoms are frequent, and they point perhaps to a corresponding variation in the severity of the compression.

In regard to the result of pressure paralyses in vertebral caries, spondylitic processes without doubt can recover, which is by no means in contradiction to their character as a local tubercular process. In this connection the fact is of great practical importance that even the pressure paralyses may be completely restored, as far as the cause of the compression can be removed by the absorption of inflammatory and tubercular new growths, so that a complete and permanent recovery may take place even after the paralysis has lasted for some months, or a year, or even a year and a half. Such recoveries have been seen in a considerable number of cases by others and also by ourselves. They are explained in part by the restoration of function in fibers which were rendered merely incapable of conduction by pressure, but not completely destroyed (page 937), and in part perhaps in old cases by the occurrence of regeneration. Fickler has made the interesting observation in the author's clinique that the ends of the pyramidal fibers in the lateral tract may grow out above an old compression, pass around the scar of the compression through the anterior fissure, and enter the cord below it to their anterior horns.

Although, according to this, the prognosis in a part of the cases of pressure paralysis from spondylitis is comparatively good, still many other cases terminate unfavorably. The cause lies either in the occurrence of dangerous sequelæ of the paralysis, such as bed-sores, cystitis, or pyelo-nephritis, with fever and increasing general weakness; or in the development of other tubercular diseases, especially phthisis, or more rarely miliary tuberculosis or tubercular meningitis, which prove fatal. In some cases there is a stationary paralytic condition, since the vertebral disease recovers and sclerosis ensues in the cord, which is capable of no further change.

2. **The Pressure Paralyses in Cancer of the Vertebræ.**—In pressure paralysis from cancer of the vertebræ, which is not very rare, the spinal affection may seem to be primary, especially when the primary growth (breast, etc.) has not up to that time caused any very striking clinical symptoms. The first symptom is usually pain, both in the region of the new growth and radiating in neuralgic form. The pain becomes distressing if the tumor grows about and compresses the posterior roots ("*paraplegia dolorosa*"). The other spinal symptoms are precisely analogous to those in compression of the cord by vertebral caries, and therefore they need not be described again in detail. Changes in the shape of the vertebral column sometimes occur, but we only rarely find a characteristic angular gibbus, as in vertebral tuberculosis. The general cancerous cachexia is usually soon apparent. The course is in general more rapid than in caries. The disease rarely lasts more than nine to eighteen months before its invariably fatal end.

Diagnosis.—The frequency of the pressure paralyses of the spinal cord admonishes us to examine the vertebral column carefully in every case of spinal disease, especially if the case in question can not be referred to one of the special types of systemic diseases (*vide infra*). We should look especially for the stiffness of special parts of the vertebral column on movements of the head or trunk, and also for the pronounced tenderness of single vertebræ to pressure, and, finally, as a most important and most certain sign, for the deformity of the vertebral column, the marked projection of a single spinous process, or the formation of an evident angular kyphosis. If we find such a Pott's boss the diagnosis is easy, and we can then at any rate refer the existing cord symptoms to a compression of the cord caused by a disease of the vertebræ.

The diagnosis is more difficult if the signs of an affection of the vertebræ be not evident. It must once for all be stated that vertebral caries does not invariably and necessarily result in a manifest knuckle, and that even the tenderness of the vertebræ to pressure is sometimes very slight in spondylitis. In such cases the examination of the vertebral column must be repeated frequently, since even slight anomalies obtain a diagnostic value if constantly present; and the whole course of the disease is also to be considered. The most characteristic features of a compression of the cord are its beginning with symptoms of sensory irritation, the persistent and more or less severe pain in the back, the preponderance of symptoms of motor paralysis with comparatively little disturbance of sensibility, and finally the frequent asymmetry of the symptoms on the two sides, which may even recall the type of the so-called "unilateral lesion" of the spinal cord (*vide infra*). Sometimes the cause of the spinal symptoms is at first obscure, and later on in the disease a marked anomaly of the vertebral column develops.

If the diagnosis of an affection of the vertebræ be certain, the next question is as to the nature of it, especially whether we have to do with a spondylitis, or with a cancer of the vertebræ. Since spondylitis is by far the more frequent disease, we must always think of that first, especially in young people predisposed to tuberculosis, and where we have the formation of a pronounced angular kyphosis. In cancer of the vertebræ the coarser changes in the form of the vertebral column are generally less marked. This develops usually in older people, after the age of forty, and is manifested, as we have said, by the great intensity of the initial symptoms of sensory irritation. The discovery of a primary nodule of cancer (in women the breasts require particular examination) and, as we ourselves have seen, the appearance of a cancerous swelling of the inguinal lymph-glands, may serve to support the diagnosis. Finally, a certain stress is to be laid on the well-known general habit of patients with cancer, on the peculiar cancerous cachexia, and on the whole course of the disease.

In regard to the distinction between caries of the vertebræ and sarcoma of the dura mater, see the chapter on tumors of the spinal cord.

The place of compression is, in the majority of cases, to be recognized by the evident localization of the disease of the vertebræ. In other respects the same rules hold for localization which we have given in detail in the second chapter of this section (page 920 *et seq.*).

Treatment.—In regard to the special treatment of spondylitis, especially the orthopædic treatment, we must refer to the text-books of surgery. The most benefit is to be expected from the various contrivances for extension of the spinal column. It can not be denied, and we have ourselves several times seen, that such extension sometimes produces a decided diminution of the pressure on the cord, which is followed by a remarkably rapid improvement of certain symptoms of paralysis. Such observations are strongly in favor of our theory that the action of vertebral caries is usually purely mechanical. Very often, however, we have had to admit that the extension apparatus was of no benefit or only of temporary benefit, or that it even materially aggravated the patient's distress. In vertebral caries with co-existing spinal symptoms we must therefore use extension of the vertebræ, plaster jackets, etc., with caution. Surgical interference to remove dislocated vertebræ, compressing masses of pus, etc., has been repeatedly tried in vertebral caries, sometimes with benefit, but often without success. In most cases we shall be limited to a general symptomatic and dietetic treatment. Persistent rest in bed, on the back, is of the greatest importance, and it must always be strongly advised. Several cases of complete recovery from severe pressure paraplegia, which we have had under our own observation, were due to this alone. The most careful treatment of the general condition (fresh air, good food, cod-liver oil, arsenic, iron, etc.) of course also demands attention. We have seen very unfavorable action from Koch's tuberculine. Local applications to the vertebral column are much used—dry cups, painting with iodine, and especially the hot iron. The use of the latter in spondylitis has even to-day warm advocates, and, in fact, deserves to be tried, the procedure with Paquelin's thermo-cautery being especially easy; we make some three or four eschars on each side of the diseased vertebræ.

Among other remedies we may mention stabile galvanization at the point of pressure, and the electrical treatment of the paralyzed extremities; also the use of baths, especially salt-baths. In regard to the symptomatic treatment, we will refer to the following chapter.

CHAPTER V

ACUTE AND CHRONIC MYELITIS

(Transverse Myelitis. Diffuse Myelitis)

Definition and Ætiology of Myelitis.—"Myelitis" is the name we give to those inflammatory degenerative diseases of the spinal cord which develop primarily in the substance of the cord itself and lead to a more or less extensive destruction of the nervous elements. We know very little of the causes of these diseases, and of the precise nature and manner in which these causes exert their morbid influences. The name of myelitis has probably been used up to the present time for several quite distinct morbid processes, since it is often by no means easy to recognize the precise origin of the anatomical changes. At any rate, we must recognize this uncertainty in the definition of myelitis, and at least try as much as possible to keep separate the forms which surely do not belong in the same group. Thus we have said expressly in the previous chapter that the purely mechanical traumatic changes in the spinal cord are to be differentiated from true myelitis and that the inflammatory processes in these cases are at most secondary. We must also emphasize the fact that anæmic necrosis of the cord from vascular obstruction (from primary endarteritis, thrombosis, or embolism of the vessels) does not seem to have the significance in the cord that it does in the brain, but that we must always bear in mind the possibility of its occurrence, and hold firmly to the fundamental distinction between such anæmic softening or infarction and primary degenerative inflammation. It is very difficult to determine the position which syphilis holds in relation to the definition of myelitis. The distinction is, of course, easy in the rare cases where there is an actual gumma in the cord, but there are also a good many cases of apparently genuine degenerative myelitis in persons who have previously had syphilis. In these cases the anatomical findings often leave us in the lurch, if the question of the syphilitic origin of the disease is to be answered with certainty. Even in cases where this origin is recognized, the way in which the syphilitic agent acts is still utterly obscure. Most stress is generally laid, of course, upon the "specific syphilitic" changes in the blood-vessels; but these criteria are not wholly unequivocal in their nature, and the question of the mutual relations between the disease of the vessels and the true parenchymatous changes still needs further discussion. It is, however, of great practical importance to know that syphilis apparently plays a considerable part in the ætiology of the spinal disease hitherto classed as myelitis. The myelitis of the upper dorsal cord, which runs its course chiefly under the form of spastic spinal paralysis (*q. v.*), seems frequently to be due to previous syphilis.

We can call only those processes genuine myelitis in which some excitants of inflammation act locally upon the substance of the spinal cord and cause diffuse inflammatory degenerative changes therein. Such genuine myelitides are decidedly rare, and little definite is yet known as to their causes. We sometimes see the disease develop in previously healthy persons, who are ignorant of any special harmful influence to which to attribute their illness. In other cases the blame is laid upon exposure to cold, repeated wettings, physical over-exertion (such as military maneuvers), etc. In women, the first symptoms of the disease sometimes follow childbed. These alleged causes, however, play at most the part of an exciting cause. It is wholly out of the question that sexual excesses, as was formerly believed, can of themselves lead to anatomical disease of the spinal cord; the same is true of severe emotional disturbances.

We may assume that the special cause of true acute myelitis is in all probability some infectious germ. This has been maintained for some time in regard to acute poliomyelitis (*q. v.*); but the evidence is increasing in favor of the infectious nature of acute transverse myelitis also. Thus we have seen a case follow a felon, and staphylococci were found in the cerebro-spinal fluid, which was withdrawn by lumbar puncture. Fürstner found pneumococci in the foci of inflammation in a case of acute disseminated myelitis. In short, it is probable that we shall obtain still clearer ideas of the ætiology of acute myelitis by continued investigations. There is no doubt that infectious causes are to be considered in the rare cases of purulent inflammation of the cord (abscess of the cord); the same is true of the myelitides following acute infectious diseases (typhoid fever, small-pox, erysipelas, influenza, malaria, gonorrhœa, etc.). As to the causes of chronic myelitis—that is, of the "myelitic" transverse diseases of the cord which are chronic from the outset—our knowledge is the more uncertain since it is very hard strictly to define this morbid process. We will return to this point later.

That secondary myelitis may arise by direct extension of the inflammatory process from the vicinity has been definitely determined only in acute purulent meningitis (especially the epidemic cerebro-spinal form) and tubercular meningitis. In the other cases formerly classed under this heading, we have to do, as has repeatedly been said, mainly with purely mechanical consequences. The hypothesis that neuritic processes may extend to the cord (ascending neuritis) we have already (page 886) characterized as doubtful and practically unimportant.

Pathological Anatomy.—Macroscopic examination of the cord in its fresh condition shows no marked pathological changes except in a small number of cases. At the first glance, the spinal cord often seems almost completely normal, even if there have been severe spinal symptoms during life, and sometimes the opacities and adhesions of the pia, which often strike us at first, have no practical importance. If we test the consistency of the cord carefully by touching it, of course a change in it often strikes the practiced examiner, since the cord over a definite extent is either softer and more flexible, or, on the other hand, harder and firmer, than normal. If we now make a number of cross-sections through the cord, we notice that the substance of the cord rises up more on section, that the outline of the gray matter is less distinct, and especially that the white matter is of a reddish-gray color, and that sometimes there is also a reddish, hyperæmic coloring of the gray matter. In some cases we can recognize small capillary hæmorrhages with the naked eye. The more acute the disease the softer, as a rule, is the cord in the part diseased ("acute inflammatory softening"). If the disease is chronic or if some time has elapsed since the expiration of the acute initial stage, the cord in the diseased spot is denser, firmer, and sclerosed, and grayer in color on section, but it is not possible to determine precisely in the fresh cord the extent and degree of the disease.

The changes are much more plainly visible if we harden the cord in chromic acid, or Müller's fluid,* for at least eight or ten weeks. All the normal parts of the white matter of the cord assume a dark-green color, which is really due to the staining of the medullary sheaths by the chromic acid. The diseased portions, in which the medullary sheaths are mainly if not entirely absent, are thus often very sharply distinguished from the healthy, dark-green portions. Since similar, although less sharply defined, differences in color between healthy and diseased tissue are also noticed in the gray matter, the cross-section of the cord, well hardened in chromic acid, usually gives quite a correct idea of the extent of the disease.

We obtain more precise disclosures, however, as to the form of the anatomical changes by microscopical examination. When made on the fresh, unhardened cord, it affords little information. The presence of numerous granular cells (*vide infra*) in fresh teased-out preparations is the only thing that is important, since they show with certainty the existence of a pathological change. If, however, we make thin cross-sections of the hardened cord, and stain them with carmine or some similar staining fluid, even the naked eye notices at once a marked difference between the diseased and the healthy tissue, since the former, which is almost always richer in connective tissue, has a much darker staining, and thus is distinguished from the brighter normal tissue. The microscopic examination now shows that in the diseased parts the normal nerve-tissue has been almost wholly or at least partly destroyed. Only occasionally do we see nerve-fibers of normal appearance remaining here and there. In other places the fibers that are still visible are smaller and atrophied, and the axis-cylinders have in part lost their medullary sheaths, or are swollen. The changes in the ganglion-cells are harder to follow, but in more advanced cases they also show marked signs of destruction; they are contracted, rounder, and have lost their processes. The increase of the neuroglia tissue corresponds to the destruction of the nerve-substance. The meshes of the neuroglia extend and swell, so that the space formed by the destruction of the nerve-tissue is in great measure taken up by connective tissue. The older the process, the firmer and more fibrous is the connective tissue. The nuclei of the neuroglia increase in number, and we often find a very great increase in those peculiar, flat cells with many processes, first described by Deiters, and named after him—the so-called "Deiters' spider-cells." The fatty granular cells are also easily recognized in hardened preparations, provided they are not treated with alcohol. They lie in the interstices between the meshes of the neuroglia, and are especially numerous about the vessels. They are to be regarded either as white blood-corpuscles, or perhaps as endothelial cells from the sheaths of the vessels, which have taken up the fat from the disintegrated nerve-substance. If, therefore, the process be still fresh, or if it be still advancing, the fatty granular globules are to be met with in great numbers, while in old, sclerosed nodules, only a few of them, or scarcely any, are to be found. The changes in the vessels are usually very striking. They are often dilated and congested. Here and there there may be hæmorrhages. The vascular walls are thickened, especially in old cases, and sometimes have become peculiarly homogeneous—"hyaline degeneration"; and a large accumulation of nuclei, lines of fatty granular cells or peculiar epithelioid cells, may be found about the vessels. The so-called corpora amylacea are sometimes present in chronic cases in great numbers, and sometimes they are only scanty. Their significance and their genesis are still unknown.

The extent of the whole process varies very much in different cases. We usually find one main focus of myelitis, which extends in a diffuse manner over the greatest part of the transverse section of the spinal cord, and which may reach up-

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[* The formula for Müller's fluid is as follows: Two and a half parts of potassic bichromate, one part of sodic sulphate, and one hundred parts of water.—K.]

ward and downward for a space of five to ten centimetres or more. The dorsal portion of the cord is most frequently affected (dorsal myelitis), the upper half being usually most involved, but in some cases the lower half is chiefly affected. Nearly the whole of the dorsal cord is often the seat of a diffuse inflammatory affection, which, of course, differs in extent at different levels. In other much rarer cases the chief focus of disease is in the cervical cord (cervical myelitis), somewhat more frequently in the lumbar cord (lumbar myelitis). We often find small, distinct foci in the vicinity of the main focus. In all severe and long-continued cases there develops later a systemic ascending and descending secondary degeneration (*vide infra*).

Acute disseminated myelitis has a special form of anatomical distribution It occurs in many small foci which may extend almost throughout the whole length of the cord. Individual columns of the cord, especially the posterior columns, are often chiefly affected. In rare cases, as has already been said, true purulent inflammation—that is, circumscribed abscess with purulent breaking down of tissue—has also been observed.

The Individual Symptoms of Myelitis.—The course of transverse myelitis differs so much in different cases that it is impossible to give a picture of the disease which will be generally applicable. According as one or another part of the cord is involved, the clinical symptoms will relate chiefly to the sensibility or the motility, the trophic functions or the reflexes, and will be present in the upper or the lower extremities, or in both at once. The following description will, therefore, be devoted first to the single symptoms, and will give the inferences which, according to the present state of our knowledge, may be drawn from their presence as to the seat and the extent of the anatomical process; not until then will we endeavor to distinguish the different clinical forms of myelitis.

1. Symptoms of Motor Paralysis are not only the chief symptoms, as a rule, in well-developed myelitis, but are often the first sign of the beginning of the disease. The patient feels at first only a slight weakness in one or both legs; he gets tired more easily in walking, and begins to "drag" his legs after him. Sooner or later the motor weakness gradually becomes greater and increases to complete paralysis. The patient is then bed-ridden, and, finally, can not make the least active movement with his legs. The symptoms of paralysis in the arms are analogous.

Since the chief paths for the conduction of voluntary motion are situated, as we have seen, in the lateral columns of the spinal cord, and especially in the lateral pyramidal tract, we conclude, in every spinal disease where symptoms of paralysis are present, that there is an interruption of this tract—that is, an implication of the posterior portions of the lateral columns. Since in transverse myelitis the whole cross-section of the cord is more or less involved, the paralysis also extends to the two halves of the body: motor paraplegia is the characteristic form of paralysis for transverse myelitis. Paraplegia of the lower extremities may of course arise wherever the myelitis is situated, whether in the lumbar, dorsal, or cervical region; but the upper extremities necessarily remain entirely free in every dorsal or lumbar myelitis. The occurrence of paretic symptoms here, and the final development of a brachial paraplegia, point with certainty to an implication of the cervical region, to a cervical myelitis. If the symptoms of paralysis are not alike in the two corresponding extremities, but are more marked on one side than on the other, the anatomical affection must also be more intense on that side of the cord than on the opposite side.

2. Symptoms of Motor Irritation of various sorts are often seen, both at the beginning and during the whole course of myelitis. Single twitchings come on spontaneously in the limbs, which are at the same time paralyzed, or at least paretic, and these twitchings are short and rapid or slow and persistent. The

thighs are drawn up on the abdomen, or there are severe spasms of the extensors. The interpretation of these symptoms is not always easy. It is often particularly hard to decide whether they are the result of a direct irritation of motor fibers in the cord, or whether they represent reflexes (*vide infra*). The value of the symptoms of motor irritation for the localization of the disease is accordingly slight, but, of course, in these cases we must consider chiefly the motor tracts in the lateral columns.

3. ATAXIA.—There can be no doubt that actual ataxia also occurs in transverse myelitis, but it is rarely pronounced because the co-existing spastic or paralytic symptoms, which are almost always present, of course conceal the ataxia. We therefore see evident ataxic disturbances rather in slow cases which do not speedily become paraplegic, or sometimes not until the disease improves. In regard to the localization of the disease, we may usually conclude, when there is ataxia, that the posterior columns are involved. Very marked ataxia of the arms and legs occurs in the acute disseminated myelitis of the posterior columns.

4. DISTURBANCES OF SENSIBILITY.—The disturbances of sensibility usually appear to a marked degree only in the later stages of the disease. At the outset we generally notice merely mild symptoms of sensory irritation in the legs, such as formication, prickling, numbness, a woolly feeling, etc., while severe pain is hardly ever present in transverse myelitis, and hence it always points rather to some affection of the vertebræ or the meninges. In very acute myelitis there may be at first a severe local pain in the back. Slight diminution of sensibility is often to be made out early on careful examination, but, in many cases, the sensibility remains for a long time wholly, or almost wholly, intact, either because the localization of the disease spares the sensory portions of the cord, or because the sensory paths of conduction are more resistant, or they can act vicariously for one another to a higher degree. The more carefully we examine, the earlier shall we be able as a rule to find at least partial disturbances of sensibility. The examination of the sensibility to temperature, especially, should never be neglected. Partial anæsthesia of the pressure sense, analgesia, etc., also occur. We must also pay attention to the not uncommon delayed sensibility to pain (page 793). In the further course of the disease, however, there are almost always more marked disturbances of sensibility, and finally frequently a complete anæsthesia. On the other hand, we see in many cases a striking hyperæsthesia to painful stimuli, such as a pin-prick.

From the presence of marked disturbances of sensibility we can conclude with certainty that there is an affection of the posterior columns, and especially of the posterior cornua of the gray matter. With marked analgesia and thermo-anæsthesia the latter are always involved. Disease of the lateral columns also may probably cause disturbances of sensibility (page 799).

The disturbance of sensibility gives important service in estimating the height at which the affection in the cord is situated. If we search on the trunk for the line where the cutaneous sensibility becomes normal, we may from this determine the upper boundary of the myelitis in accordance with the data furnished on page 924; but since each cutaneous region, as has been said, is supplied with sensory fibers from at least two or three spinal segments, we must look for the upper boundary of the disease not simply in the segment corresponding to the upper boundary of the anæsthetic area, but one or two segments higher. In myelitis in the lumbar region the disturbance of sensibility reaches to the umbilicus, or even a little higher; in myelitis in the lower dorsal region it reaches about to the lower end of the sternum; in myelitis in the upper dorsal region to the level of the axillæ; and in cervical myelitis the sensibility of the upper extremities is also impaired, but complete anæsthesia is very rare.

5. Cutaneous Reflexes.—As is generally assumed, the reflex arcs in the cord are found at about the same level as the centripetal sensory and the centrifugal motor fibers. They are also connected with fibers which come from above and have properties of reflex inhibition. If these fibers above the reflex arc are put into a state of irritation, the reflex is thereby impaired; but if the conduction be broken in these fibers, the reflex activity appears increased, the reflex comes on with a weaker irritation, and the contraction is more vigorous. If the reflex arc itself be broken at any point, the reflex must disappear.

The data from the examination of the patient may generally be harmonized with this scheme, although, of course, the reality probably shows more complicated conditions. In extensive lumbar myelitis, by which the reflex path in the lumbar cord is broken, the cutaneous reflexes in the lower extremities must be diminished or absent. In these cases the loss of sensibility runs about parallel to the diminution of the reflexes. In dorsal and cervical myelitis, however, the reflex arc in the lumbar cord remains unimpaired, but the conduction of sensory impressions to the brain may very well be interrupted. In these cases the cutaneous reflexes are retained, even when there is anæsthesia; or, if the reflex inhibitory influences be removed, they are decidedly increased. [There is often dorsal extension of the great toe on irritating the sole of the foot—the Babinski reflex. —K.] The cutaneous reflexes in the legs, however, may also be diminished or delayed in myelitis of the lower dorsal cord, in which case we must imagine as a rule a secondary loss of excitability of the fibers and cells presiding over the reflex, or perhaps an irritation of inhibitory fibers. It is often important in determining the seat of myelitis to examine the cremaster reflex, the abdominal reflex, etc. If the reflex paths described above (page 927) be interrupted, the reflexes of course must be absent.

6. Tendon Reflexes.—The same rules generally hold in judging of the tendon reflexes as are to be considered in judging of the condition of the cutaneous reflexes. We know that the reflex arc of the patellar reflex is at the level of the second to the fourth lumbar segments (page 927). If the entrance zones of the posterior sensory roots or the ganglion-cells in the anterior horns be diseased at this level, the patellar reflex must disappear. The Achilles tendon reflex and the ankle clonus have their reflex arcs in the sacral segments of the cord. The Achilles tendon reflex is always absent in extensive disease of the posterior columns and of the gray matter in the corresponding portion of the lumbar cord. The absence of the tendon reflexes in the lower extremities is therefore one of the most important points besides the other symptoms for the diagnosis of a myelitis of the lumbar cord. In almost all inflammations above the lumbar cord—that is, in dorsal and cervical myelitis—there is, however, a very decided increase of the tendon reflexes, the result, as we must suppose, of the loss of the reflex inhibitory influences. We have much reason to assume that the fibers which influence the condition of the tendon reflexes run chiefly in the lateral columns (pyramidal tract) of the spinal cord, but that they are not identical with the fibers of the lateral pyramidal tracts which serve for voluntary motion (see the chapter on spastic spinal paralysis). We may therefore assert that, with a considerable increase of the tendon reflexes in the lower extremities, the seat of the myelitis must be above the lumbar cord—that is, in the cervical or dorsal cord—and that in these cases we have to suppose that the lateral columns are chiefly implicated. In cervical myelitis the tendon reflexes in the upper extremities are often considerably increased.

We have already said, on page 840 *et seq.*, what is necessary in regard to the different signs of the increased tendon reflexes, the exaggerated patellar reflex, ankle clonus, the periosteal reflexes, etc. The peculiar character which the paralysis of the legs assumes from a considerable simultaneous increase of the tendon reflexes (spastic paraplegia) will be described more fully in the chapter on "spastic spinal paralysis" (*vide infra*).

7. Disturbances in the Bladder and Rectum.—Disturbances in micturition are one of the commonest symptoms of myelitis. The first manifestation is usually a difficulty in micturition; the patient has to strain and to wait longer before urinating. There may finally be a complete retention of urine from paralysis of the detrusor urinæ. In the later stages of the disease, however, there is usually a paralysis of the sphincter vesicæ, and consequently more frequent desire to micturate, and finally incontinence of urine. The disturbances of the bladder give no points for the localization of myelitis, since they may occur with disease at any level of the spinal cord; but we believe we are right in assuming that they always permit us to decide that the posterior columns of the cord are involved.

The clinical significance of disturbances of the bladder in myelitis, and in many other diseases of the cord, apart from the great distress and discomfort for the patient, lies in the fact that they very often—almost always in severe cases—give rise to the development of cystitis. In retention of urine the use of the catheter, by which inflammatory irritants are often brought into the bladder, in spite of all attempts at disinfection, leads to decomposition of the urine and to cystitis; but where there is also incontinence, the imperfect closure of the sphincter and the constant presence of stagnating and decomposing urine in the urethra are the causes of the entrance of these irritants into the bladder. If cystitis have developed, it may be followed under some circumstances by pyelitis and purulent pyelo-nephritis (*q. v.*), which conditions are often the immediate cause of death from the sequelæ connected with them, such as fever—sometimes associated with chills—general weakness, and emaciation.

Defecation is also disturbed in many cases of myelitis. There is usually constipation at first, which may depend either upon weakness of the intestinal peristalsis, or upon paresis of the abdominal muscles. Sometimes the constipation reaches such a degree that the bowels move only at intervals of one or two weeks. In many severe cases there is finally incontinence of fæces, as a result of paralysis of the sphincter ani. We can give no details as to the localization of the nerve-tracts in the cord which take part in defecation.

We have yet to note that micturition and defecation are often aroused reflexly in an abnormal fashion where there is increased reflex irritability. On irritation of the skin over the thighs, the perineum, the gluteal region, etc., there is often an involuntary contraction of the bladder, associated with loss of urine.

In conclusion, we may mention, as an addendum, that the sexual functions are often considerably disturbed in many cases of myelitis, and finally they may be wholly lost.

8. Trophic Disturbances.—The trophic condition of the paralyzed muscles affords extremely important points for diagnosis. In cervical and dorsal myelitis the trophic centers in the lumbar cord for the muscles of the legs remain intact; the paralyzed muscles, therefore, retain essentially their normal volume, and especially their normal electrical excitability. Even in such cases the muscles are sometimes flabbier and of less girth than under normal conditions, but this depends partly on the decline in the general nutrition, and partly perhaps on the lack of movement, the "atrophy of inactivity." Only occasionally do we find more marked muscular atrophy, but without reaction of degeneration; but if we find in myelitis a marked atrophy, with reaction of degeneration in the muscles of the lower extremities, we can from this draw a definite conclusion that the anterior gray cornua or the fibers of the anterior roots in the lumbar cord are affected (see page 851). In an analogous fashion degenerative atrophy with reaction of degeneration in the muscles of the upper extremities points to an affection of the anterior gray matter in the cervical cord.

Trophic disturbances in the skin are also frequent. We often find the skin dry, hard, with a scaly epidermis, and the nails thickened and brittle. Exceptionally there are eruptions of herpes, urticaria, etc. Vaso-motor disturbances also occur. Sometimes the paralyzed extremities show a mottled, cyanotic reddening, and feel cold. Slight œdema is quite frequently present in the paralyzed parts. Disturbances of the sweat secretion are not infrequent. We find either that it is absent or that there is a great increase in it, so that the paralyzed parts are constantly moist. All these symptoms have no value at present for the special topical diagnosis.

The frequent occurrence of bed-sores in the sacral region, over the glutæi, or more rarely over the trochanters on the feet or the inner side of the knees, is of great practical importance. Although trophic and vaso-motor influences may play a part in their origin, still their ultimate cause is always to be found in external conditions, pressure, uncleanliness, etc. The more faulty the care of the patient is, the easier bed-sores arise. With completely paralyzed and anæsthetic patients, with incontinence of urine and fæces, of course they sometimes can not be wholly and permanently avoided, even with the most careful management. The extent which a bed-sore may reach is sometimes absolutely frightful. A large part of the sacrum may be laid bare, after the overlying soft parts and the periosteum have become gangrenous and been thrown off.

9. Disturbances in the Region of the Cerebral Nerves are entirely absent in most cases of transverse myelitis. In rare cases of cervical myelitis the process may gradually extend upward and give rise to bulbar symptoms. We sometimes see changes in the pupils in cervical myelitis, such as inequality and spinal myosis; and finally myelitis has been repeatedly combined with an optic neuritis. In general, however, any co-existing cerebral symptoms always require a special explanation. They are either co-existent from the same cause (for example, syphilis), or, instead of an ordinary transverse myelitis, there is multiple sclerosis, or some other disease.

Different Forms of Myelitis. Acute and Chronic Myelitis.—The whole picture of transverse myelitis in its different forms may be constructed from the symptoms described in detail in the preceding paragraphs. We can usually determine without difficulty, at least approximately, the seat and extent of the disease. If we group the chief symptoms of the different forms of myelitis together, according to the location of the disease in the cord, they are as follows:

Cervical Myelitis.—Paraplegia of the legs, combined with more or less extensive disturbances in the upper extremities, and eventually disturbances of sensibility over a like extent. At times atrophy of single muscular regions in the arms. No marked atrophy in the legs. Increased tendon reflexes and spastic symptoms in the legs and often in the arms. Cutaneous reflexes in the legs retained, and sometimes even increased. Disturbances of the bladder and rectum. Sometimes changes in the pupils.

Dorsal Myelitis.—Upper extremities free. Motor, and eventually sensory, paraplegia of the legs, without degenerative atrophy. Increased tendon reflexes and pronounced spastic symptoms in the legs (especially in myelitis in the upper dorsal cord); cutaneous reflexes retained, sometimes increased. Disturbances of the bladder and rectum.

Lumbar Myelitis.—Upper extremities free. Motor, and eventually sensory, paraplegia of the legs. Cutaneous and tendon reflexes in the legs diminished or absent. Under some circumstances degenerative muscular atrophy in the legs with reaction of degeneration. Disturbances of the bladder and rectum. In myelitis of the *lower sacral cord* the paralysis is confined chiefly to the distribution of the sciatics and the bladder and rectum. The disturbance of sensation is found on the posterior aspect of the thighs, the buttocks, and the genitals (page 925).

The above division of the different forms of myelitis refers only to the different locations of the disease and the difference in symptoms thereby produced, so that we must further discuss the division of myelitis into an *acute* and a *chronic* form. This division is made solely by observing the course of the disease, or from the statements in the history of the case as to duration; but we must bear in mind that this refers only to the first stage of the disease, to the rapidity with which the myelitic symptoms develop at the start. In most cases of genuine primary myelitis the myelitic symptoms come on very suddenly, lead in a very short time (sometimes in a few days or a few weeks) to the most complete paralysis (paraplegia, vesical paralysis, anæsthesia, bed-sores, etc.), and advance irresistibly, causing death in a short time (from four to twelve weeks). Such cases are, of course, called acute transverse myelitis. We sometimes see, however, the disease beginning acutely, and rapidly developing a more or less complete paraplegia, which after some time remains entirely stationary, or at least shows only extremely slight further changes. Such cases must also be classed definitely as acute myelitis, although they present later a chronic type of spinal disease, which sometimes lasts for many years; but they are also undoubtedly cases of acute myelitis, ending in relative recovery—that is, in recovery with a scar or defect in the cord. We can call only those cases actual chronic myelitis which are chronic from the first—that is, which begin slowly and progress gradually—but such forms of actual chronic myelitis are very rare, for the not very rare cases which seem to belong under this heading during life usually prove at the autopsy, at least as far as our experience goes, to be something different—multiple sclerosis, tumor, combined columnar or systemic disease, etc. We might rather speak of chronic myelitis in those cases which have an acute initial stage, and then take later a chronic progressive course. At any rate, in our definition of chronic myelitis we must pay especial attention to the clinical course and an accurate history, for the pathological lesions alone can not be conclusive. The pathologist can not determine whether a given transverse sclerosis of the cord is the result of a process chronic from the first or of a long-past acute process.

We can not give at present a definite picture of acute disseminated myelitis. We would be most ready to suspect this form of myelitis if an acute type of spinal disease developed apparently primarily or especially as a sequel of some previous acute infectious disease, and if it indicated an isolated or combined columnar affection rather than a transverse affection. Acute ataxia with loss of tendon reflexes and evident vesical disturbances, or a combination of ataxia with paresis and spastic symptoms, have been most frequently observed.

Recovery from acute myelitis is perhaps not impossible, but it is certainly very rare. We know of no case that recovered completely where the diagnosis could be made with certainty. The cases reported where a recovery is claimed are usually cases of pressure paralysis, multiple neuritis, poliomyelitis, etc. We may speak somewhat more frequently of relative recovery if the severe initial symptoms subside in part and change to a mild, stationary, morbid state. The fatal termination of acute or chronic myelitis is the result of the general weakness which finally sets in; or it comes from cystitis or pyelo-nephritis, both of which are often combined with pyæmic conditions; or from extensive bed-sores; or finally from some complication, such as tuberculosis or acute diseases.

Diagnosis.—The diagnosis of diffuse transverse myelitis is always made by considering the whole group of symptoms prominent in the individual case. The possibility of a compression of the cord must be excluded by a careful examination of the vertebral column, and by consideration of the course of the disease. We must also be sure that the existing symptoms do not correspond to a definite typical disease, or a systemic disease, but that they can agree only with the assumption of a diffuse transverse disease at a certain point in the cord, to be made out accurately according to the symptoms. The further decision, as to whether this diffuse transverse disease is a myelitis, may be made quite positively in acute cases or cases beginning acutely, but in cases chronic from the outset it can hardly ever be made definitely, for precisely the same type of disease may sometimes be produced by multiple sclerosis (*q. v.*), by new growths in the cord (glioma), and finally by combined systemic disease. In the description of all these diseases we will once more return to the likeness sometimes observed between them and transverse chronic myelitis.

Treatment.—Although our therapeutic endeavors are rarely crowned with permanent and complete success, still in many cases we can relieve the suffering and delay the end.

We can try to meet the causal indication in cases where the history or the examination shows evidence of the existence of previous syphilis (page 943). Even if the connection between previous syphilis and myelitis can not be assumed with absolute certainty—and usually it can not—still we must always try inunction thoroughly, using half a drachm to a drachm (grammes 3–5) of mercurial ointment a day. We give twenty to thirty grains (grammes 1.5–2.0) of iodide of potassium daily at the same time. We sometimes see decided improvement from this; but in some cases, of course, the result is uncertain, or the treatment seems to exert even an unfavorable influence upon the disease. In the latter case we must stop it at once.

Of the other methods of treatment the chief are electricity, baths, and cold-water cures. We alternate these. New attempts at cure raise the patient's courage and hope afresh.

Electricity may give symptomatic improvement in many cases, but of course it causes recovery only exceptionally, at most. In severe and hopeless cases, however, it is at least the best means of consoling the patient. The constant current has the greatest therapeutic value. We use large electrodes placed on the vertebral column, and pass not too strong a stabile or slowly labile current through the cord for about four or five minutes, chiefly through the region where we suppose the seat of the disease to be. We usually take the ascending current, and alternate with the two poles on the diseased part. We should avoid reversals and great variations in the strength of the current. We associate with this peripheral galvanization, and often faradization of the muscles and nerves of the paralyzed extremities. Single symptoms sometimes deserve special attention—faradization of the skin in anæsthesia, galvanization of the bladder in vesical weakness, etc. The sittings should take place daily or every other day. If we would be successful, the treatment must be kept up persistently for months.

The treatment of myelitis by baths, if prudently used, may also be of evident service. Even simple tub-baths, such as can be had in almost every household do good service under some circumstances. The chief rules are never to make the baths too warm—about 88° or 95° at most (25°–28° R.)—to limit them at first to ten or fifteen minutes, and to give them at first not oftener than three or four times a week. Sometimes hot baths (95°–100° F. [28°–30° R.] or more) seem to act well, but in other cases they certainly are injurious. If the baths are well borne, they may be employed daily. We should be most cautious in incipient and still advancing cases. The best action of the simple warm bath is seen in chronic myelitis with predominant spastic symptoms. In these cases the duration of the baths may be increased to an hour or more. Sometimes the baths work still better when certain substances are added, especially if we use salt-baths, which are made by the addition of five or ten pounds of common salt (Stassfurt salt), or four to six pounds of brine salt, or one to three quarts of brine to the water.

By introducing carbonic acid into the water through a perforated tube in the floor of the tub we can easily make artificial carbonic-acid salt baths.

If we can send patients in easy circumstances to a health-resort, the carbonic-acid thermal salt springs at Oeynhausen and Nauheim are most suitable for this purpose, also the baths at Kissingen, Wiesbaden, and Baden-Baden, the thermal baths of Ragatz, Teplitz, Wildbad, and Gastein, the mud-baths in Elster, Marienbad, etc.

A methodical cold-water treatment sometimes gives quite good results; but in these cases we should wholly avoid all the more heroic treatments, such as douches, violent rubbing, and very cold baths, and employ only short, cool, full or half baths or packs or mild cold sponging. Hydrotherapeutics are usually combined with electricity.

We can expect but little success from internal treatment, but it can not be dispensed with in practice. A trial of strychnine (subcutaneously or by the mouth) is most to be recommended, and also ergotine, nitrate of silver, and especially iodide of potassium. Salicylate of sodium may be tried in fresh acute cases.

The general hygienic and symptomatic treatment is very important. If the first symptoms of a beginning spinal disease show themselves, we should urgently advise the patient to take the best possible care of himself (avoiding all physical over-exertion), and to enjoy mental rest. The diet should be strengthening but easily digestible. Large amounts of spirits, much smoking, much tea and coffee, etc., are to be avoided. If the patient becomes bedridden, we must employ the utmost care to get a good bed in order to guard against bed-sores. In severe cases, especially where there are disturbances of sensibility, a water-cushion is most desirable. The patient's position must also be frequently changed, and the sacral region must often be washed and rubbed. Every incipient bed-sore must be very carefully treated by Peruvian balsam ointment (1 to 30), iodoform, salicylate plaster, or mercurial plaster, which acts very well, in order to prevent its spreading. When the bed-sore is very extensive the continuous bath is the best remedy.

If there is retention of urine and the patient has to be catheterized, the most extreme care must be employed in cleaning and disinfecting the catheter, or else cystitis will develop in a few days. If it does, it is best in severe cases to wash out the bladder regularly with boracic-acid solution, permanganate of potassium, and like remedies. In milder cases we may try chlorate of potassium internally, fifty to seventy-five grains a day (grammes 3–5), salol, urotropine, or balsams. If there is complete incontinence it is advisable to introduce a permanent catheter (*sonde à demeure*) into the bladder—that is, a Nélaton's catheter, which lies in the bladder and is fastened to the thighs by strips of plaster. The urine runs away through a rubber tube, and we avoid the constant wetting of the skin and linen. In men, good urinals are often perfectly satisfactory, but in women, their application usually causes great difficulties.

Constipation must be met according to the general rules. We should be as sparing as possible with cathartics at first, and try to make an appropriate diet and enemata suffice. If there is severe pain, subcutaneous injections of morphine are unavoidable, but we always delay this as long as possible, although finally we let the dose of morphine be unlimited in hopeless cases.

CHAPTER VI

MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD

(Disseminated Nodular Sclerosis. Sclérose en plaques)

Ætiology and Pathology.—Multiple sclerosis of the central nervous system is a special chronic form of disease, whose anatomical basis consists in the development of numerous disseminated "sclerotic nodules" (*vide infra*) in the brain and cord. We know practically nothing as to its ætiology, for the significance of exposure to cold, over-exertion, and mental emotions, sometimes given as causes of the disease, is wholly doubtful. It is also very improbable that syphilis plays any part in the ætiology of multiple sclerosis. P. Marie claims to have seen the disease come on very frequently as a consequence of some acute infectious disease (typhoid fever, scarlet fever, etc.), and therefore he suspects that the different foci of disease have an infectious inflammatory origin; but our own experience does not permit us to confirm this, nor does our experience pronounce in favor of a relation between multiple sclerosis and chronic intoxication, as Oppenheim supposes; on the contrary, in the majority of our own observations we could find absolutely no material external cause, in spite of careful inquiry. We are therefore disposed to regard the disease as of endogenous nature, as a multiple gliosis due to congenital abnormal predisposition. The anatomical data also harmonize very well with this view. Any injury preceding the development of the disease, acute diseases, intoxications, emotional disturbances, etc., would therefore act at most as exciting causes. If, however, a "disseminated myelitis" should actually at any time follow an acute disease and lead to the formation of multiple, sclerotic nodules in the nervous system with corresponding symptoms, this would be an affection similar to multiple sclerosis, but yet essentially different.

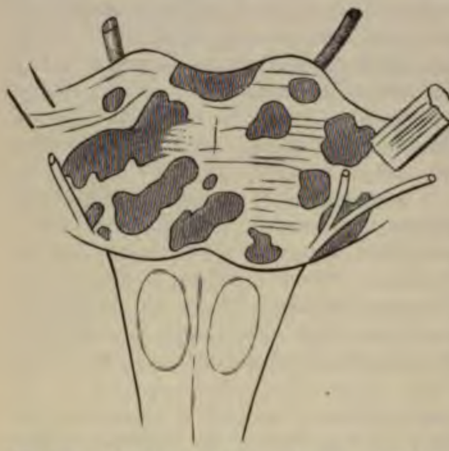

FIG. 128.—Distribution of the sclerosed nodules on the surface of the pons. (From LEUBE.)

A hereditary predisposition seems to be present in only a few cases. The affection occurs chiefly in youth, somewhere between the ages of eighteen and thirty-five, but we have ourselves seen one case, which came to an autopsy, in a man of sixty. The disease also occurs in children. No material distinction has been made out as to sex.

In regard to the development of the different sclerotic nodules, nothing definite has been established at present as to their genesis. The suspicion has repeatedly been uttered that the disease is due to primary changes in the vessels, but no proof of this can be given. The nodules are in part easy to recognize with the naked eye, from their gray color, and we can also feel an increased resistance. They are scattered over the whole central nervous system. Their favorite seats in the brain are the centrum ovale, the walls of the lateral ventricles, and the corpus callosum; but on careful examination we usually find many nodules in the gray matter of the central ganglia and the cortex; the nodules are also quite abundant, as a rule, in the pons, less frequent in the medulla, and very abundant and variously distributed in the cord (see Figs. 128 and 129), chiefly in its white substance. Examined microscopically, the nodules consist of an abundant, reticulated, fibrillary neuroglia tissue, in which the nerve-fibers are lessened in number, but, as a rule, not wholly destroyed; on the contrary, we usually find many axis-cylinders preserved in the nodules, as Charcot first found and as has often been confirmed since by other observers. This fact is important clinically, because it explains why multiple sclerosis only rarely or only in its advanced stages leads to complete paralysis. It also indicates, as it seems to us, that the process does not start from the nervous tissue, but from the interstitial neuroglia tissue. It also explains the remarkable fact that the ordinary secondary degenerations are almost always absent, in spite of the presence of many nodules in the spinal cord. In the vessels we usually notice thickening of the walls, which may be surrounded by neuroglia cells. The author regards these changes as of secondary nature. Fatty granular cells are often to be found in fresh cases.

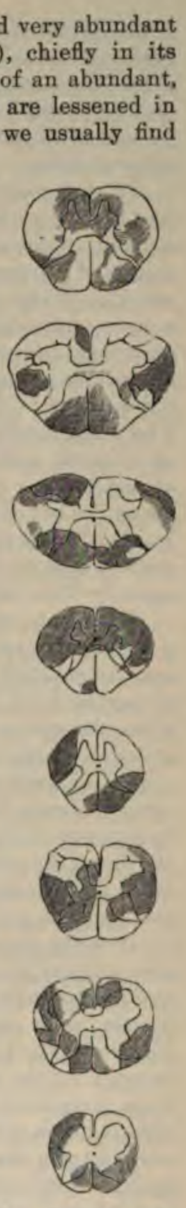

Fig. 129.—Example of disease of the cord in multiple sclerosis. The dark portions are the parts diseased.

Clinical History.—From the variations which the number and the localization of the nodules show, it may be understood from the outset that a picture of the disease which represents all cases can not be drawn; but many cases show so characteristic a group of symptoms that the diagnosis can often be made with considerable certainty. We will first describe this typical form of disease, for the knowledge of which we must thank Charcot chiefly, and then we will add some remarks upon the cases which differ from this type ("*formes frustes*"), which are by no means very rare.

The symptom of the typical cases of multiple sclerosis which we must first mention is an extremely striking disturbance of motion which manifests itself either as actual ataxia or as the so-called "intention tremor." This "tremor" was apparently the reason why multiple sclerosis was repeatedly confounded in former years with paralysis agitans, although the two diseases have not the slightest resemblance one to the other. The one fact that the motor disturbance of multiple sclerosis comes on only exceptionally when at rest, but almost always on voluntary movement, affords an effectual distinction. Moreover, the motor disturbance of multiple sclerosis can be called an actual tremor only in part of the cases. Such a true oscillatory tremor is often seen, for example, in the arms when the patient tries to hold them out unsupported, but the tremor, in distinction from that of paralysis agitans, seems to occur less in the fingers than in the larger joints. We have scarcely ever seen oscillatory tremor in the legs in multiple sclerosis. In purposive movements there is rarely a true oscillatory tremor in the arms, but rather an ataxic motor disturbance—that is, an uncertainty and an excursive movement, which is especially plain when the patient tries to touch a given point with his forefinger, to put the tips of the forefingers together, to bring a glass of water to the mouth, to pour water from one test-tube into another, etc. In the legs the ataxia usually occurs precisely as in tabes dorsalis (as in putting the heel on the other knee). We sometimes see considerable wavering and shaking of the head when unsupported. The ataxia of the trunk is usually very evident. If we make the patient stand with the feet together, it usually causes a marked swaying of the whole body. This swaying is often much greater when the patient shuts his eyes. We therefore find "Romberg's system" in multiple sclerosis precisely as in tabes dorsalis (*q. v.*). If the patient with multiple sclerosis is excited, his uncertainty and unrest usually become greater.

In two other muscular regions the motor disturbance often makes itself manifest in a very characteristic way—in the muscles of articulation (tongue) and the muscles of the eye. Even on protruding the tongue we sometimes see an uncertainty of movement, but the disturbance is still more evident when the patient speaks. Many patients with multiple sclerosis, though of course not all, show a very characteristic disturbance of speech. The speech is slow, monotonous, scanning, indistinct, and sometimes finally almost unintelligible. The patient sometimes pronounces almost every syllable, or even every sound, separately, thus: "S-w-al-low." We sometimes notice that the voice often breaks into a falsetto. There can hardly be a doubt that this disturbance of speech indicates the presence of sclerotic nodules in the pons and medulla, and that the anomaly of motion at the bottom of it is analogous to the ataxia of the arms and legs described above. The nystagmus sometimes seen in multiple sclerosis, especially when looking to one side, is also nothing but ataxia of the ocular muscles; but we must confess that we have seen manifest nystagmus in multiple sclerosis much less frequently than might be expected from the statements of some writers.

Besides ataxia and intention tremor, actual paresis or even paralysis of the muscles may be wholly absent or may be only a late symptom. At any rate, in spite of marked ataxia the gross strength of the arms and legs may be very well preserved for a long time. The thing, however, which, in addition to the ataxia, gives the motor disturbance of multiple sclerosis its special stamp is the almost constant increase in the reflexes, and the associated spastic symptoms. In the upper extremities the spastic symptoms are less prominent, but even here we almost always find very vigorous tendon and periosteal reflexes on striking the lower ends of the bones of the forearm or the tendon of the biceps or triceps. In the lower extremities we see not only a marked patellar reflex and a very intense and persistent ankle clonus, but often an evident hypertonicity of the muscles, or even a pronounced tonic rigidity of both legs.

It is the spastic symptoms which materially affect the patient's gait. If they are comparatively slight the gait is simply uncertain, wavering, stumbling, and ataxic, although even here the stiffness and jerkiness of the spastic gait is usually plainly to be seen ("spastic-ataxic gait"). The ataxia of the gait is due (unlike the ordinary tabetic gait) not to the uncertainty of the legs, but to the swaying of the whole body, as in cerebellar ataxia. If the spastic symptoms are more prominent, the gait also becomes more spastic, but the individual steps are always quick and long on account of the absence of paresis. In many cases with slight ataxia and marked hypertonicity of the muscles the gait is almost purely spastic. Only when paresis of the legs ensues does the gait become more dragging and labored, with slow, stiff steps ("spastic-paretic gait"). The ataxia is then of course no longer very noticeable.

It is not easy to explain the occasion of all these motor symptoms of multiple sclerosis thus far described, but we may certainly say this: that the persistence of the axis-cylinders in the sclerotic nodules mentioned above explains the fact that there is so often in multiple sclerosis not a real paralysis, but only ataxia (intention tremor) and spastic symptoms (increased tendon reflexes). The general tendency is to refer the ataxia to nodules in the posterior columns or in the superior leminiscus, and the spastic symptoms to nodules in the lateral tracts. The occasion of the oscillatory tremor is still wholly unknown.

Other symptoms besides the motor symptoms just mentioned are much less common in multiple sclerosis. It is especially noteworthy in respect to diagnosis that disturbances of sensation and of the bladder are usually quite subordinate. On careful testing we of course find not infrequently in some places slight or temporary diminution of the cutaneous sensibility or insignificant disturbances of micturition, but such symptoms are not part of the characteristic picture of multiple sclerosis. The cutaneous reflexes usually show no special disturbances, but we have so frequently found certain reflexes absent, especially the abdominal reflex, that we must ascribe to this symptom a certain significance in diagnosis.

A number of cerebral symptoms are far more important, although they are present only in a part of the cases. First to be mentioned are the not uncommon ocular disturbances. The disturbances of vision are the most important; these may be either a simple diminution of the acuteness of vision or a contraction of the visual field and disturbance of the color sense. These symptoms are due to an atrophy of the optic nerve, which as a rule appears first in the temporal half of the papilla. The disturbance of vision only rarely goes on to complete blindness. The atrophy of the optic nerves may sometimes develop from an optic neuritis. Motor disturbances of the eyes are also not uncommon, and sometimes they may even be the first noticeable symptom of the disease. The paralysis almost always affects the external ocular muscles only, and usually causes diplopia. It may affect one or both eyes, and sometimes there is associated paralysis. Reflex immobility of the pupils is hardly ever seen.

Of other cerebral symptoms we may mention headache and especially vertigo. The latter is often a very early symptom of the disease. It may be paroxysmal or persistent. In the course of the disease there often appears a certain mental weakness, an imbecility, which sometimes increases to marked dementia. Conditions of melancholy or exaltation are much rarer. We must also mention the occurrence of apoplectiform attacks as a symptom which is often important in diagnosis. After slight prodromal symptoms, such as headache and vertigo, loss of consciousness and hemiplegia come on quite suddenly. With this the face is usually red, the pulse is frequent, and the temperature may rise to 104° or 106° (40°–41° C.). After a day or two the consciousness gradually returns, and the hemiplegia soon disappears. Epileptiform attacks are much rarer. We saw these repeatedly in a typical case; they were mainly unilateral, and were followed by a hemiplegia which soon passed away. The precise cause of these attacks is still wholly unknown. We know only that they are not due to complicating gross changes in the brain, but that they arise merely from functional conditions of paralysis or irritation. Finally, we must mention an interesting symptom not infrequently observed in multiple sclerosis, which may also be of value in diagnosis—namely, the so-called impulsive laughing and crying. In many patients these emotional manifestations occur very frequently and in an uncontrollable fashion, either almost spontaneously or especially upon the slightest occasion. Not infrequently the two manifestations pass almost directly into each other.

In regard to the general course of typical cases, the disease develops very slowly and gradually. Motor symptoms, tremor, uncertainty of movements, and disturbances in gait, usually appear in the extremities first. The patient often complains at the same time of occasional headache and vertigo. The speech gradually becomes more indistinct, the intelligence weaker, and the other symptoms of the disease described above develop. The affection almost always lasts for years and years. Variations in the course, temporary improvement, and new relapses are common. It would even seem in mild cases as if there were sometimes a recovery or at least an arrest of the symptoms, but in general we must always regard the prognosis as very unfavorable. We often see the condition rapidly grow worse, especially after the above-mentioned apoplectiform attacks. The last stage is characterized by the gradually increasing disturbance of the general nutrition, and, finally, by paralysis and bed-sores. Death ensues from intercurrent diseases, from the increasing weakness, or sometimes in an apoplectiform attack.

Unusual Forms.—Besides the typical form of multiple sclerosis described, there are often, as we have said, cases that vary from the type (“*formes frustes*”). We will mention briefly the following possibilities:

1. The disease may be very latent. We saw one case in which, for a long time, the only symptom was a complaint of slight headache and vertigo. Finally, there was a transitory apoplectiform attack, several months later an epileptiform attack, and a few days after that death took place. The autopsy showed a completely developed multiple sclerosis.

2. Sometimes the disease appears under the exact type of a chronic myelitis. The cerebral nodules cause no symptoms, they are present, perhaps, only in small numbers, and the spinal nodules cause a gradually increasing paraplegia of the legs, with vesical disturbance, loss of sensibility, etc. We have notes of several autopsies of multiple sclerosis in which, during life, the diagnosis of a simple transverse myelitis had been made (*vide supra*, page 951).

3. Cases have been repeatedly known where multiple sclerosis has appeared under almost the exact type of a spastic spinal paralysis (*vide infra*). In these cases many nodules were situated in the lateral columns of the cord. If the spastic symptoms be combined with muscular atrophy (nodules in the anterior gray cornua), the disease may even simulate the type of an amyotrophic lateral sclerosis, with at times co-existing bulbar symptoms (*vide infra.*) If multiple sclerosis be localized to an unusual extent in the pons and medulla, the symptoms of a chronic bulbar paralysis may be prominent.

4. Much less frequently the type of the disease may resemble tabes (immobile pupils, ataxia, pain). We do not know whether absence of the patellar reflex has been seen in undoubted multiple sclerosis. There are, however, combinations of multiple sclerosis and gray degeneration of the posterior columns (syphilis?).

5. It sometimes happens that multiple sclerosis is the reason for a slowly developing hemiplegia, which may then be falsely regarded as cerebral, while the autopsy shows several nodules in the corresponding side of the cord and pons.

6. In some cases the mental disturbances (dementia) are so prominent that there is the pronounced picture of paralytic dementia with disturbances of speech, etc.

Diagnosis.—In all such atypical cases the diagnosis of multiple sclerosis is sometimes quite impossible, or at best it can be made with a fair amount of probability only when some, at least, of the characteristic symptoms of the disease are present besides the anomalous symptoms. The circumstance, indeed, that the anomalous cases will not properly fit the moulds of any other form of disease, should make us think of the possibility of a multiple sclerosis; for in these anomalous cases, of course, there may be all possible combinations of symptoms.

In typical cases, however, the diagnosis is easy, and often it can be made with absolute certainty. The slow development of the disease, usually without any discoverable cause, the comparative youth of the patient, the ataxia of the legs and the ataxia or tremor of the arms, the swaying of the body when standing with the feet together, the uncertain spastic-ataxic gait, the occasional absence of the abdominal reflex, the increased tendon reflexes in the arms and legs, the absence of any marked disturbances of sensation or of the bladder, in some cases also the disturbance of speech, the nystagmus, the impulsive laughing and crying, the slight mental weakness which often comes on later, and finally the occasional apoplectiform or epileptiform attacks—all these symptoms give a very characteristic picture, which is usually quickly recognized and correctly interpreted. It is hardly possible to confuse it with paralysis agitans (*q. v.*) if we remember the characteristic attitude of the latter affection, the oscillatory tremor of the fingers when at rest, the rigidity of the face, etc. It is very seldom difficult to distinguish it from true tabes dorsalis. It is easier to confuse multiple sclerosis with Friedreich's hereditary ataxia (*q. v.*); but the condition of the tendon reflexes is usually decisive; these are wholly absent in tabes and Friedreich's disease, but they are almost invariably increased in multiple sclerosis. It sometimes happens that hysteria assumes a form very similar to multiple sclerosis (tremor, ataxia, increased tendon reflexes, etc.), but in such cases we can usually arrive at a correct diagnosis by careful observation if we pay attention to the whole condition of the patient, the course of the disease, and the characteristic hysterical "stigmata." There is, however, a combination of multiple sclerosis and hysteria (especially hysterical hemianæsthesia), as has been maintained especially by Charcot's pupils. The distinction between multiple sclerosis and the so-called pseudo-sclerosis will be stated below.

Prognosis.—The most important features in prognosis have already been mentioned. We have seen that an improvement in the symptoms is not uncommon, but that the final outcome of the disease, with perhaps a few exceptions, is unfavorable. The disease may last for many years.

Treatment.—In the treatment we may make a trial of the same remedies that have been mentioned in the description of chronic myelitis. Absolute rest and good care, which many patients can obtain only in a good sanitarium, are often beneficial. We may also use galvanism, baths, and sponging, and internally iodide of potassium, ergotine, and nitrate of silver. The uncertainty of movement can sometimes be distinctly improved by methodical gymnastic exercises (see the following chapter).

APPENDIX

THE SO-CALLED PSEUDO-SCLEROSIS

Cases of disease have been observed, first by C. Westphal and later by the author and others, the symptoms of which seemed to warrant the diagnosis of multiple sclerosis, but the autopsy proved wholly negative. The victims are usually young and often have a predisposition to nervous disease. The symptoms of the disease consist of a marked rapid or slow oscillatory tremor with wide excursions, which sometimes occurs during rest, but which becomes unusually violent on motion. The tremor affects the arms particularly, but also the trunk and legs. There seems to be no real ataxia as in multiple sclerosis. The tendon reflexes are increased and a distinct spastic rigidity may develop in the legs. The speech is sometimes quite as decidedly scanning as in multiple sclerosis. Nystagmus is usually absent. We have seen apoplectiform attacks in a pronounced form, and also impulsive laughing and crying. The face often has a peculiar rigid expression. Certain mental disturbances are very characteristic, especially a marked tendency to outbursts of wrath and actual maniacal attacks. Signs of confusion and hallucinations are less common. The course may extend over many years. The disease seems to be incurable. Death ensues through increasing general weakness or in an apoplectiform attack. Anatomical investigation, as has been said, is essentially entirely negative, even with a careful microscopical examination of the nervous system.

The resemblance between the type of this "pseudo-sclerosis" and true multiple sclerosis is evident; nevertheless, now that we know the disease, we can often make the distinction. The chief points to bear in mind are the very marked true oscillatory tremor, the absence of real ataxia, and the mental disturbances just mentioned.

The treatment of pseudo-sclerosis is the same as that of multiple sclerosis.

CHAPTER VII

TABES DORSALIS

(Locomotor Ataxia. Gray Degeneration of the Posterior Columns. Posterior Spinal Sclerosis)

We give at present the old name of tabes dorsalis, "consumption of the spinal cord," to a perfectly definite chronic disease of the central nervous system, whose chief, but by no means sole, anatomical basis is regarded as a primary systemic degeneration of the peripheral sensory neurones, and especially of their processes in the posterior columns of the spinal cord. The first description of tabes, which, of course, is defective in many respects, is found in a work of W. Horn in 1827. We must thank especially the investigations of Romberg in Germany, in 1851, and of Duchenne in France, in 1858, for a more comprehensive knowledge of the disease, and for a precise distinction between it and the other chronic diseases of the spinal cord.

Ætiology.—But little that is definite was known until lately as to the cause of tabes dorsalis. Hereditary conditions play a very slight part in genuine cases, and even a general "neuropathic taint" can only rarely be made out in ataxic patients. Much weight in regard to ætiology was formerly laid upon previous exposure to cold. It can not be denied that sometimes the first symptoms of the disease follow some pronounced exposure to wet or cold, but much more frequently nothing of the sort can be made out. The case is similar with regard to physical and mental over-exertion, which were formerly made answerable for the origin of tabes. It is an utterly ungrounded assertion that sexual excesses may be the cause of tabes. Tabes may rarely develop as a result of acute diseases or somewhat more frequently as a result of injury (general concussion of the body from a fall, fractures, etc.); but we may ascribe to these factors merely the significance of an exciting cause. In these rare cases, too, it is hard to confirm the connection. The earlier teaching that tabes develops after "suppression of the foot-sweat" is manifestly due to a confusion of cause and effect. The absence of foot-sweat is not the cause, but a symptom of incipient tabes.

The only ætiological fact which in our opinion is beyond any doubt is the relation of tabes to a previous syphilitic infection. This relation of the two diseases to each other was first confirmed in France by Fournier and in Germany by Erb, and in spite of the vigorous contradiction which the views of these observers at first met with on many sides, this theory of tabes is constantly acquiring more and more general recognition.

The connection between tabes and syphilis can be confirmed, first of all, by statistics. Erb was able to find a history of syphilis, with secondary symptoms, in about 62 per cent. of his patients; and Fournier, in 103 cases, found syphilitic antecedents as many as 94 times. Our own observations agree exactly with Erb's data, since 61 per cent. of our patients stated definitely that they had formerly suffered from syphilis. If we also reckon the cases where the patients admit a former sore but no secondary symptoms, the percentage becomes much greater—90 per cent. In general it is worthy of note that, as a rule, in most cases of tabes the previous syphilis has not had a great intensity. Only quite infrequently do we find tertiary syphilitic symptoms as well as tabes; we have seen, for example, severe ulcers of the skin, gummatous periostitis, etc. The time between the infection and the beginning of the first symptoms of tabes varies very much; it may be from two to twenty years.

The connection between tabes and previous syphilis also explains naturally a number of peculiarities in the occurrence of tabes, especially the fact that tabes is much commoner in men than in women, that it is particularly very rare in women of the better class, while it is found much more frequently in women of the lower classes, that it comes on particularly in middle life, that it is much more common in the higher classes and in urban populations than in the country, that certain classes (merchants, artists, officers) show a much larger percentage of cases of the disease than others (clergymen, teachers, etc.). If we see, however, any cases of tabes which show striking differences in these respects, we can almost always find evidence, even in such cases, of a previous syphilitic infection. Such individual cases therefore afford material for the most convincing proof of the connection between the two diseases. Thus we sometimes see tabes in perfectly irreproachable women; here we can almost always detect previous syphilis in the husband which has been transmitted to the wife. We ourselves and other observers have seen tabes arise in this way in both husband and wife. We may also find tabes in very young or very old persons, and then we can discover that syphilis was acquired at a very early or a very advanced age. A few cases of tabes occur in children, and are in all probability to be referred to hereditary syphilis.

Although the connection between tabes and previous syphilis may be regarded as definitely established, the nature of this connection, on the other hand, is not yet fully explained. This much, of course, is certain, that tabes is not an ordinary tertiary syphilitic disease. Both its anatomical changes (*vide infra*) and the extremely slight effect of antisyphilitic treatment remove every doubt as to this. The connection between the two diseases must be of some other nature, and therefore we long ago expressed the opinion that tabes was due only to certain chemical poisons (toxines), which develop in the body from the action of the syphilitic infection. Such toxines may very well cause definite systems of fibers (*vide infra*) to degenerate, and their presence is also a good explanation of certain clinical peculiarities of tabes, especially the paroxysmal occurrence of certain symptoms (the so-called crises). Many of these crises, especially the gastric and intestinal crises, are very similar to toxic symptoms (vomiting, pain, pronounced general debility and weakness, etc.). Tabes and the closely allied general paralysis therefore stand in the same relation to syphilis as "post-syphilitic nervous diseases," that the common "nervous sequelæ" do to the acute infectious diseases—for example, as post-diphtheritic paralysis and ataxia do to previous pharyngeal diphtheria. Of course this hypothesis does not afford a completely clear insight into the nature of the tabetic process. Thus the constant advance of most (but of course not all) cases of tabes requires a further special explanation. We may perhaps imagine the process thus: that the toxines absorbed at first merely injure the affected nervous system (*vide infra*), but that the complete atrophy and cessation of function comes on gradually through the wear and tear associated with the normal functioning of the nervous elements. In this sense other external influences (fatigue and physical over-exertion, exposure to cold, injury) may be regarded as aiding the process.

It can not at present be maintained that every tabes is of syphilitic origin, although we have never yet seen a typical genuine tabes in which syphilis could certainly be excluded; but it would not be impossible that other noxious influences might cause the same systems of fibers to degenerate as in tabes, and thus produce a form of disease similiar to that of genuine tabes. Thus we know at present, from the investigations of Tuczek, that symptoms may develop in chronic ergot poisoning (ergotism) which are perfectly analogous to those of tabes and depend upon a corresponding disease of the posterior columns of the cord which can be demonstrated anatomically. These cases of "tabes," however, which are traced to some other cause, must later be distinguished from the post-syphilitic cases which constitute by far the great majority.

Pathological Anatomy.—As we have seen on page 796, the cell bodies of the peripheral sensory neurones lie in the spinal ganglia. The processes of the spinal ganglion-cells divide into two branches. One goes to the periphery of the body, the cutaneous surface; it forms the so-called peripheral sensory nerve. The other process serves for the centripetal conduction of sensory irritations; it enters the cord through the posterior roots and goes, as we have said above, either upward in the posterior columns or into the posterior gray cornua, there to connect with spinal sensory neurones of the second order. Upon this peripheral (first) sensory neurone system (spinal ganglion system), whose anatomical arrangement we have just briefly described, the (toxic) cause of tabes acts especially and causes it slowly, fiber by fiber or neurone by neurone, to degenerate. In tabes, as in most other primary systemic diseases, we find that the evident visible anatomical degeneration of the different neurones always begins in the prolongations, and the destruction of the cell body itself follows later, if at all, and is the last process. Since the prolongations of the spinal ganglion-cells are, first, the peripheral sensory nerves, and, second, the fibers in the posterior columns, and, in part, in the posterior horns of the spinal cord, we understand, without further explanation, that the anatomical process in tabes must manifest itself as a degeneration both in the peripheral nerves and in the posterior columns and posterior horns of the spinal cord. So far as we can at present appreciate the conditions, the spinal process of the neurone cells in the spinal ganglia seem to be affected earlier, more regularly, and more extensively than the peripheral process; but it seems to us probable that there are individual differences in this respect, and that many differences in the course of the disease in different cases of tabes may be explained by

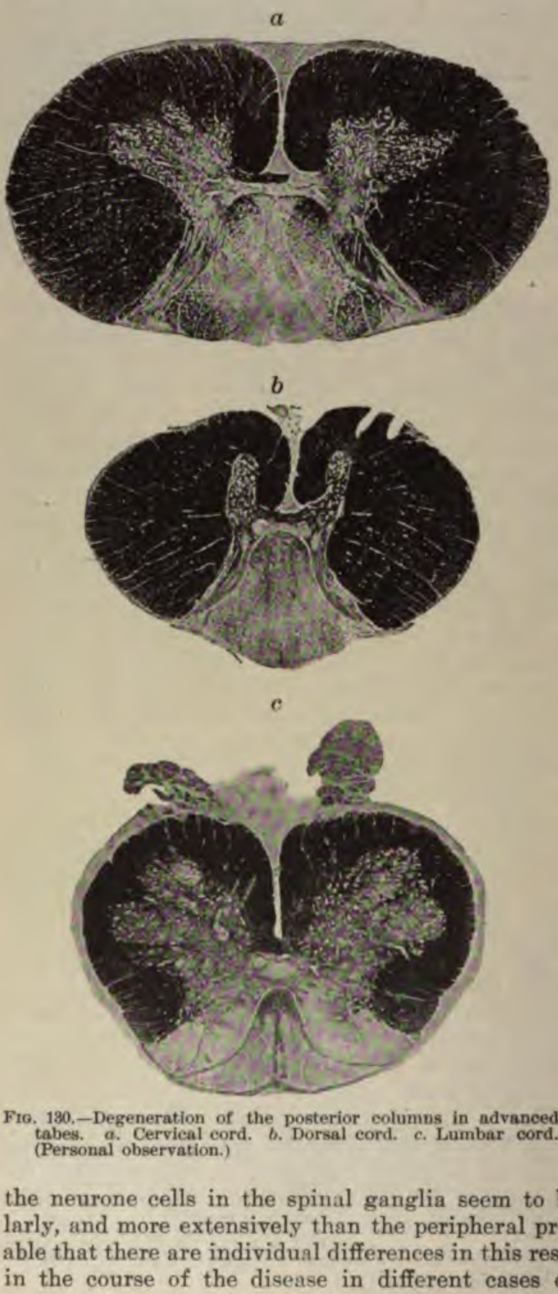

FIG. 130.—Degeneration of the posterior columns in advanced tabes. *a*. Cervical cord. *b*. Dorsal cord. *c*. Lumbar cord. (Personal observation.)

such variations in the particular anatomical localization of the tabetic degeneration.

If we examine the spinal cord of a patient who has died in the advanced stage of tabes, the smallness and thinness of the cord usually strike us at once. The pia mater is thickened and opaque, especially on the posterior surface. We often see the posterior columns appearing through the pia as a gray band extending the whole length of the spinal cord. On cross-section we notice that the smallness of the cord is due chiefly to the atrophy of the posterior columns, which is often very considerable. These no longer protrude at all backward, as they normally do, but seem flat and sunken. From their pronounced gray color they are very plainly distinguished on cross-section from the rest of the white matter of the cord. The posterior cornua of the gray matter, and the posterior nerve-roots, show exceptionally a considerable atrophy, and appear very small and thin and also of a gray color.

Microscopic examination gives more exact information as to the extent and form of the degeneration. This shows (Fig. 130, *a*, *b*, *c*) that all portions of the posterior columns are not affected in like manner, but only those portions which contain the processes and collaterals of the posterior root-fibers, which therefore belong to the system of spinal ganglion neurones. The other tracts (short tracts, commissural fibers) are spared. The degeneration is almost always most intense in the lumbar cord; here it affects chiefly the middle and posterior portions of the posterior columns, while the most anterior portion and the so-called dorso-median sacral bundle (Fig. 130, *c*) remain normal in almost all cases. These are indeed fibers which have another origin and do not belong to the spinal ganglion system. In the dorsal cord (Fig. 130, *b*) the posterior columns are almost completely degenerated. There are usually small normal areas still preserved in the posterior external and the most anterior portions. In the cervical cord (see Fig. 130, *a*) the so-called columns of Goll are chiefly affected, together with the prolongation of the fibers from the root-zones of the lumbar cord, and also the "lateral root-areas"—that is, those portions in the columns of Burdach where fibers enter directly from the posterior nerve-roots, and from which fibers may be traced farther into the gray matter of the posterior cornua; but the so-called posterior external areas, and also two little antero-lateral areas, remain entirely, or at least for a long time, free from the disease, because these are also bundles of fibers which do not belong to the system of the spinal ganglia or posterior roots. If we examine cases of tabes in the earlier stages which happen to come to autopsy, we find in the posterior columns only a few root territories affected—that is, the fibers arising from individual spinal ganglia or posterior roots and lying near one another, while other root territories remain free from degeneration (Figs. 131 and 132). A system of very fine fibers, entering through the posterior roots, is affected very early, as a rule. It branches outward immediately after the entrance of the roots, and occupies a small but very sharply defined territory at the point of the posterior cornua, between the posterior and lateral columns (the so-called zone of Lissauer). In all the affected root territories the degeneration slowly involves fiber after fiber. The order in which the different root territories

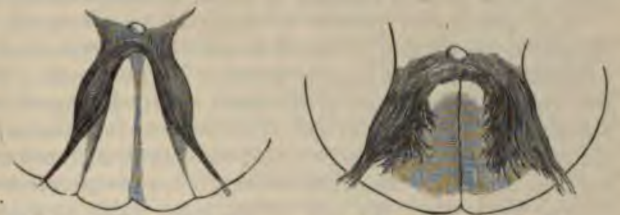

FIG. 131.—Dorsal region. FIG. 132.—Lumbar region.

Transverse section through the posterior columns of the cord in beginning tabes dorsalis.

degenerate, after one another and in part at the same time with one another, is certainly not always the same in individual cases, as is evident from the variations in the clinical course of the disease. As a rule, the neurones of the lumbar and lower dorsal spinal ganglia certainly degenerate first, and the neurone systems of the cervical ganglia later.

We must state, with reference to the participation of the gray matter in the disease, that the posterior cornua, as we have already said, are also found considerably affected, which is explained mainly by the atrophy of the posterior root-fibers which enter them directly. It also can not appear strange that the medullated fibers found in Clarke's columns seem very much reduced in number, since they also are direct processes of the posterior root-fibers. The cells of the columns of Clarke remain normal. The ganglion-cells of the posterior horns, which belong to the second sensory neurones, also remain normal, and no changes are found in the processes of these cells in the lateral columns. The lateral and anterior columns of the cord usually remain unaltered (*vide infra*).

On the other hand, the peripheral sensory nerves—that is, peripheral processes of the spinal ganglion-cells—are by no means spared. In all cases of tabes which have been carefully examined thus far, we find in the peripheral nerve-trunks (sciatic, crural, etc.), and usually still more in the finer branches of the sensory nerves, a considerable number of degenerated centripetal fibers (Dejerine, Oppenheim and Siemerling, and others). We regard this degeneration as precisely analogous to the disease of the posterior columns and by no means a secondary degeneration. Like the degeneration of the posterior columns, it also is an expression of the primary disease of the peripheral sensory neurone system. In regard to the cell-bodies in the spinal ganglia, those certainly are well preserved for a long time, at least in regard to their external aspect, but finally we may find more or less definite changes in them, although hardly ever complete atrophy.

The anatomical process of tabes is by no means limited exclusively to a chronic degeneration of the first sensory neurone system. Other neurones and neurone systems may also be involved in the disease. The occasional affection of the trigeminal neurones is of course wholly analogous to the degeneration of the sensory spinal neurones; but other regions sometimes become degenerated, especially the optic nerve, more rarely the vagus and acoustic, and also certain motor regions, especially the ocular nerves, other peripheral motor nerves, the pyramidal lateral tract, etc. We will return more fully to these "complications," or, to speak more correctly, to these rarer localizations of the tabetic process, in the clinical portion of this chapter. If we consider, however, that the whole neurone territory of the spinal ganglia evidently contains neurones of very different physiological importance (fibers for the conduction of the different qualities of sensation, for the co-ordination of movement, for the innervation of the bladder and rectum, for reflex processes, etc.), and that also other most diverse neurone territories may be involved in tabes, we can understand that tabes can be termed not a simple, but far more properly a "*combined systemic disease.*"

The form of the tabetic affection consists of a primary degenerative atrophy of the nerve-fibers, and a corresponding secondary increase of the connective tissue. The gray color of the posterior columns is due to the loss of the medullary sheaths. Since the destruction of the nerve-fibers advances but very slowly, we find only a few fatty granular cells. In old cases we find numerous corpora amylacea, whose origin and significance are still unknown. The thickening of the pia mater is a purely secondary phenomenon, which is always most marked on the posterior surface of the periphery of the cord, corresponding to the atrophy of the posterior columns.

Clinical History.—A disease which has as its basis so definite and strictly limited an anatomical change as is the case with tabes dorsalis would also be expected to give a very characteristic clinical picture. This supposition is entirely correct, and there are few diseases which can be diagnosticated, even in their earliest stages, with as much certainty as tabes. This fact is explained only by regarding tabes as a systemic disease, in which certain systems of fibers are always affected, while others are as constantly spared by the disease. The difference between individual cases of tabes lies, therefore, less in the symptoms themselves than in their intensity, their duration, and the order of their occurrence. In this regard, however, the differences in the clinical pictures are extremely varied, so that, even with a comparatively great personal experience, we often see new combinations of symptoms and also peculiarities in their course.

For the majority of cases we may sketch the following general description of the disease, in which it is better to divide the whole course into several stages; but, of course, this division can have only a schematic value.

Tabes dorsalis begins, as a rule, with a stage of initial symptoms, which develop very gradually and insidiously, and which may be of very varying duration. The most characteristic symptoms of this stage are those of sensory irritation, most frequently in the form of the so-called lightning-like, "lancinating" pains in the lower extremities. They are sometimes very severe, but at other times only of slight intensity, and are comparatively little noticed by the patient, who regards them as "rheumatism." Many patients have a feeling of numbness and tingling in the tips of the fingers, especially of the ring and little fingers, and there is often a pronounced girdle sensation in the trunk. In some cases, too, neuralgic and migraine-like pains in the head may appear in the early stages.

Besides these symptoms of sensory irritation, which may often be for years the only symptoms of which the patient complains, two objective symptoms appear very early, which are of the greatest importance in the diagnosis of incipient tabes: the disappearance of the patellar reflex and the reflex immobility of the pupils. The absence of the patellar reflex is the most constant of all the known symptoms of tabes. It is, as a rule, to be found so early that we can hardly ever decide with exactness upon the time of its occurrence. The reflex immobility of the pupil—that is, the failure of the pupil to contract to light, while the changes on accommodation may be perfectly retained—is, indeed, not so constant as the failure of the patellar reflex, but it is present in almost all cases. If all three symptoms—lancinating pains, absence of patellar reflex, and immobility of the pupils—are present at the same time, the diagnosis of tabes is absolutely certain, even if all other symptoms are wanting, because this peculiar combination of three such apparently heterogeneous symptoms is seen in this disease alone. If only two of these symptoms are present, but if the reflex immobility of the pupils be one, the diagnosis is also practically certain; but if this symptom be absent we should be very cautious in admitting that the affection is genuine tabes.

Among the rarer initial symptoms we shall also learn to recognize diplopia, caused by paralysis of certain ocular muscles, and loss of vision, from optic atrophy. In many other cases certain disturbances of cutaneous sensibility (a girdle feeling, paræsthesia, a furry feeling) announce the approach of the disease. Sometimes disturbances in micturition appear quite early, while in other cases gastric crises (*vide infra*) or some joint affection are the first symptoms which the patient notices.

After this first stage of the disease has lasted for a very varying period, from a few months to two or five or even twenty years, the second stage begins; this we usually term the ataxic stage of tabes.

The beginning of this stage is recognized by the appearance of disturbances of gait. The gait becomes more difficult and more uncertain, and there are certain peculiarities which we will describe more fully later. Careful examination shows that the disturbance in gait is due not to a paresis of the muscles but to a disturbance of co-ordination, ataxia of the lower extremities. This symptom usually increases very slowly, until it reaches a degree where the patient can walk only with assistance, and finally can not walk at all. There is often later, but almost always not for years, ataxia of the upper extremities.

Besides the persisting symptoms of the first stage, there are often now more marked disturbances of sensibility, as well as ataxia. The patient has a feeling as if he were walking on wool, felt, or similar substances. If he closes his eyes there is great swaying of the whole body—"Romberg's symptom." Physical examination of the sensibility almost always shows considerable disturbance of the cutaneous sensibility, especially of sensibility to pain, or even of other forms of sensibility. A loss of muscular sense is very common. The disturbances of micturition, such as incontinence, gradually become more marked, and very often cystitis gradually develops. This stage also may last for years. Sometimes the disease seems to stand still, frequently even manifest improvement is seen, but then the condition becomes worse again.

The third stage, the terminal stage of the disease, develops if the patient has not previously succumbed to an intercurrent disease. The symptoms are the same as in most of the other chronic diseases of the spinal cord. The patient gradually becomes more and more wretched and helpless, and finally is confined almost wholly to his bed. The ataxia reaches an extreme degree, and finally there is sometimes an actual motor weakness or complete paralysis of the legs. In these cases, which are by no means frequent, we are correct in calling the third stage of tabes the "paralytic stage." A severe pyelo-cystitis usually develops, bed-sores appear, and death finally frees the patient from his lamentable condition.

We must now complete this briefly sketched picture of the disease by a more careful description of the single symptoms.

1. Disturbances of Motility in the Extremities.—The typical motor symptom of developed tabes dorsalis is the disturbance of co-ordination, the ataxia (see page 837). This is almost always seen in the lower extremities first. To test the ataxia it is most convenient to ask the patient to touch the knee-pan of one leg with the heel of the other foot ("knee-heel test"). We see then that the leg moved is often carried beyond the point designated several times before it reaches it. The ataxia is often noticeable, even in crossing one leg over the other, as the leg raised makes much too great and too "throwing" a movement.

The alteration of the gait is very characteristic—the ataxic gait, from which we can often perceive the patient's disease at the first glance. If the patient sits down and tries to get up again to walk, there is difficulty in rising. He separates his legs to find a firm point of support, he takes a stick to help himself if he can, and he often gets the proper balance to keep himself erect only after several attempts. The gait itself is straddling, and the legs are raised abnormally high and set down with a stamp. The direction of the gait, however, is as a rule well preserved, and the trunk (*vide infra*) is often but little involved in the swaying. This is the distinction between the ataxic gait of tabes (due to ataxia of the legs) and the staggering, wavering gait in cerebellar disease ("cerebellar ataxia" with ataxia of the legs and trunk), in multiple sclerosis, etc. If we have the patient turn rapidly or make a proper military "about face," the uncertainty of movement is still more marked, as it also is if the patient rises suddenly and begins to walk or makes a sudden halt when walking. These methods of testing are therefore especially suitable for ascertaining the first beginnings of ataxia. Very often the first uncertainty in the gait is noticed in going down-stairs, because the patient can not put his foot securely on the next step below. Going up-stairs is at first far less affected because the perpendicular rise of the stairs prevents any excessive movement. If the ataxia reaches a higher degree, most tabid patients always walk with a stick and control the movements of their legs by keeping their eyes fixed on the floor as they walk. This control is particularly necessary when the sensibility of the legs, especially the muscular sensibility, is diminished at the same time.

In more advanced cases the ataxia becomes apparent even on standing with the feet close together (static ataxia). We see continually in the muscles and tendons of the legs slight contractions which are necessary to maintain the constantly shifting equilibrium. These contractions become much more marked as soon as the patient stands with his eyes shut. There is then usually an evident swaying of the body, which is sometimes so great that the patient is in danger of falling. This phenomenon (Romberg's symptom) is due chiefly to the defective control of the muscular movements, which are necessary to preserve the equilibrium, as a result of the impaired sensibility of the skin of the soles of the feet and that of the joints and the muscles themselves. If this control be supplied by the eyes, the swaying is insignificant, but it at once becomes more marked if the control by the eyes is lost. From a like reason it is much harder for most ataxic patients to walk in the dark than by daylight.

In many cases of tabes, especially when the disease is far advanced, the co-ordination of the maintenance of the position of the trunk is evidently disturbed. Such patients finally can not sit up, since the trunk sways so much. The gait of tabid patients with great ataxia of the trunk is still more uncertain and also plainly staggering.

If ataxia of the upper extremities occur in the course of the disease, it is easily recognized if the patient tries to take hold of some definite object, such as his ear, or if he brings the tips of his two forefingers together from a certain distance, or if he tries to touch exactly with the tip of his finger the finger of the physician when held before him. The ataxia is of course also apparent in all fine complicated movements (eating, sewing, writing, tying a knot, drawing straight lines, etc.). The movements are irregular and uncertain, and the excursions are marked. If there be at the same time any sensory disturbance in the arms, the anomaly in their movements is still greater with the eyes shut.

There has been much written and much argued as to the cause of the ataxia in tabes dorsalis. The main reason why it is at present impossible to give an incontrovertible explanation of the occurrence of ataxia lies in the fact that we are not yet in a condition to know and to analyze with sufficient accuracy the process of normal co-ordination of motion (page 837); for manifestly every theory as to the causes of ataxia must begin with the processes involved in the co-ordination of normal movements. If we try to get a clear idea of this, the most essential point to bear in mind is that co-ordination of motion is not a congenital function, but a power of our organs of motion acquired by practice. The movements of little children who are learning to walk are ataxic, and even in later life it often happens that we have to learn how to perform certain complicated and difficult movements (so-called manual dexterity, exercises of skill). We can get no other idea of this "learning how to co-ordinate" than that it takes place by the aid of the constant action of controlling and correcting impressions coming from the periphery—that is, centripetal—but we must bear in mind that these actions are mainly unconscious. The surer we become in the execution of the movements, the more the regulatory influence of the centripetal irritations falls into the background, without ever wholly disappearing. In these cases we must not consider, by any means, merely the irritations which are brought to the central organs from the skin of the parts moved; but we should consider, just as much or even more, those irritations which are due to the varying tension and position of the deeper parts, the muscles, the fasciæ, the ligaments, and the articular surfaces. Even the special organs of sense, particularly the eye, under some circumstances, assist materially in regulating motion.

A disturbance of co-ordination must accordingly take place when this constant regulation of our movements by centripetal irritations ceases. Since the anatomical lesion in tabes, as we have seen, consists almost exclusively of a degeneration of centripetal systems of fibers passing through the posterior roots, there can scarcely be a doubt in our opinion that tabetic ataxia is due to the degeneration of centripetal fibers regulating movements; but we must emphasize the fact that these centripetal co-ordinating fibers must not be identified without further proof with the centripetal fibers serving for conscious sensibility, for we can by no means establish a parallelism between the degree of ataxia and the severity of the sensory disturbances (in the skin and in the muscles), although of course disturbances of co-ordination and disturbances of sensibility very often co-exist in this disease. There are, however, undoubtedly cases of tabes with marked ataxia of the legs in which we can find on examination only slight disturbances of the cutaneous and muscular sensibility. This is still more frequently the case in other diseases attended with ataxia.

We may therefore very well imagine that ataxia is the consequence of a degeneration of those collaterals which go from the centripetal neurones to the motor ganglion-cells, and there provide for the regulation of the strength of motor innervation, although there are still enough conducting paths to conduct sensory impressions to the centers of consciousness. We do not as yet know, however, in what part of the motor tract the regulatory influence of centripetal irritations takes place; whether in the cells of the anterior horns of the cord, which is the simplest explanation of the theory of tabetic ataxia, or higher up in the motor cortex or in part in the cerebellum, etc. This can be made perfectly clear only by further investigation.

Ataxia is the chief motor disturbance in tabes. The crude strength of the muscles may be perfectly normal, and it is chiefly a service of Duchenne's to have made clear for the first time the principal distinction between ataxia and paralysis. He showed that ataxics, who can no longer walk a step alone, can nevertheless exert the greatest strength with their legs. We have ourselves treated a teacher of gymnastics who, in spite of the most marked ataxia of the arms, had still so much strength in them that he could support himself in bed on his arms and keep his whole body, with his legs extended, in the air. It sometimes happens, however, that the crude strength also disappears in tabes, and that the muscles become paretic. There may be a simulation of a certain "pseudoparesis," since the patient with severe ataxia loses the ability to concentrate his muscular strength in the necessary direction. There is also in tabes occasionally, as we have said, an actual paralysis of the legs ultimately. We have stated above that even a complete paraplegia may finally develop in the course of the disease. In these cases we find, on anatomical examination, that the process is no longer confined to the posterior columns, but that there is also a systemic degeneration of the lateral pyramidal motor tracts in the lumbar cord. In some cases paraplegia has been observed to come on quite suddenly even in the early stages of the disease, and usually to disappear again in the course of a few weeks. Its origin has not yet been explained. The rare paralyses of certain motor nerves have another significance. They have usually been noted at the beginning of the disease, and affect the radial, peroneal, accessory, etc. They are probably due to changes in the affected peripheral nerves, but as a rule they are transitory, and, in our opinion, so far as they are not accidental complications (such as pressure paralyses), they are to be put in the same category with tabetic oculo-motor paralyses (*vide infra*).

We may add, finally, that slight symptoms of motor irritation, slight twitchings in the muscles, especially in the fingers, are not uncommon, but they are noticed only when the attention is especially directed to them. It is not certainly known how they arise; perhaps they are of reflex origin, or perhaps they are connected with the ataxia and loss of muscle sense.

The condition of the muscles on passive motion is very characteristic. We notice in most cases a very striking flaccidity of the limbs, so that there is hardly any muscular resistance to be felt. In bedridden tabid patients we can often, for example, flex the leg with ease at the hip far enough to touch the head. There is evidently a diminution of muscular tonus, a so-called hypotonia of the muscles. Since there are many reasons for believing that the normal muscular tonus is of reflex origin, we are led to think of a connection between the absence of muscular tonus and the loss of centripetal irritations. The diminution of the muscular tonus in tabes is evidently in close relation to the absence of the tendon reflexes.

The electrical excitability of the nerves and muscles remains perfectly normal in uncomplicated tabes.

2. Disturbances of the Cutaneous and Muscular Sensibility.—As we have already said, tabes begins, in the great majority of cases, with symptoms of sensory irritation, which usually persist in the later course of the disease. Besides the simple paræsthesia—the feeling of tingling and numbness in the legs, and sometimes, too, a similar feeling which appears quite early in the upper extremities (especially often, as we have said, in the ulnar region)—the tabetic pains are remarkably characteristic of the disease.

The intensity of the pains differs very much in different cases; but we very rarely see a complete absence of them. The patient's attention is often first called to his slight and infrequent pains by direct questioning; but in some cases the severe pains are a constant distress to him. The pains most characteristic of tabes are the lightning-like, "lancinating" pains, which shoot like neuralgic pains for some distance along the course of the nerves. They often come on in very severe paroxysms ("pain crises," sometimes associated with considerable disturbance of the general condition, loss of appetite, etc.), last one or more days, and then disappear for a shorter or longer time. There are also boring, stabbing pains, which are fixed at one point and have their seat especially in the vicinity of the joints; and finally "constricting pains," which are felt most frequently in the back and loins. The well-known "girdle feeling" of tabid patients —that is, the sensation of a band tightly encircling the trunk, or a tight, "drawn-together" pressure on the lateral portions of the trunk—is a similar symptom of sensory irritation. The girdle feeling is manifestly due to irritative processes in the region of the lower dorsal or upper lumbar nerves. Since it is comparatively frequent, and often appears quite early, it also has a certain diagnostic significance.

The tabetic pains almost always show themselves first in the legs. but paræsthesia also occurs, and often very early, in the intercostal region (girdle sensation). In the ulnar region quite analogous pains sometimes appear, and in very advanced cases we have also observed pains in the region of the occipital nerves and of the trigeminus. On the other hand, neuralgic pains in the face, especially in the region of the frontal nerve, or in the occiput, or even migraine-like attacks, also occur, even in the initial stage of tabes, as we have ourselves observed. In some cases the lancinating pains in tabes may be accompanied by the appearance of herpes zoster.

Usually much later than the pains appears also a diminution of sensibility which can be made out objectively. As a rule, it may be stated that in most, but not in all, cases of tabes the sensibility does not remain normal; although well-marked anæsthesia never appears until the more advanced stages of the disease.

The form of the disturbances of sensibility varies extremely, and no disease furnishes so many opportunities for the study of interesting details in the region of anomalies of sensation as tabes dorsalis. Our knowledge of the occurrence of partial paralyses of sensation especially is very largely based on the examination of tabid patients. In order to form a correct opinion upon the condition of the cutaneous sensibility it is therefore absolutely necessary to test carefully all the different qualities of sensation. Beginning with the examination of simple sensibility to contact (tactile sense), we often find this very well preserved even in advanced cases. There is often, of course, a certain blunting of sensibility. A slight touch with a camel's-hair brush is often, even in the early stages, no longer felt, or felt only very indistinctly, on the lower legs and feet, or on certain zones on the trunk (*vide infra*). The disturbance of the sensibility to pain is more marked. A blunting of the sensibility to pain on the legs is one of the most constant and early symptoms of tabes, but, in order to determine it, it is absolutely necessary to test at first only by brief pricks. We then find, as a rule, that such pricks can not be positively distinguished from a touch with the head of the pin or with the finger, because the specific sensation of pain from the pin-prick is lost. The case is quite different if we keep up a continuous pin-prick. In that case the patient at first usually feels only a painless touch, but after a short time (sometimes only some seconds later) there ensues often considerable pain accompanied by a reflex twitch in the leg pricked. This symptom was formerly explained by Naunyn, E. Remak, and others as "double sensation," due to "delayed conduction of the sensibility to pain," but, in our opinion, it is dependent merely upon the slow summation of pain irritation which requires a longer time (*vide* page 793), but there is also perhaps a delay in conduction.* Only in far-advanced cases can the analgesia, especially in certain parts of the skin, reach so great a degree that even continuous painful irritants can no longer excite a sensation of pain. The patient may be nearly or wholly insensible to deep pin-pricks or strong faradic currents.

The after-sensations of tabid patients are another peculiar symptom. It may happen that a patient after a single prick may have five, six, or more painful after-sensations at varying intervals.

Disturbances of the sense of pressure, and still more that of temperature, are also quite frequently found, especially as partial paralyses of sensation, when the sensibility is otherwise well preserved. We should note particularly the occurrence of partial anæsthesia of the sensibility to heat or cold. On the other hand, the sensibility to temperature may sometimes be very keen, while in other respects there is quite a high degree of anæsthesia. Fischer has termed a peculiar disturbance of sensibility occurring in tabes, polyæsthesia; the patient asserts, when examined with an æsthesiometer, that he feels several (four or five) points, although he was touched with but one.

The considerable anomalies of the muscular sense, which are often to be made out in more advanced cases, have a special interest (see page 794). If the patient shuts his eyes, he is often entirely unaware of the situation and position of his limbs. He makes an indefinite or wholly false report as to the direction and extent of passive motions.† If the muscular sense in the arms be disturbed, and we put the arms into any unusual position, the patient has considerable trouble in bringing the hands together with his eyes shut. He gropes about in the air with his arms until he accidentally touches one arm with the other hand, and then he feels down this to the hand. The action of ataxia and muscular anæsthesia are accordingly combined in these cases. Pitres has lately described peculiar attacks of muscular rigidity, and attacks of a decided feeling of fatigue in the muscles, apparently coming on spontaneously in incipient tabes ("*crises de courbature musculaire*").

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* If, while the patient's eyes are shut, we prick the leg and the arm or neck as nearly as possible at the same time, the prick on the leg would necessarily be felt much later than that on the arm, if there is a delayed conduction of sensory impressions from the leg; but we have never as yet been able to make out this condition with certainty.

† We can describe different letters and figures in the air with the patient's extremities, and try whether they can be correctly recognized with the eyes shut.

The limitation of the anæsthetic or hypoæsthesic cutaneous territories is of much interest, if we consider them with regard to the territories of distribution of the different spinal ganglia (page 924 *et seq.*). We would mention in particular that in the very early stages of the disease we can often find one or more girdling zones of hyperæsthesia [or hypoalgesia] on the trunk, especially in the region of the upper and middle dorsal nerves (Hitzig and Lähr and others). In the legs very anæsthetic and more sensitive regions sometimes alternate.

Only in rare and far-advanced cases is there finally a complete anæsthesia of the lower, and exceptionally of the upper extremities. We then see also at times disturbances of sensibility in the region of the trigeminus, in the skin of the face. We must also mention the fact that the peripheral nerve-trunks, especially the ulnar nerve at the elbow, are often insensitive to direct pressure (ulnar phenomenon [Biernacki's symptom]).

That the cause of all these disturbances of sensation is to be looked for in the disease of the peripheral sensory neurones needs no further argument. It is a question only what share the peripheral nerves and what share the spinal fibers and cells have in the causation, and how far in general such a localization of the symptom is justified. In general we regard it as most probable that the tabetic pains and paræsthesiæ are due to irritation in the processes of the spinal ganglia leading to the periphery—that is, in the sensory peripheral nerves. Anæsthesia must of course arise from any degenerative destruction of sensory fibers. If there is marked anæsthesia, and especially analgesia and thermoanæsthesia, we may always assume an atrophy of the root-fibers entering the posterior horns. With sensory disturbances in the region of the trigeminus, we may look for anatomical disturbances in the neurones of this nerve.

3. Disturbances of the Reflexes.—The cutaneous reflexes show no constant changes in tabes. They are usually approximately normal, but sometimes they are diminished, especially if there be at the same time a marked disturbance of sensibility. In many respects their condition corresponds to the condition of the sensibility to pain. The abdominal and cremaster reflexes are often preserved for a very long time.

The absence of the tendon reflexes, especially of the patellar and Achilles tendon reflexes, is, however, an almost constant sign of tabes, and is a sign of the highest diagnostic value. As we have already said, the disappearance of the patellar reflex is one of the earliest symptoms of the disease, and is therefore of the greatest significance in the diagnosis of initial tabes. Since this symptom was first discovered by Westphal it is not infrequently called "Westphal's sign." There are certainly cases of tabes in which the patellar reflex may persist for a long time, in spite of the development of many other morbid symptoms. These, however, are only very rare exceptions, which do not disprove the rule, and do not at all contradict our general views as to tabes. In individual cases those fibers which serve to set free the reflex may be spared for a long time, just as any other characteristic symptom of the disease may under some circumstances occasionally be absent. Concerning the precise anatomical cause of the disappearance of the patellar reflex, it can be due only to a degeneration in the centripetal portion of the affected reflex arc—that is, only in the fibers which belong to the territory of the posterior roots. It is in accordance with this that disease of the middle portion of the posterior columns in the lumbar cord (that is, the root-zones, see Fig. 132) always seems to be accompanied by a failure of the patellar reflex. The direct mechanical irritability of the muscles, especially of the quadriceps, is almost always retained in tabes.

4. Disturbances in the Eye and the Other Organs of Special Sense.—The justification for regarding tabes as a combined systemic disease lies in the frequency with which certain cerebral symptoms, as well as the spinal, are found in it.

The symptoms in the eyes deserve the first attention. We find, of course not in all cases, but still in the great majority of them, that the pupils show no trace of contraction to light, although the well-known changes in the pupils are, as a rule, yet not invariably, very manifest upon any variation of the accommodation—dilatation of the pupils on looking at distant objects with approximately parallel axes of vision and contraction of the pupils on fixing a near object with the greatest possible convergence of the eyeballs. We give this phenomenon, first described by Argyll Robertson, but the precise anatomical cause of which is not yet known, the name of reflex immobility of the pupils with retained mobility on accommodation [reflex iridoplegia]. It is a very characteristic feature that the immobile pupils are often not perfectly round, but on careful inspection seem rather irregular or polygonal. As we have already said, the immobility of the pupils is often a very early symptom, so that it also has a very great diagnostic importance, the more so that it is rarely found except in genuine tabes and the allied general paralysis. In regard to the average size of the pupils, they are often about normal, but we very often find both pupils unusually contracted (spinal myosis) or more rarely dilated. Quite frequently we find in tabes inequality of the pupils and, as a rare symptom, a striking change in the size of the pupils, so that first one and then the other pupil is the larger ("springing pupil"). We may also add that the dilatation of the pupil which is normally produced by painful irritation of the skin of the cheek is often absent in tabes (Erb).

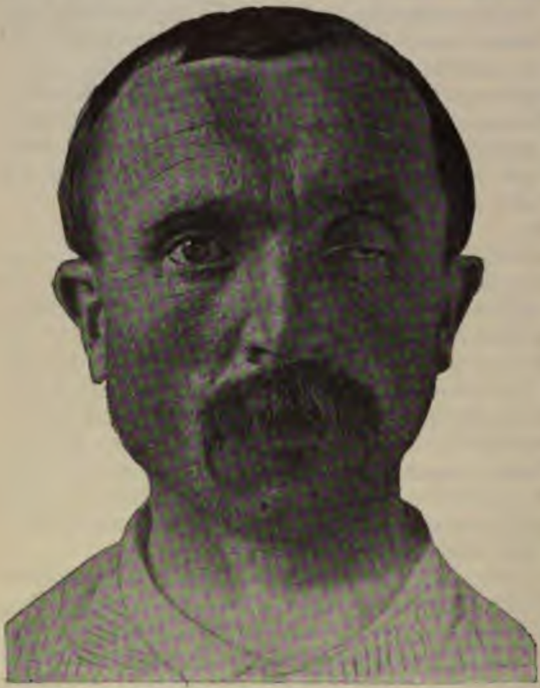

Fig. 133.—Paralysis of the left oculo-motor nerve in tabes. (Erlangen Medical Clinique.)

The paralyses of the ocular muscles in tabes are also very interesting (see Fig. 133). They are usually unilateral, but sometimes bilateral, and often appear even at the beginning of the disease, so that diplopia may be the first subjective symptom of which the patient complains. In every sudden oculo-motor or abducens paralysis, coming on without any other cause, we must think of the possibility of an incipient tabes. It is remarkable that these paralyses in many cases disappear permanently and entirely after some time, but it is always possible that they may recur later. We once saw a case of tabes with pronounced periodical oculo-motor paralysis (*vide* page 857). The oculo-motor paralysis, however, when it has once appeared, may be permanent, as we have repeatedly seen, especially in a case with bilateral abducens and unilateral oculo-motor paralysis, and also in a case with almost complete bilateral oculo-motor paralysis. In the autopsies on such cases we find the trunks of the affected nerves and their nuclei markedly atrophic, but it is very probable that the temporary ocular paralyses in tabetic patients depend upon changes in the peripheral nerves of the ocular muscles.

The third complication in the eyes in tabes is optic atrophy. It occurs in about ten or fifteen per cent. of all cases, and is usually an initial symptom, at a time when the absence of the tendon reflexes, which may usually also be noticed, is the only thing, except this, to render the diagnosis of the disease possible. The patient complains of diminution of vision; and the power to distinguish colors, especially green, disappears quite early. On objective examination, we find, besides this anomaly in the color sense, usually a limitation of the field of vision, and the beginning gray degeneration of the optic nerve can easily be made out on ophthalmoscopic examination. The affection sometimes makes little halts and slight apparent improvements, but it usually ends with complete blindness. The optic atrophy more rarely may not appear until the later stages of the disease, when all the other symptoms are already fully developed.

Auditory disturbances are much rarer than those of sight, but they also occur. The cause, in at least a part of the cases, is an atrophy of the acoustic nerve. We had a patient with tabes under treatment for a long time who was finally entirely blind in both eyes, entirely deaf in both ears, and almost entirely anæsthetic up to the forehead. The only possible way of communication was by writing the different letters of each word on his forehead with the finger! Symptoms are also frequently seen which resemble those of Ménière's disease—tinnitus, vertigo, and deafness.

Changes in the senses of taste and smell have been observed only in a few cases.

5. Disturbances in the Bladder, the Rectum, and the Sexual Organs.—Difficulty in emptying the bladder is an almost constant symptom in the later stages of tabes. It sometimes, indeed, appears very early. The patient feels the desire to urinate more frequently than normal, there is often a slight involuntary micturition or dribbling of urine, and at other times there is retention of urine, sometimes coming on quite suddenly; in advanced stages there is frequently complete incontinence. A cystitis very often develops as a result of all these disturbances, which may be the starting-point of severe pyelo-cystitis and pyelonephritis, and thus be the cause of death.

Persistent constipation is also a very frequent symptom of tabes, the reason for which is perhaps to be sought in the defective reflex excitation of intestinal peristalsis. The constipation may in many cases give rise to very painful sensations in the loins and abdomen. The coccygodynia, which sometimes occurs in tabes, has already been mentioned (see page 820). Incontinence of fæces occurs quite rarely in the last stages of the disease.

A diminution in sexual power is found almost constantly in advanced cases; and it is also often one of the initial symptoms.

6. Symptoms in the Internal Organs.—We see, not very infrequently, in tabes certain nervous symptoms in the internal organs which are in part very characteristic. The most important and the most frequent are the so-called "gastric crises." These almost always come on suddenly and paroxysmally, and consist of an extremely severe cardialgic pain, which is accompanied by violent vomiting. The vomitus usually consists of fluid slimy masses stained with bile, and usually contains so much hydrochloric acid that we can call it an actual hypersecretion (*vide* page 459). In these attacks the patient feels very wretched, and there is

often at the same time palpitation, acceleration of the pulse, vertigo, etc. The attacks last about two or three days. In many patients they are repeated every few months. As we have said, the gastric crises may appear very early. We ourselves know cases where, in consequence of severe gastric crises, a serious gastric affection has been falsely diagnosticated, in the beginning of the disease. Attacks of diarrhœa, "intestinal crises," usually not associated with pain, have also been repeatedly observed.

We term attacks of severe dyspnœa "laryngeal crises." These depend perhaps upon a (reflex?) spasm of the glottis, and may attain a very alarming degree. They are also associated with a severe spasmodic nervous cough like an attack of whooping-cough. These laryngeal crises may come on so suddenly that the patient may sink to the floor with symptoms of the most extreme suffocation. Paralysis of the laryngeal muscles (posterior crico-arytænoids) has also been observed, and it is analogous to the other tabetic paralyses (ocular muscles, etc.). We may assume changes in the vagus-accessory nucleus, or in the vagus or recurrent itself (Oppenheim), as the anatomical cause of all these symptoms.

In a few cases "renal crises" ("*crises nephritiques*") have also been described. They consist of severe attacks of pain, like renal colic. French authors also describe "urethral crises" and "*crises clitoridiennes*," the paroxysmal appearance of voluptuous feelings with a vaginal secretion in women, in the beginning of the disease.

We may mention, finally, that we sometimes see in tabetic patients a constant and very great frequency of the pulse, 100 to 120 a minute. We also frequently see the combination of tabes with aortic insufficiency which has been mentioned by some authors. The essential connection between the two affections lies probably in the previous syphilis.

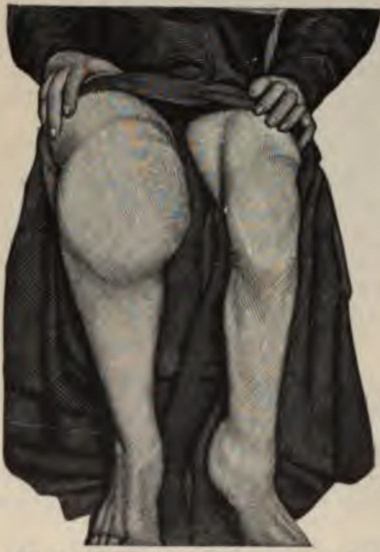

FIG. 134.—Tabetic arthropathy of the right knee and left ankle. (Personal observation.)

7. Trophic Disturbances.—Although trophic disturbances are wholly absent in many cases of tabes, they may, on the other hand, occur so frequently and in such manifold forms as actually to be classed among the most interesting although least understood symptoms of tabes. We must first mention that the general nutrition of many patients with tabes, although of course not of all, gradually suffers in a marked fashion. The patients grow thin and have a peculiar suffering, faded look. We can not wholly deny a possible connection between this symptom and the primary disease. The trophic disturbances in the different tissues are still more striking. We have already spoken of the occasional appearance of herpes zoster. In some cases a marked exfoliation of the epidermis has been seen, and also a falling out of the hair, nails, and teeth. Peculiar paræsthesiæ and hypoæsthesia of the mucous membrane of the jaws and gums often precede the loosening and falling out of the teeth. Sometimes there are small hæmorrhages, apparently spontaneous, into the skin or into the visible mucous membranes, especially into the conjunctiva, as we have seen in several cases. The so-called *mal perforant du pied* is the worst trophic disturbance of the skin; this is a round, deep ulceration, situated on the ball of the foot or on

the heel, which is very hard and slow to cure in spite of the most careful treatment.

The most interesting trophic disturbances in tabes, however, are sometimes found in the bones and joints. These are the tabetic osteopathies and arthropathies, first accurately described by Charcot (Fig. 134). In the bones there sometimes develops a peculiar brittleness, which may lead to a sudden fracture, which apparently occurs quite spontaneously or on the slightest occasion, even in bed. The painlessness of these fractures, which is due to the tabetic analgesia, is very striking; the bones get out of place without the patient's noticing it, and thus the fracture often heals with displacement and an unusual callus formation. The tabetic joint affections are found most frequently in the knee and hip, more rarely in the foot, shoulder, or even in one thumb. They often seem to begin very acutely, a very great serous effusion sometimes taking place into the cavity of the joint. Besides this effusion there gradually develop the manifold processes of a chronic arthritis deformans: on the one hand, atrophy of the articular ends of the bones; on the other, thickening of the capsule, the formation of osteophytes, etc. In connection with the arthropathy there is often permanent dislocation of the joint. Thus we often see, following a tabetic affection of the knee, a *genu recurvatum* (Fig. 135). Arthropathic muscular atrophy is often seen, and is worthy of note (atrophy of the quadriceps in disease of the knee-joint, etc.). Tabetic arthropathy may thus finally lead to monstrous changes in the joints such as can scarcely be found under other circumstances. Corresponding changes are also seen in the vertebral column. They may give rise to fracture of the vertebræ, spinal curvature, etc. The development of the so-called "*pied tabétique*" (Charcot) is also an osteopathic process; this consists chiefly of a considerable thickening and protrusion of the dorsum of the foot with a marked flattening of the sole.

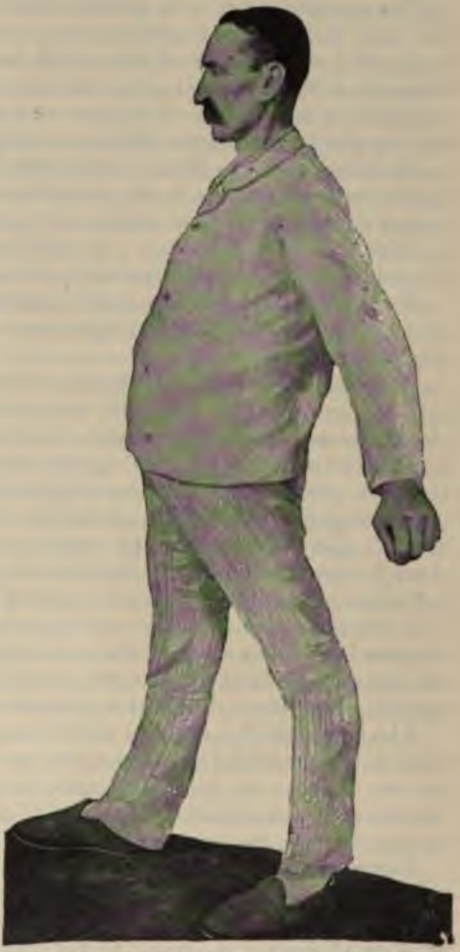

FIG. 135.—Genu recurvatum in tabes dorsalis. (Personal observation.)

We are not yet able to explain fully the occurrence of all these changes. It is hard to avoid entirely the assumption of trophic nervous disturbances. It has been claimed that degenerative changes have been found in cases of tabetic osteopathy and arthropathy in the nerves leading to the bones and joints; but it is also possible, on the other hand, that many of the so-called tabetic arthropathies may be of syphilitic origin. It is an important fact that conditions may exist in tabes, which must have a very unfavorable influence upon any existing bone or joint affection. The most important of these is analgesia. That is the reason why patients, in spite of a beginning arthropathy, do not spare the affected joint but continue to irritate it still more. We saw a patient, for example, where an affection of the knee-joint developed in a comparatively very early stage of tabes, which up to that time had not been diagnosticated at all. As the patient felt no pain whatever in his knee, he hunted most vigorously through a whole autumn, until finally an extremely severe swelling of the knee-joint and an actual subluxation of the leg ensued. It is also possible that pronounced ataxic movements may frequently aid in irritating the articular surfaces.

The muscles preserve in general their normal state of nutrition, except as they take part in some joint affection or in a general emaciation. Charcot described a case of a combination of tabes with genuine progressive muscular atrophy, in which the autopsy showed a degeneration of the anterior gray cornua in the spinal cord besides the atrophy of the posterior columns. The first report upon a unilateral atrophy of the tongue, which sometimes develops quite early in tabes, is due to the same observer. These and similar atrophies sometimes seen in other muscular territories are due to degeneration of the corresponding motor nuclei (hypoglossal nucleus, etc.); but peripheral neuritis may also lead to muscular atrophy in tabes. Joffroy described a special form of tabetic club-foot ("*pied bot tabétique*"), which seems to be due to an atrophy of the calf muscles.

Some observations of apparently spontaneous rupture of the tendons (Achilles tendon, ligamentum patellæ) show that peculiar trophic disturbances may also develop in them.

8. CEREBRAL SYMPTOMS.—Besides the frequent important disturbances on the part of certain cerebral nerves, such as the optic and oculo-motor, which have already been discussed, we must mention here the relation between tabes and progressive general paralysis (*q. v.*). On the one hand, the symptoms of tabes are often present in the course of general paralysis, so that the autopsy shows a typical degeneration of the posterior columns (Westphal); and, on the other hand, it also happens that the whole process begins with tabes, which may exist alone for years without any mental symptoms, and then only at the close do the symptoms of paralytic dementia (mental weakness, disturbances of speech, paralytic attacks, delusions of grandeur, etc.) appear. This combination of the two diseases is nothing strange, since general paralysis is also a post-syphilitic nervous disease, and is to be regarded as almost wholly analogous to tabes. Paralytic dementia has been called, not without reason, "tabes of the brain."

The complication of tabes with hemiplegia repeatedly occurs. The latter depends upon a cerebral hæmorrhage or an embolic or thrombotic softening, so that the two diseases are probably connected only so far as they are both perhaps related to previous syphilis. It seems to us worthy of note that in two such cases we saw scarcely any contracture develop in the paralyzed limbs.

Course and Prognosis.—Although most of the characteristic symptoms of tabes develop in almost all cases, still the order and the intensity of their onset vary greatly. We have already briefly described the general type of the disease which most frequently comes under observation, and many other peculiarities in its course have been mentioned from time to time.

We have stated that the initial period is usually characterized—apart from the symptoms that can be made out only objectively, such as absence of the patellar reflex and reflex immobility of the pupils—by the lancinating pains; that these may differ very much in intensity; and that the duration of this first stage may vary between a few months and ten or twenty years. The optic atrophy, the ocular paralyses, gastric crises, vesical disturbances, etc., were mentioned as rarer initial symptoms. The passage from the first stage to the second—the stage of ataxia—is sometimes very gradual, but in other cases very rapid and sudden. We have repeatedly seen such transitions. Frequently they come on clearly after some special exciting cause, as after some injury, some physical over-exertion, etc. If the previous symptoms were slight, the patient dates his disease from this point, and says that he was quite suddenly broken down by some cause, and that since then he has not been able to walk at all, or else only with difficulty. Even in such cases there is often slow improvement following the sudden change for the worse, but the gain, of course, is not permanent. In other cases, however, the ataxia develops only very slowly and never reaches a very marked degree. Experience teaches, in particular, that tabid patients with early atrophy of the optic nerves or with unusually severe attacks of pain are often without more than a trace of ataxia for a very long time—many years!

No rules of general value can be given as to the further advance of the disease, the invasion of the arms by the ataxia, or the occurrence of the rarer symptoms, such as the joint affections, etc. Almost every individual case affords its idiosyncrasies, since one group of symptoms is often especially prominent, while another is entirely absent or developed only to a slight degree. On the whole, however, we can almost always make out a gradual, even if a very slow, advance in the disease. New symptoms appear, the old ones increase, the general condition becomes worse, until finally the last stage of the disease comes on.

Recovery from tabes in the sense that all the existing symptoms (immobile pupils, absence of patellar reflex, etc.) wholly disappear, have never yet been observed with certainty, and *a priori* such a thing is hardly conceivable; but it is not so very rare for the tabetic process to be arrested for at least a year. We have become acquainted with such cases only since we have been able to recognize with certainty the initial stages of the disease. We have ourselves known for many years a number of mild cases of tabes in whom it is scarcely possible to detect any recognizable progress in the disease and who regard themselves as almost perfectly well. They go to their business and have little or no trouble from their disease. Of course we must be prepared for an ultimate change for the worse even in such cases. In more advanced tabes, treatment may at most cause some improvement, delay the course of the disease, and alleviate certain symptoms; many patients, especially under favorable external conditions, may even in this stage lead a tolerable existence for years.

Diagnosis.—There is scarcely any other disease of the spinal cord whose diagnosis can in most cases be made with so great certainty and such comparative ease. Since tabes is a combined systemic disease, it affords a definite combination of symptoms such as can occur under no other conditions. The diagnosis, therefore, is never to be made from any one single symptom, but only from the combination of all and from the whole course of the disease.

The diagnosis of initial tabes is especially important. In every case of obstinate "rheumatic" pains, or similar pains in the lower extremities, we should think of the possibility of tabes, and examine the tendon reflexes and the pupils. The combination of characteristic pains, absence of the patellar reflex on the two sides, and reflex immobility of the pupils, usually makes the diagnosis almost absolutely certain, as was said above; two of these symptoms, especially if the reflex immobility of the pupils be one, make it at least very probable. Ocular paralyses, temporary ptosis, or temporary diplopia, may be very important for the diagnosis. We should also remember that the disease may begin with optic atrophy, and that early gastric crises may simulate a gastric affection, early disturbance in micturition a vesical trouble, or swelling of the joints, a joint disease, until careful examination of the other symptoms explains the true nature of the disease. The essential point is to think at once in every such case of the possibility of tabes, and look for the distinctive objective symptoms. Among the latter we would call especial attention here, besides the symptoms generally recognized (absence of tendon reflexes, immobile pupils, slight vesical weakness), to the limited girdle-like disturbance of sensation on the trunk, since in doubtful cases this is often of decided significance in diagnosis.

In the fully developed ataxic stage of tabes the diagnosis is almost always easy, and it often can be made at the first glance. The history, the characteristic ataxic gait, the swaying with the eyes shut, the absence of the reflexes, etc., exclude every doubt. The diagnosis may be more difficult if we happen to see the patient for the first time in the final stage, when actual paralysis has set in, when a complicating hemiplegia has arisen, etc. In such cases we must lay stress on the development of the disease and find out what characteristic tabetic symptoms—pupillary symptoms, absence of patellar reflex, vestiges of ataxia, or pains—can now be discovered. With proper attention and knowledge of the case, the diagnosis can even then almost always be made correctly.

Vertebral affections are to be mentioned first of the diseases which may be confused with tabes. These also cause, under some circumstances, lancinating pains and an absence of the patellar reflex, as a result of compression of the spinal roots; but in these cases the later course of the disease is entirely different, apart from the changes in the vertebral column and the absence of other characteristic symptoms of tabes, especially the immobile pupils. The same holds true of certain deep-seated tumors in the vicinity of the spinal cord. We have already said that in rare cases a multiple sclerosis may offer symptoms similar to those of tabes. In these cases the chief stress in regard to diagnosis is to be laid on the condition of the tendon reflexes, the pupils, the disturbances of sensation, and the bladder. It is of greater practical importance that certain toxic nervous diseases may have a great similarity to tabes. Chronic alcoholic neuritis has already been spoken of in this connection—the so-called pseudo-tabes of alcoholic subjects. In these cases, however, the reflex immobility of the pupils and the disturbances of the bladder are usually absent, while atrophic paralysis may develop at the same time, which hardly ever happens in tabes. The ætiological factors (alcoholism) are also, of course, to be taken into account. In adults post-diphtheritic ataxia may be mistaken for tabes. In such cases a consideration of the ætiology, and especially the condition of the pupils and of the vesical disturbances, will usually make the correct diagnosis easy. "Diabetic tabes" (page 758) can scarcely, with proper attention, be mistaken for true tabes. The differential diagnosis between tabes and Friedreich's hereditary ataxia will be described in the next chapter.

Finally, it may be mentioned here that we have twice seen a group of nervous symptoms in men who have worked many years in tobacco factories, which resembled tabes in so many points that we might term it "nicotine tabes." The morbid symptoms, which resemble tabes, consist of painful sensations, absence of the patellar reflex, contracted pupils with reflex immobility, and uncertainty of gait; but the whole type of the disease is distinguished from tabes by a peculiar tremor, by a marked increase of the cutaneous reflexes, especially in the lower extremities, etc.

Treatment.—The tedious course of tabes demands that the physician have at hand a number of remedies and methods of treatment which he can vary according to the predominating circumstances, either to obtain a certain amount of improvement by a new way of attacking the disease, or at least constantly to kindle the patient's hope and courage anew.

The first question which is usually raised at present in regard to the treatment of tabes is in regard to the efficacy of a specific antisyphilitic treatment. In our opinion, the ability to answer this question correctly depends not merely on recognizing the fact of the connection between tabes and syphilis, but also on forming a clear conception of the nature of this connection. There can then be no doubt that in tabes there is not even a remote question of such efficacy of mercury or iodide as we see almost as a rule in true tertiary, gummatous syphilitic diseases. We very often see in tabes no benefit at all from an inunction cure or from the internal use of iodide of potassium, and in some cases the condition seems even to grow worse under such treatment. Such observations were for a long time cited as the chief evidence against the connection between the two diseases. If, however, we adopt the position which we have advanced that tabes is a post-syphilitic degenerative nervous disease, just as ataxia may sometimes be a post-diphtheritic disease, we can easily understand the inefficacy of a specific remedy, which acts only on syphilitic new formation of tissue. Nevertheless we may not wholly exclude antisyphilitic methods of treatment from the therapy of tabes; for, in the first place, it is always possible (and it seems to us to be confirmed by many trials) that an antisyphilitic treatment (inunction, iodide of potassium) may influence in a way beneficial to the patient the conditions which cause tabes to advance. It has, furthermore, been fully attested by repeated observations that, besides the tabetic changes in the cord, there may also be at the same time actual syphilitic gummatous processes, which of course may contribute their part to the symptoms of the disease. That these morbid processes require thorough specific treatment is certain, and therefore we regard it as justifiable and even desirable to make a trial of inunction in beginning tabes, and under some circumstances even in advanced tabes, especially if any anomalies in the type of the disease suggest the possibility of co-existing gummatous changes. Very experienced observers, especially Erb, have gained the impression that a careful inunction treatment sometimes visibly benefits the patient. We can not, of course, report much that is favorable from our own experience, but we often employ specific treatment, especially iodine preparations, in cases that seem suitable. Every inunction treatment prescribed in tabes must be carefully watched, that no injury be done to the patient by this potent remedy. In many advanced cases of tabes, especially where the patient's general condition is feeble, we must abstain wholly from mercurial treatment.

If the antisyphilitic treatment be not indicated, or if it has been unsuccessful, electricity and balneo-therapeutics or hydro-therapeutics deserve relatively the most confidence, although of course we must beware of exciting too sanguine hopes.

The electrical treatment consists chiefly of the passage of the ascending constant current through the spinal cord. The current must not be too strong, and the sittings should take place daily, or every other day. Erb recommends placing the medium-sized cathode in the vicinity of the sympathetic, and the large anode close to the spinous processes on the other side of the vertebral column, moving it at intervals from above downward. This procedure lasts about four or five minutes for each side. We also obtain good results symptomatically when there are severe pains, vesical weakness, etc., by peripheral galvanization. If we find painful points on the vertebral column, as is rarely the case, they should be especially treated with the stabile anode. The treatment recommended by Rumpf with the faradic brush has also been used at times with good results. This consists in brushing the skin of the back and the extremities with a strong current for five or ten minutes. Every form of electrical treatment, in order to obtain results, must be kept up for months.

Hydro-therapeutics, used rationally, have often resulted in considerable improvement in tabes, although they may cause much mischief. Hot baths, especially vapor-baths, are often followed by a rapid change for the worse—a fact which, unfortunately, we can often observe where vapor-baths have been prescribed for patients at the beginning of their disease "for rheumatism." Continuous wet packs and severe rubbings are also often accompanied by unfavorable results. Tepid half or full baths, however, of 75° to 86° at most (20°-24° R.), associated with gentle rubbing of the skin, or brief cold sponging, etc., often do good service. Wet compresses, applied around the abdomen or the legs at night, often have a favorable influence on the pains. In general, it is a good plan to send well-to-do patients in summer to a water-cure establishment which is conducted by an experienced director and well equipped, but the necessary procedures may also be undertaken at home.

Of the baths whose use is recommended in tabes, Oeynhausen has the greatest reputation. The baths in Nauheim have a composition and action very similar to those at Oeynhausen. We have also seen very good results from the baths at Kissingen. Many physicians praise mud-baths and iron-baths (Pyrmont, Driburg, Cudowa, Elster, Franzensbad, etc.), while the indifferent thermal baths (Teplitz, Wildbad, Ragatz, etc.), formerly much in favor, are at present rarely used in tabes. For the preparation of artificial carbonic-acid salt baths see pages 952, 953.

Besides the methods of treatment thus far mentioned, there are still a number of internal remedies, the use of which seems sometimes to be of advantage. The chief ones to be mentioned are nitrate of silver, first recommended by Wunderlich, one-sixth-of-a-grain pills (gramme 0.01), at first three, gradually increasing to six a day, before meals; and ergotine,* one-grain pills (gramme 0.05), three to six a day; we may also try iodide of potassium, phosphorus, arsenic, etc. All these remedies, especially the two first named, may be used for a long time, and, with interruptions, even for years.

In consequence of a somewhat too sanguine indorsement by Langenbuch, nerve-stretching (usually the operative stretching of both sciatics) was for some time practiced on many tabid patients, but since experience has taught us that nerve-stretching, in spite of some apparent successes, scarcely ever exerts a permanent favorable action, and is also not wholly without danger, the operation has been almost entirely given up in tabes. It may still be tried in those cases where we have unusually severe attacks of pain in the region of definite nerves. In such cases we should first try bloodless stretching (page 820). The "suspension treatment" of tabes has also had only a brief vogue. This consisted in hanging up the patient for a short time by an apparatus fastened under the chin [and arms], thus stretching the spinal cord and the nerve-roots by the weight of the body. The successes at first reported are of very doubtful significance, while there were certainly some very unfortunate consequences. We ourselves cannot advise the use of this method. The treatment of tabes by firmly fitting corsets is also much praised and recommended. If we listen to the patients who have just left the sanitarium of some eminent orthopædic surgeon, they know no end to their praise. If we see them again in a year and a day, we find them ataxic as before, and the corsets—in some corner! Temporary symptomatic benefit should not be denied, but of course the corset treatment can not affect the tabetic process itself.

Symptomatically, the same remedies are to be considered as were mentioned in the treatment of chronic myelitis. We try to alleviate the pains by narcotic embrocations and bandaging the legs. Of internal remedies, sodic salicylate and antipyrine, and also antifebrine, phenacetine, or a mixture of these remedies, sometimes undoubtedly alleviate and shorten the pains. [Chloride of aluminium, in doses of two to four grains (gramme 0.1–0.25) three times a day, occasionally relieves the pain.—K.] Some observers praise the action of the so-called *points de feu* (circumscribed burning of the skin with the thermo-cautery) along the spine at the points of exit of the affected nerve-roots. In bad cases morphine is indispensable. We try to remove the constipation by prescriptions as to diet, or by mild cathartics, such as the bitter waters, tamarinds, and rhubarb, by enemata, or by electrical treatment and massage of the abdomen. Galvanic treatment often acts very favorably on the bladder symptoms. For these symptoms Charcot praises the action of ergot, and other physicians recommend strychnine. Morphine is the best remedy in the gastric crises. We may also try warm poultices, washing out the stomach, belladonna, chloral, cocaine, etc. Severe laryngeal crises also require morphine injections as a rule. Optic atrophy unfortunately defies any treatment. We usually try strychnine injections to encourage the patient.

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* There is only an apparent contradiction in the fact that, in spite of the occurrence of an "ergotine tabes" (*vide supra*), ergotine is also used as a remedy against tabes. It may very well be that the same remedy, which in large doses causes certain systems of fibers to atrophy, may in smaller doses have some favorable (stimulating) action on the same system of fibers; but we must always be cautious in the use of ergotine.

One method for the symptomatic treatment of tabes deserves special mention, and that is the treatment of ataxia by methodical exercises (G. Frenkel). If ataxic patients are systematically admonished to execute all the movements of the several joints slowly and as regularly as possible several times each day for a definite time, and also to perform certain other movements (touching a definite point with the fingers or toes, walking a chalk mark, walking between the rungs of a ladder, seizing swinging balls, putting stoppers into definite holes, etc.), we may often recognize that the practice actually has an influence upon the certainty of the movements and leads to an improvement in the ataxia. Certain sanitaria carry out this method of treatment by the use of complicated apparatus. The benefit, however, should not be overvalued.

It is also of the greatest importance that the physician should regulate and supervise the patient's mode of life in its general hygienic and dietetic aspect. We must warn him especially against any physical or mental over-exertion, prescribe a prudent but strengthening diet, withdrawing all large amounts of alcoholic drinks, and enjoin good air, a country residence in summer, or a medium mountain climate, and in winter at times southern resorts. How effective this general dietetic treatment of tabes is, may be seen in hospitals, where tabid patients of the lower classes are sometimes much benefited by simple rest and good care. The earlier we get the patient under treatment, the more persevering and careful should we be in our treatment, because then we can still hope for a certain success. In old and far-advanced cases we must confine ourselves to a purely symptomatic treatment.

CHAPTER VIII

HEREDITARY (JUVENILE) ATAXIA (FRIEDREICH'S DISEASE)

Ætiology and Pathological Anatomy.—In 1861 Friedreich first described a peculiar disease observed by him in several members of the same family. He at first regarded the disease as a special form of tabes on account of the marked ataxia attending it, but later investigations have shown that this "Friedreich's disease" has nothing in common with true tabes except a certain similarity of symptoms (especially ataxia), which is due to the fact that the anatomical changes in the cord agree in part in the two affections. While tabes represents an acquired degeneration of different neurone systems, Friedreich's disease is undoubtedly due to a congenital morbid predisposition of certain neurone systems, as a consequence of which these neurone systems have not developed their full and permanent functions and therefore atrophy slowly and prematurely. This congenital origin of the disease is most evident from the fact that the disease is not infrequently seen in several (two to five) brothers and sisters, and in some cases in members of two or more generations of the same family. Of course sporadic cases of Friedreich's disease are seen with comparative frequency, but in such cases the youthful age at which the first symptoms of the disease are noticed indicates the congenital abnormal tendency. The first symptoms sometimes appear at the age of seven to ten years, but frequently not until puberty, and in some cases even somewhat later (eighteen to twenty years). It is usually hard to determine accurately the period of onset, because the disease begins very gradually, and therefore its onset may certainly remain unnoticed for a long time. The disease is about equally common in the two sexes, perhaps a little commoner in the male. The first symptoms sometimes seem to follow some acute disease (scarlet fever, measles, influenza), injury, etc., but these are exciting causes which make the existing morbid tendency manifest.

Clinical History.—The first symptom invariably to be noticed, which is by far the most important later, is ataxia. It shows itself at first as a slowly increasing uncertainty of gait; almost at the same time or only a little later the ataxia is noticed in the upper extremities. When the disease is fully developed the gait is unusually characteristic. In regard to the ataxic movements of the legs the gait resembles that of tabes, but it usually differs from it in the much greater swaying of the trunk. In this respect the gait is more like that of the so-called cerebellar ataxia. The fact that in Friedreich's disease the muscles of the trunk and pelvis are also affected by the ataxic disturbance, and even to a greater degree, is the reason why the patient usually manifests more of the so-called static ataxia than is the case in tabes—that is, that he continually sways very much when standing quietly (*vide* Fig. 136). The patient therefore always stands and walks with the legs wide apart to give the body a better support. If we make him stand still with the legs as close together as possible, we notice the constant muscular contractions necessary to maintain the equilibrium. With the removal of the visual control by closing the eyes, the uncertainty and swaying of the body increases. The ataxia is also very well marked in the legs, as is most readily shown by the well-known "knee-heel test," and also in the arms, as tested by bringing the finger-tips together, by reaching for an object held before one, writing, etc. The disturbance of speech, which is often noticed in the later stages, is due to an ataxia of the muscles requisite for speech, and consists of an indistinct articulation and a slow, scanning, badly modulated manner of speaking. Nystagmus of the eyes, especially on looking far to one side, sometimes occurs as a further ataxic symptom, just as in multiple sclerosis, but it is by no means invariable, or at least it may not appear until late in the disease.

Besides the ataxia, which finally becomes very pronounced, for a long period we can usually detect only one other important symptom, which is probably always of very early occurrence—the complete absence of the patellar reflex. All other nervous functions may remain wholly unaffected for a long time. The motor strength of the muscles is very well retained. We know a patient who, in spite of the most marked ataxia, has traveled several miles a day by the aid of a cane. The muscles are usually not remarkably well developed, but they show no striking atrophy, apart from rare complications. Many patients show a peculiar choreic muscular unrest. The sensibility of the skin and deeper parts (joints, muscular sense) may for a long time remain perfectly normal, a condition which has been justly brought forward, especially by Friedreich, as an argument against the dependence of ataxia upon sensory disturbances. Of course this preservation of sensibility must not be too much emphasized, since many other observers and we ourselves have repeatedly found slight disturbances of sensibility (especially on the feet, toes, and lower legs) on careful testing: for example, a slight diminution of the tactile sense, the pressure sense, sometimes the muscular sense, etc. The sensibility to pain seems almost always to remain unaffected. Paræsthesiæ and spontaneous pains are also entirely absent. The cutaneous reflexes show no material disturbance and the pupil reflexes are always preserved. Disturbances of the bladder are also absent. In some cases, however, there may be atrophy of the optic nerves, just as in tabes. We may also note that the toes, especially the great toes, have a very striking tendency to assume a position of extreme dorsal extension, and we sometimes see a tendency for the foot to assume the equinus position. The vertebral column often shows an evident scoliosis.

The course of the disease is very chronic and it may last for years and years. The ataxia finally becomes so great that the patient is bedridden. In such cases there is finally actual paralysis, greater disturbance of sensation, disturbances of intelligence, etc. Death often ensues from some intercurrent disease.

Pathological Anatomy.—Hereditary ataxia was the first form of disease in which a combined systemic lesion of the spinal cord was discovered by careful anatomical investigation by Kahler and Pick. Later observers (F. Schultze, Rütimeyer, and others) have confirmed these discoveries. We find the posterior roots and the posterior columns most diseased, and chiefly the zone of the entering root-fibres. Lissauer's areas are usually exempt, in contrast to the degeneration of tabes. The cerebellar lateral tract (including probably the cells of Clarke's columns) is also regularly affected, and also Gowers' tract (see the chapter on secondary degenerations in the cord) and the neighborhood of the pyramidal lateral tract, although in lesser degree. The histological process, as in all systemic diseases, consists of a degenerative atrophy of the nervous elements and a considerable secondary increase of the neuroglia.

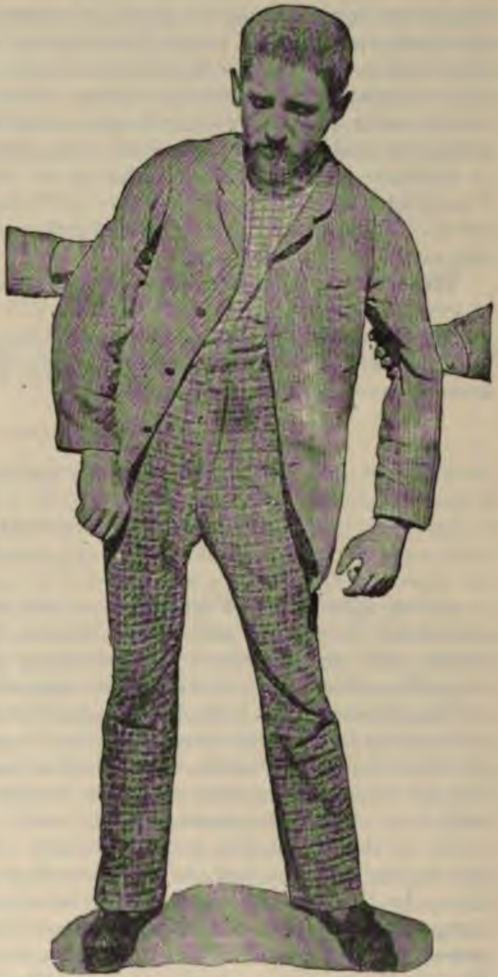

FIG. 136.—Attitude of a patient with hereditary ataxia. (Personal observation.)

It would be very interesting to make a careful comparison between the anatomical lesions and the symptoms in Friedreich's disease, on the one hand, and in tabes on the other; but we are still greatly in want of a sufficient number of careful observations. The absence of marked sensory disturbances and of lancinating pains in hereditary ataxia is striking. The absence of pain is explained perhaps by the integrity of the peripheral nerves, or perhaps by the different nature of the disease: in tabes we have the action of irritating toxines, in hereditary ataxia simple endogenous atrophy.

Diagnosis.—The diagnosis of Friedreich's disease is easy, if we are acquainted with the disease, and it is usually perfectly definite. The marked ataxia, especially the tottering, uncertain gait, besides the youth of the patient, and at times the hereditary conditions, as a rule soon render a correct decision possible. The absence of immobility of the pupils, of marked sensory disturbances, of symptoms of sensory irritation, of vesical disturbances, etc., distinguish the disease from ordinary tabes. It is easier to mistake it for multiple sclerosis, since this also comes on in youth; but Friedreich's disease often begins in childhood, which is only exceptionally the case with multiple sclerosis. The examination of the patellar reflex is most distinctive; this is entirely absent in hereditary ataxia and increased in multiple sclerosis. The condition of the patellar reflex also distinguishes Friedreich's disease from hereditary cerebellar ataxia, soon to be described (*vide infra*). The distinction from post-diphtheritic and similar forms of ataxia should also give rise to no difficulty.

Treatment.—Although there are some variations in the course of the disease, it seems in general always to advance and to be incurable. We can therefore merely try to obtain temporary improvement and an arrest of the disease by intelligent care of the patient and by all the methods employed in chronic nervous diseases (see the previous chapter).

APPENDIX

HEREDITARY CEREBELLAR ATAXIA

(Hérédo-ataxie Cérébelleuse)

As an appendix to Friedreich's ataxia we must here mention still another remarkable hereditary and family disease, the chief symptom of which is also ataxia, while the most essential anatomical change consists of an atrophy of the cerebellum (Nonne, P. Marie, Londe, and others).

The disease occurs in brothers and sisters and members of different generations of the same family, but there are probably sporadic cases besides. The two sexes are about equally affected; the male sex seems to predominate somewhat. The first symptoms of the disease usually appear between the twentieth and the thirtieth year, sometimes earlier, rarely later. They consist of the slow onset of ataxia of the extremities and particularly of a staggering, reeling gait. Standing is also insecure, but on the whole it is only slightly altered by closing the eyes. In the arms the ataxia may become so great as to render all the finer occupations (writing, etc.) almost impossible. The ataxia of the facial muscles causes mimetic movements of expression which are often extremely peculiar and bizarre; the speech is usually considerably disturbed. The muscular strength is not diminished, although the patient often complains of feeling fatigued. The sensibility is perfectly retained and, in particular, we find the muscular sense wholly unaffected. The cutaneous reflexes are normal or somewhat diminished, but the tendon reflexes are almost invariably increased, although marked spastic symptoms are absent. The innervation of the bladder undergoes no change. The pupils usually retain their normal reaction, but atrophy of the optic nerves with the corresponding disturbance of vision has been repeatedly observed. Pronounced mental disturbances, such as weakness of memory, abnormal irritability, etc., may also develop.

The disease has a very tedious course and usually lasts many years. The prognosis is absolutely unfavorable. Death ensues from some intercurrent disease or as a result of the ultimate general weakness. Anatomical examination, as has been said, shows as the most striking change an atrophy of the cerebellum, which is diminished about one half in size. The spinal cord is sometimes poorly developed on the whole, but it shows no columnar degeneration.

The disease is distinguished very essentially from Friedreich's hereditary ataxia, which otherwise is very similar to it, by this anatomical condition, which is, however, still in urgent need of thorough study. Clinically the two diseases are to be distinguished particularly by the condition of the tendon reflexes and the fact that cerebellar ataxia usually comes on somewhat later in life. The differential diagnosis between cerebellar ataxia and multiple sclerosis is often very difficult. The generic (family) occurrence is the chief distinction.

The treatment can be merely symptomatic, and it has little prospect of success.

CHAPTER IX

THE PRIMARY DEGENERATIONS OF THE MOTOR TRACT, INCLUDING THE MUSCLES

1. Preliminary Remarks

In the two preceding chapters we have become acquainted with two diseases whose anatomical basis is a primary degeneration of a number of systems of fibers whose conduction was chiefly *centripetal.* In tabes we found the cause of the degeneration to be exogenous, acquired noxious influences, coming from without and probably of chemical nature; in hereditary ataxia, on the contrary, the cause was endogenous anomalies—that is, anomalies in the embryonic predisposition, and therefore congenital, which caused a premature atrophy of nerve-fibers which had from birth an abnormally weak power of resistance. In this chapter we shall become acquainted with a number of diseases, which are due chiefly to primary degeneration of the great *centrifugal* motor conducting tract, especially the pyramidal tract. We will see that, just as with hereditary ataxia, the basis of the disease is chiefly an endogenous, congenital weakness of this portion of the nervous system, so that it is therefore very often a family, generic disease; although in some cases exogenous noxious influences are probably also to be considered.

In order to obtain a preliminary review of the whole group of diseases here under consideration, we may picture to ourselves, as in the accompanying diagram ((Fig. 137), the course of the long conducting tract leading from the central convolutions to the muscles, as has been fully described on page 829. In the first place we will consider a form of disease in which ultimately the whole cortico-muscular conducting tract, from the cortical cell, *C*, up to and including the muscle, *M*, is found degenerated. This is *amyotrophic lateral sclerosis.* We will next describe a form of disease in which only the second portion (the so-called second neurone) of the whole tract—namely, the portion from the ganglion-cell in the anterior horn of the cord (*V*) up to and including the muscle—is affected. This is *spinal muscular atrophy,* and the completely analogous *progressive bulbar paralysis.* In the third place we shall see that the first main portion of the tract, *PyS,* may be alone affected up to the cell of the anterior horn, but without involving it. We then have the true *primary pyramidal* *lateral sclerosis*, with the clinical picture of pure spastic spinal paralysis. In the fourth place the disease may involve only the peripheral motor nerve, *m*, with its corresponding muscles (*neurotic muscular atrophy*), and finally we may also place here by far the commonest form of disease, the *progressive muscular dystrophy*—that is, that disease in which only the terminal apparatus of the whole tract, the muscle itself, undergoes a progressive atrophy.

The forms of disease just mentioned are probably, so far as they are endogenous diseases, all closely allied, but it is still necessary for a time to keep them separate, at least so far as they show very definite marks of distinction, both clinically and anatomically. Only with a more advanced knowledge of the special nature of all these morbid conditions can we make a strict ætiological division of them. At present we must remember that we can not consider the different clinical and anatomical forms as different diseases, strictly separate from one another. The more our knowledge increases, the more prominent seem to be transitions and connecting links between the different forms.

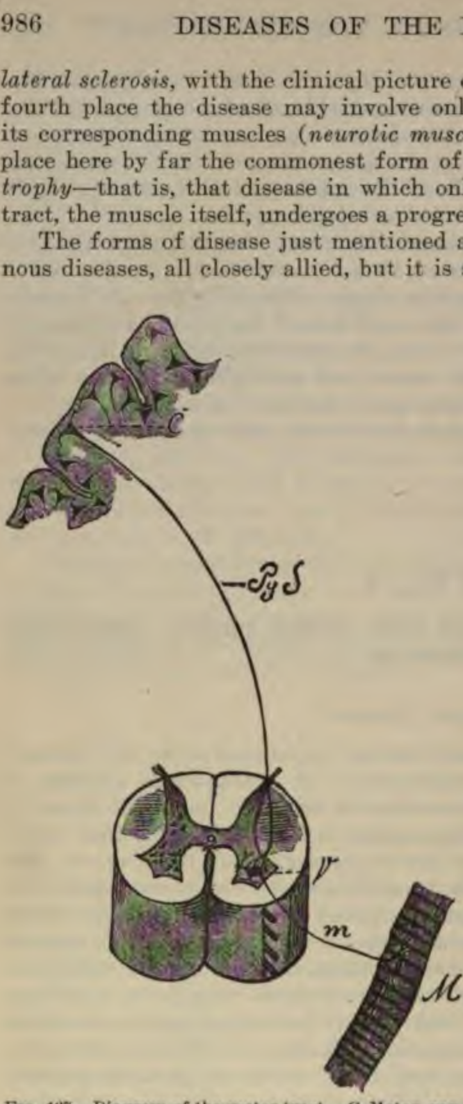

FIG. 137.—Diagram of the motor tract. *C.* Motor ganglion-cell in the cortex. *PyS.* Pyramidal lateral tract. *V.* Ganglion-cell of the anterior horn. *m.* Motor nerve. *M.* Muscular fiber.

2. AMYOTROPHIC LATERAL SCLEROSIS

Ætiology and Pathological Anatomy.—For the first accurate knowledge of amyotrophic lateral sclerosis we must thank Charcot, who published in 1869, in company with Joffroy, his first observations upon such cases, and who in 1874 was able to give quite a complete description of the disease; but an exact knowledge of amyotrophic lateral sclerosis was first made possible by Flechsig's investigations upon the course of the conducting tracts in the spinal cord. From these we can now say that the disease is manifested anatomically essentially as a primary systemic degeneration of the whole cortico-muscular conducting tract, and not only of the spinal portion thereof belonging to the extremities, but also of the bulbar portion belonging to the muscles of the face, tongue, soft palate, etc. These two parallel portions must be regarded as completely equivalent parts of one great motor system—a point to which we shall frequently return later. The motor-nerve nuclei in the medulla and pons (for the hypoglossus, facial, motor, trigeminus, etc.), and also the nuclei for the oculomotor nerves, are to be regarded as precisely analogous to the ganglion-cell groups in the anterior horns of the cord for the muscles of the extremities.

In amyotrophic lateral sclerosis a large part of this motor conducting tract undergoes degeneration, and sometimes, as it seems, throughout almost its whole extent. If we examine the nervous system in such a case we find in the spinal cord that the large ganglion-cells of the anterior horns in the cervical and lumbar cord, or in the cervical cord only, are atrophied and in part wholly absorbed (Fig. 138). We find the pyramidal tracts greatly degenerated in both lateral columns, and, of course, in one or both anterior columns when there is an anterior pyramidal tract. In the anterior horns also the number of medullated fibers, part of which are certainly processes of the pyramidal lateral tract (*PyS*) * fibers, is diminished. Farther upward, as was first discovered by Charcot and Marie, we can follow the degeneration of the pyramidal tract, not in all, but certainly in many cases, through the pyramids of the medulla, the pons, the crura, and the internal capsule to the central convolutions, in whose ganglion-cells we can also find atrophy. The bulbar motor nuclei, especially the hypoglossus nucleus and the vagus-accessory nucleus, more rarely the facial nucleus, etc., are also atrophied as a rule, just like the anterior horns of the cord. From all these bulbar and spinal ganglion-cells a further (secondary?) degeneration also extends toward the periphery into the corresponding nerve-roots (hypoglossus, etc., spinal anterior roots) and nerve-trunks. It is difficult to make out atrophied fibers in the peripheral nerves, and up to the present time they have not always been looked for with sufficient care, but it can scarcely be doubted that the affected motor fibers, which are processes of the atrophied ganglion-cells, are likewise to be found in a condition of degeneration. Finally, the muscles show a considerable atrophy, as is plainly manifest even during the life-time of the patient. Their volume is much diminished; many muscles (for details *vide infra*) finally almost wholly disappear, so that in their place there is little left but connective tissue and fat. In the other muscles we find, besides a number of normal fibers still preserved, many very small fibers, showing various degrees of atrophy, with their transverse striation usually preserved, but more rarely indistinct. The nuclei of the sarcolemma are much increased, and the interstitial fat tissue is often, but not always, abundantly developed.

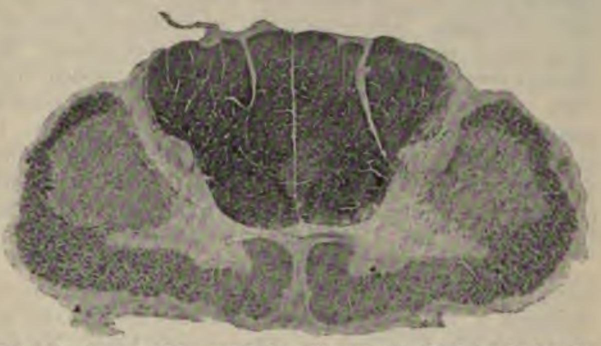

FIG. 138.—Section of the cervical cord in amyotrophic lateral sclerosis. Degeneration of the pyramidal tracts, in part of the other anterolateral tracts, pronounced atrophy of the anterior horns. (Personal observation.)

Although we may undoubtedly designate amyotrophic lateral sclerosis as, in the main, a primary degeneration of the pyramidal tract, yet in many cases the disease is not limited absolutely to this one system. It has long been noticed that the diseased area in the lateral columns extended farther forward and inward than the actual pyramidal lateral tract, *PyS*, and that, accordingly, bundles of fibers in the anterior portion of the lateral columns and in the so-called lateral limiting layer were involved in the disease. Indeed, it often seems as if the whole region of the anterior and lateral columns were poorer in fibers than the area of the posterior columns. Only toward the lateral cerebellar tract, *KlS*, is the degeneration almost always very sharply limited—that is, the lateral cerebellar tract remains as a rule perfectly normal, but disease has repeatedly been found in Goll's columns. All these degenerations, last mentioned, have no known clinical significance. They show only how other bundles of fibers may sometimes suffer under the same noxious influence as well as the pyramidal tract.

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* Abbreviations in frequent use in what follows: PyS = pyramidal lateral tract (Pyramiden-Seitenstrangbahn); PyV = anterior pyramidal tract (Pyramiden-Vorderstrangbahn); KlS = lateral cerebral tract (Kleinhirn-Seitenstrangbahn.)

Our knowledge in regard to the questions as to where the degeneration begins, in what way it progresses, and what is primary and what secondary atrophy, is most uncertain. The anatomical examination gives only the result of the destruction, but it shows very few details of its course. Much may be inferred from clinical observation, but much work is still needed to attain perfectly clear knowledge. Perhaps different possibilities should be considered, which would explain many variations in the clinical course. At any rate, the different portions of the system may be affected in varying sequence, and the disease may also extend with varying rapidity, but the destruction of the nerve-fibers is always the primary process; the increase of the interstitial connective tissue and the slight changes in the vessels are a secondary, accidental process.

We know as yet hardly anything definite as to the causes of amyotrophic lateral sclerosis. For one class of cases we believe we may assume a congenital weakness of the motor system. The disease usually begins at the period between thirty-five and fifty, but not infrequently later. No special external causes of the disease have thus far been found. The male sex seems to be affected somewhat more frequently than the female.

Clinical History.—In all typical cases the clinical symptoms give a perfectly characteristic type of disease, limited strictly to the motor sphere, corresponding to the perfectly systemic anatomical lesions just described.

The first signs of the disease almost always begin in the arm. The patient notices a difficulty in working, and becomes easily tired. The weakness of the arm gradually increases, and finally, usually some months later, involves the other arm. A wasting of certain muscles, which gradually increases and becomes more extensive, is now often noticed by the patient. About six months or a year later, symptoms appear in the lower extremities. The gait becomes stiff and uncertain, the patient gets tired more easily, and quite a marked tremor of the legs often comes on, apparently spontaneously.

If we now examine the patient carefully, the type of the disease is usually perfectly plain. We notice, first in the upper extremities, a very pronounced and more or less extensive muscular atrophy. This is usually most marked where it begins—namely, in the thenar and hypothenar eminences. The interossei are also plainly atrophied, and the muscles on the extensor side of the forearms, but the flexors of the hand and of the terminal phalanges of the fingers remain intact longer. In the upper arm the triceps and the deltoid are usually the most atrophied, and later, and to a less degree, the biceps and the muscles of the shoulder. We find a functional disturbance of the muscles—a paresis—corresponding to the degree of atrophy. The functional capacity depends upon how much muscle is left, and only with a complete disappearance of the muscle is there a complete loss of the corresponding motion, but a marked paresis can sometimes be noticed in muscles which are not yet much atrophied. Fibrillary muscular contractions are almost always to be detected in the affected muscles on careful observation. The electrical excitability of the muscular fibers still preserved is normal. The strength of contraction of the muscle irritated by the faradic current is therefore proportional to the amount of muscular substance still present. In the much atrophied muscles the effects of irritation are finally very slight, and then we can always detect a distinct reaction of degeneration in the muscular fibers that are still left, especially in the ball of the thumb. We can scarcely ever make out with certainty a loss of excitability in the nerve-trunk, probably because here a greater number of normal fibers are always present.

The examination of the tendon reflexes is very important. They are invariably much increased, even from the early stages of the disease. We obtain vigorous reflex contractions from a gentle blow on the tendons of the biceps and the triceps, and on the lower ends of the bones of the forearm. This increase of the tendon reflexes is extremely important in diagnosis, because it never occurs in this way in ordinary "progressive muscular atrophy"—that is, in that disease in which the degeneration extends merely from the muscles to the motor ganglion-cells in the anterior cornua, while the lateral motor tracts remain free (*vide infra*). In the later stages of the disease marked contractures in the arms and hands sometimes, but not always, develop. The sensibility of the skin and deeper parts, however, remains absolutely normal.

The first morbid symptoms usually develop in the lower extremities some months later than in the arms. The pure spastic symptoms are here remarkably prominent, while the muscular atrophy is late in its development, and is but slight. The legs become stiff, and oppose considerable muscular resistance to attempts at passive motion, but the crude strength of the muscles is decidedly below the normal. There is an obvious paresis, although there is, as it seems, hardly ever a complete paralysis of the legs; and the disturbance of motion, at any rate, is considerably increased by the spastic symptoms (*vide infra*, spastic spinal paralysis). The spastic symptoms are connected with the hypertonicity of the muscles (the increased muscle tonus) and the increased tendon reflexes. These symptoms depend mainly upon the great increase of the tendon reflexes. The patellar reflex is very vigorous, and we often find a marked and persistent ankle clonus. The patient can still walk quite a distance, but the gait is, of course, difficult and laborious. The patient walks with short, slow, dragging steps—the spastic-paretic gait. Only in a few cases does a marked atrophy of the muscles appear in the early stages in the legs as well as in the arms. The sensibility of the skin and deeper parts is also perfectly preserved in the legs in amyotrophic lateral sclerosis. The cutaneous reflexes from the soles of the feet show no striking changes. [There may be dorsal extension of the great toe on testing the plantar reflex—Babinski's reflex.—K.] Disturbances in micturition are also entirely absent. The bowels may be somewhat costive, but they are otherwise normal.

After the condition has lasted for some time (a year or two) in this form—muscular atrophy and increased tendon reflexes in the upper extremities and spastic paresis in the lower—and has slowly grown worse, bulbar symptoms come on in the third and last stage of the disease. The speech gradually becomes more indistinct, and there is difficulty in swallowing. If we examine closely we find the lips atrophied, so that puckering the mouth, whistling, etc., are difficult. We also notice a decided atrophy of the tongue. Its surface is uneven, and we notice more or less marked fibrillary twitchings of single muscular bundles. The sensibility is also normal here. We sometimes find a vigorous masseter reflex (jaw-jerk) on striking the lower jaw, analogous to the increased tendon reflexes in the extremities. The other muscles of the face, the muscles of mastication and the ocular muscles, usually remain unaffected. The patient's intelligence is unchanged. The ready onset of marked mimetic emotional movements is sometimes striking. Many patients with amyotrophic lateral sclerosis break out into spasmodic laughter or weeping and wailing upon the slightest provocation ("impulsive laughing and crying"). This remarkable symptom, which also occurs in multiple sclerosis (page 957), is probably due to the disappearance of certain fibers which inhibit affective movements. The face is sometimes peculiarly distorted and broadened by a persistent tonic contraction of the facial muscles (see Fig. 156, page 1010). In one case under our observation there were frequent attacks of severe spasm of the glottis with danger of suffocation.

If the patient have trouble in taking food, from difficulty in swallowing, the state of the general nutrition soon becomes worse. Respiratory disturbances are usually the final immediate cause of death, if an intercurrent disease, such as inhalation pneumonia, etc., does not previously put an end to the patient's melancholy condition.

Variations from this typical picture of the disease sometimes occur, but only as the sequence may vary in which the different symptoms come on or the different muscular regions are affected. If the anatomical lesion begins exclusively in the central motor neurones, there are at first purely spastic symptoms, either spastic rigidity of the lower extremities only or rigidity of the trunk and arms as well, and a distorted facial expression; and only later, when the second motor neurones are affected, is added atrophy of the muscles, at first usually either in the forearms and hands or in the bulbar region (tongue, lips, etc.). In other cases however, the type of progressive muscular atrophy is from the first more prominent, and the spastic symptoms, if marked anywhere, appear only in the legs. In these cases the disease of the peripheral motor neurones is at first evidently greater than that of the central motor neurones; but even here the increased tendon reflexes point from the outset to an implication of the central motor neurones. Increased tendon reflexes, however, can of course occur only in those muscles whose ganglion-cells in the anterior horns are not yet completely destroyed. The bulbar symptoms are wholly analogous to the other muscular atrophies; they are due to the degeneration of the nerve-nuclei in the medulla. We shall become more fully acquainted with them later (*vide infra*, progressive bulbar paralysis).

The **diagnosis** of the disease is rarely difficult. Its usually typical course, the muscular atrophy co-existing with increased tendon reflexes, the rigidity in other muscles, especially in the legs, the complete absence of sensory or vesical disturbances, and the final appearance of bulbar symptoms, are chiefly to be considered in diagnosis. Mistakes may arise from the fact that tumors or myelitis may for a long time have a similar localization, as in the gray matter of the cervical cord, and therefore provoke analogous symptoms. It is probable that in the past there was often confusion with syringomyelia (*q. v.*), but such mistakes may easily be avoided if we remember the sensory disturbances in the arms which are so very characteristic of this disease. The distinction between amyotrophic lateral sclerosis and the other primary diseases of the motor system will be self-evident from the description of these diseases which follows.

The **prognosis** of amyotrophic lateral sclerosis must be regarded as absolutely unfavorable. The disease advances slowly but unceasingly, and usually leads to death in a few years. Only in a few cases, occurring in early youth (Seeligmüller), does the disease seem to come to a standstill.

We can therefore expect but slight results from **treatment.** At most we can perhaps check the advance of the disease by an electrical or gymnastic treatment, kept up with very great patience and perseverance.

3. SPINAL PROGRESSIVE MUSCULAR ATROPHY

Preliminary Remarks and Pathological Anatomy.—Few diseases of the spinal cord have been the subject of such different conceptions and interpretations as progressive muscular atrophy. The reason for this lies chiefly in the fact that its main symptom, the progressive atrophy of the voluntary muscles, may be found in many totally distinct diseases, and hence this has given rise to constant confusion and mistakes. If, at the present time, we read the older and even a part of the newer literature on this subject, we find everywhere a mixture of different cases, not at all of the same disease, and only the latest accurate clinical and anatomical methods of investigation have enabled us to bring at least some order out of this chaos.

Except for a few earlier observations, Duchenne and Aran, in 1849 and 1850, gave the first good description of progressive muscular atrophy. The French observers, therefore, at present speak of the disease, in distinction from other similar affections, as "*atrophie musculaire progressive, type Duchenne-Aran.*" A short time after, in 1855, Cruveilhier, on the ground of a positive lesion on autopsy, first pronounced the opinion that a disease of the gray matter in the spinal cord was to be regarded as the special anatomical cause of the disease. Since then a tedious dispute has been carried on, and even in part kept up to the present time, as to whether, in fact, the disease has its seat in the spinal cord, or in the muscles themselves—a dispute which was necessarily without results for a long time; the more because the actual pathological data were very scanty, and because the results of examinations often contradicted one another, owing to the confusion between different morbid processes which did not belong together at all.

At present we know that the great majority of progressive muscular atrophies certainly have their starting-point in the muscles themselves (*vide infra*, progressive muscular dystrophy), but that there is also a rare but definitely established spinal form of progressive muscular atrophy. This is closely allied to amyotrophic lateral sclerosis, which we have just described, but although in amyotrophic lateral sclerosis the whole pyramidal motor tract may be degenerated, and the lateral pyramidal tracts in the cord are in particular always affected, in "spinal progressive muscular atrophy" the degeneration, as we have said, is confined to the peripheral motor neurone—that is, to that portion of the motor conducting tract which extends from the ganglion-cells of the anterior cornua to the muscular fibers themselves. The second central motor neurone—that is, the lateral pyramidal tract—remains perfectly normal. It is very improbable that this difference in the extent of the anatomical localization causes a fundamental distinction between the two diseases mentioned. The clinical distinctions due to the different anatomical localization are, however, of sufficiently marked prominence to justify, at least provisionally, a special limitation of amyotrophic lateral sclerosis on the one side and spinal progressive muscular atrophy on the other, as two different clinical forms of the same morbid process.

The precise anatomical lesion in progressive (spinal) muscular atrophy is as follows:

In the spinal cord, particularly in the cervical cord, we find the anterior gray cornua very small; the ganglion-cells have wholly or largely disappeared, and those remaining are atrophied; and the neuroglia is changed to a fine fibrous tissue, sometimes studded with spider-cells; but the lateral columns, especially the pyramidal tracts—that is, the portion of the motor conducting tract central to the ganglion-cells of the anterior cornua—are perfectly normal; the anterior roots and the affected motor fibers in the peripheral nerves, however, are atrophied, although in the nerve-trunks the discovery of degenerated fibers mixed with many other normal fibers is not perfectly easy. In the muscles the atrophy is, of course, still more noticeable on a post-mortem examination than on examination during life. The muscles most affected are reduced to small, pale, and flabby bundles, in which fat and connective tissue outweigh the proper muscular tissue. On histological examination, we find, besides some muscular fibers which are still of normal diameter or even hypertrophied, all degrees of diminution and atrophy in the other fibers, so that finally all that seems to be left of many fibers is merely the empty sheaths of sarcolemma; but most of the diminished fibers retain their transverse striation to the last, and only occasionally do we see signs of granular (fatty?) and waxy degeneration of the muscular fibers, a splitting longitudinally and transversely, etc. The interstitial connective tissue is always hyperplastic, the muscular nuclei are considerably increased in number, and we often find an abundant deposit of fat between the fibers which are still preserved.

So far the actual lesion. In comprehending it, the only difficulties arise from the questions as to the mode of development, and as to the reciprocal dependence of the different disturbances. Is the atrophy of the anterior cornua to be regarded as primary, and the atrophy of the nerves and muscles as a secondary descending degeneration; or does the process begin in the muscles or nerve-endings, and extend upward from them to the spinal cord; or, finally, do we have to do with an approximately simultaneous degeneration of the whole motor portion affected? These are questions to which at present no definite answer can be given. Many reasons seem to us to favor the belief that the degenerative process begins in the last terminal branches of the motor nerves, and from these ascends gradually to the spinal cord. This, however, is not yet proved, and possibly the starting-point of the disease and its further progress may differ in different cases.

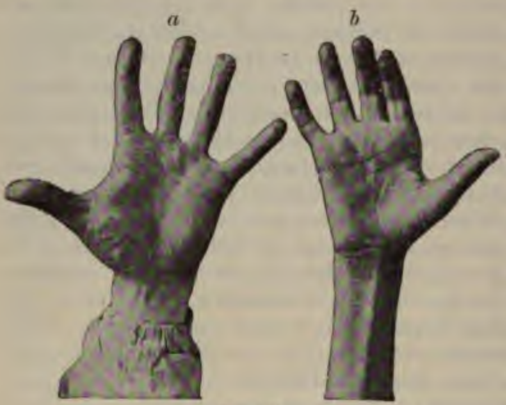

FIG. 139.

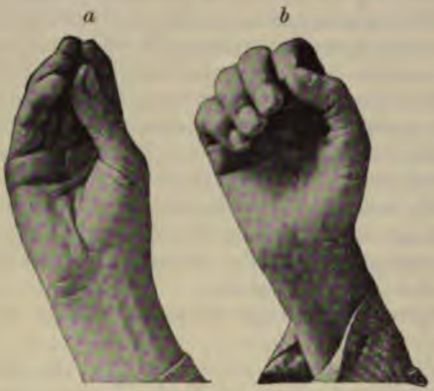

FIG. 140.

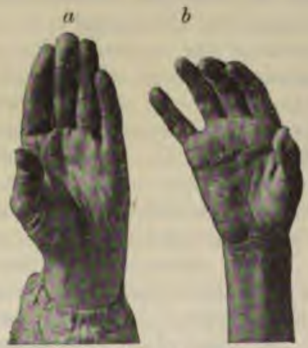

FIG. 141.

FIGS. 139-141.—Atrophy of the small muscles of the hand, especially of the thenar and hypothenar muscles, in spinal progressive muscular atrophy. *a*. Hand of a healthy man. *b*. Atrophied hand. (Erlangen Medical Clinique.)

Ætiology and Clinical History.—Progressive muscular atrophy is an extremely slow and chronic disease from the beginning. The special causes are as little known as those of amyotrophic lateral sclerosis. Here, too, we shall probably have ultimately to fall back upon the hypothesis of conditions of congenital weakness in the affected systems of fibers. Hereditary and generic influences are often not to be detected, but they are sometimes undoubtedly present. We ourselves and other observers have repeatedly seen spinal muscular atrophy occur in several members of the same family. The disease sometimes begins in childhood and sometimes in youth, but often in more advanced age (from forty to fifty). Although, as we have said, a congenital weakness of the motor system is probably always to be regarded as the special cause of the disease, in some cases certain external conditions, especially persistent over-exertion of the muscles in hard work, seem to favor its outbreak.

The disease begins, by far the most frequently, in the upper extremities, and especially, as it seems, in the right arm, but sometimes in the left, or in both arms at once. As a rule, it begins with an atrophy of the short muscles of the thumb and of the hypothenar eminence, which is accompanied by a corresponding disturbance of functions (see the accompanying figures, 139 to 141). Other symptoms—disturbances of sensibility, paræsthesia, or pain—are usually entirely absent. The atrophy usually affects the abductor pollicis brevis and the opponens first, and then the flexor brevis and the adductor. We notice very early the characteristic sinking in and flattening of the ball of the thumb, and the abnormal position of the thumb, which is persistently approximated to the second metacarpal bone—the "ape hand." It becomes constantly harder and finally impossible to oppose the thumb to the other fingers (performed by the flexor brevis for the index and middle fingers and by the abductor brevis and opponens for the ring and little fingers). At the same time, or a little earlier or later, the interossei begin to atrophy, as is recognized by the sinking in of the interosseous spaces and the increasing incomplete extension of the terminal phalanges of the fingers. The atrophy of the lumbricales causes a visible flattening of the hollow of the hand. If the disturbance in the function of the interossei has reached a certain degree, the same claw-like position of the fingers appears, as we have already learned to recognize in ulnar paralysis (see Fig. 142), due to the contracture of the antagonist of the interossei, the extensor communis digitorum.

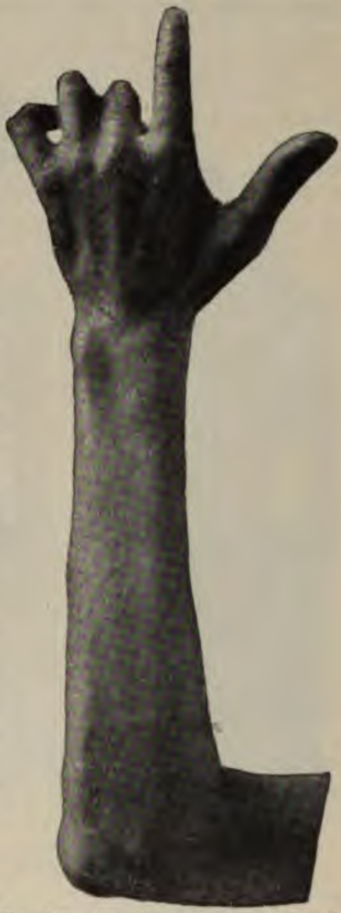
FIG. 142.—Atrophy of the small muscles of the hand and the muscles on the extensor side of the forearm in spinal progressive muscular atrophy. The last three fingers can no longer be extended. (Personal observation.)

In the further progress of the disease the atrophy either extends to the muscles of the forearm, or, what is not at all rare, it jumps over these and attacks the muscles of the shoulder, usually the deltoid first. In the forearm it is usually the muscles on the extensor side which are first attacked, the abductor and extensor longus pollicis, the extensors of the wrist and fingers, and only later the supinators, flexors, etc. In the upper arm the deltoid almost always atrophies first, and then the biceps, while the triceps may remain intact somewhat longer. There are also cases in which the atrophy begins in the deltoid and the muscles of the upper arm, while the small muscles of the hands and fingers are not affected until later. The muscles of the trunk are usually added to the list quite late: the muscles that extend the head and keep it erect, so that the head sinks forward, the trapezii, the infra-spinati and supra-spinati, the rhomboidei, the latissimi dorsi, the pectorals, etc. (see Figs. 143 and 144). The disturbance of function caused by the atrophy of all these muscles is readily apparent from what was said in the chapter on the different form of paralysis. In advanced cases the arms hang down laxly by the sides of the trunk. Only with the greatest difficulty, if at all, can the patient do anything with them—as to dress or undress himself; but he sometimes learns to help himself, at least in some degree, by "throwing" movements, by motions of the trunk, by using his mouth in taking hold of things, etc. Quite rarely the atrophy finally attacks the muscles of the neck. The severest respiratory disturbances, and finally a fatal termination, may be brought about by an implication of the diaphragm and the other muscles of respiration.

The time which elapses before the gradual appearance of the more marked disturbances of function is almost always very long. Years may elapse before the atrophy extends from the little muscles of the hand to the other muscles of the arm. In the muscles of the legs the first signs of atrophy almost always develop very late if at all. The arms are often perfectly useless when the patient can still walk for hours. Of course there are some exceptions to this rule, and then the muscles of the lower leg (peronei) especially seem to be first affected.

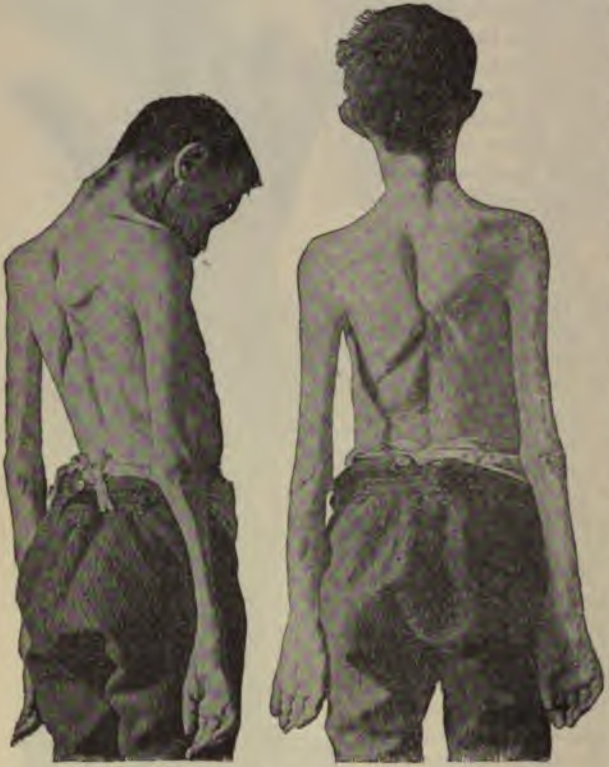
FIG. 143. FIG. 144.

FIGS. 143 and 144.—Spinal progressive muscular atrophy. (Personal observation.)

Besides the atrophy, and the loss of function that runs parallel to it, we must mention some other changes in the muscles. The fibrillary muscular twitchings are often very striking. A constant tremor and wave of the muscle may be provoked by them. In some cases they are weak and they can be detected only by special attention. They usually become vigorous if one irritates the muscle mechanically by a blow. The electrical excitability of the diseased muscles varies, inasmuch as it depends entirely upon the number of normal fibers still preserved in the muscle. Since the atrophy affects only one fiber after another, the faradic and galvanic excitability decrease gradually, and are lost completely only when the greatest part of the muscle is destroyed. On careful testing, we can then, however, almost always make out a decided galvanic reaction of degeneration in single muscles that are much diseased, especially in the form of the so-called partial reaction of degeneration: the excitability of the nerves is retained, but the contractions in the muscles themselves seem very slow, and the anodic closure contractions (AnSZ) predominate (see page 851).

In many cases there is an increase of the fatty tissue (which is always of moderate degree) at the same time with the atrophy of the muscular substance, and this often makes it very hard to judge of the atrophy; but the loss of function in the muscles, the diminished electrical excitability, and the peculiar soft feeling which atrophied muscles have when covered with fat, reveal the true condition of things. There are no trophic disturbances in the skin. Almost all the former statements in regard to this refer probably to the confusion between progressive muscular atrophy and similar forms of disease, especially syringomyelia. The coldness and cyanosis sometimes seen in the hands is due to the fact that the arms constantly hang down and to the lack of any muscular movement.

The examination of the tendon reflexes is of great diagnostic importance. While they are invariably decidedly increased in the upper extremities in amyotrophic lateral sclerosis, they are only feebly manifest or entirely absent in genuine progressive muscular atrophy, a condition which is easily explained by the atrophy of the motor ganglion-cells belonging to the reflex arc, or by the atrophy of the centrifugal motor fibers. Since no degeneration of the lateral pyramidal tract precedes this atrophy, it goes without saying that the muscular rigidity, associated with paresis and increase of the tendon reflexes, so characteristic of amyotrophic lateral sclerosis, is also absent. Muscular weakness and muscular atrophy are completely parallel in spinal progressive muscular atrophy. In the lower extremities the patellar reflex is retained as long as the disease spares the legs, but it is not increased. If the atrophy involve the legs, the patellar reflex is usually lost.

In distinction from all these manifest disturbances in the motor region, the sensibility of the skin and deeper parts remains perfectly preserved. A careful test of the sensibility is therefore indispensable to avoid mistaking it for similar morbid conditions, such as syringomyelia. There are also never any morbid symptoms on the part of the bladder or rectum.

In many cases the affection finally invades the muscular region innervated from the medulla; the symptoms of "progressive bulbar paralysis" (*vide infra*) are added to those of progressive muscular atrophy. This combination of spinal and bulbar disease, as we have previously shown, is a usual occurrence in amyotrophic lateral sclerosis, even when the disease has lasted a comparatively short time. In genuine progressive muscular atrophy, however, the bulbar symptoms usually develop, if at all, only after the disease has gone on for years. Then the speech begins to become indistinct from the atrophy of the tongue, swallowing is difficult, and the patient finally succumbs to increasing inanition or to respiratory disturbances. In their principal characteristics the muscular atrophy of the extremities and the bulbar symptoms, as we have said, are precisely analogous phenomena, inasmuch as the nerve nuclei in the medulla have precisely the same significance for the muscles of the tongue, the pharynx, and the face, as the anterior gray cornua of the cord have for the muscles of the trunk and the extremities. In many cases, however, bulbar symptoms do not develop at all, since the patients die early of some intercurrent disease, or the fibers of the bulbar region are not involved in the morbid process; but the whole process may begin, as we shall see later, in the region of the bulbar nerves as "bulbar paralysis," while the spinal muscular atrophy does not come on in the arms until afterward.

Diagnosis.—The diagnosis of spinal progressive muscular atrophy can easily be made if we confine ourselves strictly to the definition of the disease, and do not confuse it with other affections in which the muscular atrophy is only one symptom which may have an entirely different origin—muscular atrophies in extensive diffuse myelitis, in tumors, in the formation of cavities in the cord, in multiple neuritis, as a result of articular affections and injury, etc. We should consider especially the typical course of the affection in most cases of genuine progressive muscular atrophy, its beginning in the upper extremities, the small muscles of the hand, or more rarely the muscles of the shoulder and upper arm, its slow advance, the peculiar "individualization" of the atrophy—

that is, the affection of some muscles while other neighboring muscles remain completely normal; the special form of atrophy recognizable by fibrillary contractions and electrical reaction of degeneration; and, finally, the absence of all disturbances of sensibility or of the sphincters. Progressive muscular atrophy is doubtless nearly allied to amyotrophic lateral sclerosis, but the latter is distinguished by its more rapid course, and especially by the increase of the tendon reflexes due to the affection of the lateral columns, and the corresponding appearance of spastic symptoms. The differential diagnosis between the spinal and the myopathic (juvenile) muscular atrophy (muscular dystrophy), which is also perfectly certain at present in most cases, will be described later.

The **prognosis** of progressive muscular atrophy is to be regarded as absolutely unfavorable. The disease appears comparatively benign only from the fact that it is apt to advance very slowly, since it may last for ten or fifteen years, or even longer. As we have already said, the fatal termination at last appears from some intercurrent disease, or as a result of the final appearance of dangerous bulbar symptoms, paralysis of deglutition and respiration.

The results of **treatment** are accordingly very slight. An electrical treatment, continued for months and years with very great persistence, can alone produce a little improvement or check the advance of the atrophy somewhat. Temporary improvement may also sometimes be attained by methodical massage of the muscles and a rationally conducted gymnastic treatment. In other respects, the treatment must be symptomatic. [Gowers claims that daily subcutaneous injections of strychnine, grains $\frac{1}{100}$ to $\frac{1}{40}$ (gramme 0.0005–0.002), often arrest the disease.—K.]

4. NEUROTIC MUSCULAR ATROPHY

(Type Charcot-Marie. Peroneal-Forearm Type of Progressive Muscular Atrophy)

This name is given to a form of progressive muscular atrophy which has become known through many recent observations (Charcot and Marie, Tooth, J. Hoffmann, and others), and which is manifested by several very characteristic clinical features. The disease is often a pronounced hereditary and family affection, which can sometimes be traced through four or five generations, but, on the other hand, it also occurs in members of the same generation without being present in the antecedents, and individual cases have also repeatedly been observed in which there was apparently no hereditary predisposition. The first symptoms of the disease usually appear in childhood, but some cases have also been observed in later life. There is no essential difference between the two sexes.

Next to the conditions mentioned (beginning in childhood, generic occurrence), the most noticeable feature is the very regular localization of the muscular atrophy (see Fig. 145). The atrophy almost always begins symmetrically in the portions of the extremities most distant from the trunk—that is, in the hands and forearms and the feet and lower legs. The lower extremities are often affected for a longer or shorter time (even for years) before the upper, but the contrary sequence may also occur. Since the atrophy of the small muscles of the feet causes symptoms that attract little attention (a claw-like position of the toes), the disease may often remain unnoticed until the peronei, the tibiales antici, and the long extensors of the great toe are involved. The gait then becomes impaired from the dropping of the toes and shows all the characteristic peculiarities of bilateral peroneal paralysis (*q. v.*). In older cases there often develops on both sides a complete talipes equinus or varo-equinus. As the atrophy progresses, the muscles of the calves gradually become affected, and finally the muscles of the thigh as well. Atrophy and paresis of the muscles go always hand in hand. The same is seen in the upper extremities. Here the small muscles of the hand, the interossei and thenar and hypothenar muscles, atrophy first, just as in spinal muscular atrophy and amyotrophic lateral sclerosis. The fingers therefore assume the claw position; the movements of the thumb become more and more impaired, so that it lies adducted against the hand. The atrophy then extends to the forearms, where the extensors are earlier and more markedly affected than the flexors. Later in the course of the disease, when the forearms and hands are already greatly emaciated, we can usually discover the beginning of atrophy in the muscles of the shoulder and upper arm. The muscles belonging to the trunk resist the morbid process the longest. The facial muscles also are usually spared, although manifest changes may finally occur even in them, but no disturbances have thus far been observed in the domain of the bulbar nerves. Finally we may mention the observations reported by Hänel where the disease was traced in many members of one family through four generations and was always confined to the upper extremities.

It is of the greatest importance for our comprehension of the whole disease that the atrophying muscles show fibrillary contractions, usually slight but sometimes lively, and that we can invariably find the most marked changes in the electrical excitability of the muscles and their corresponding nerves. The nerves soon show a marked diminution or even a total loss of galvanic and faradic excitability. In the muscles we find either the same conditions or a pronounced galvanic reaction of degeneration. It is a very interesting fact that, on electrical examination, we can often detect manifest changes in excitability, just as in lead paralysis, in muscles which apparently show no atrophy or paralysis at all.

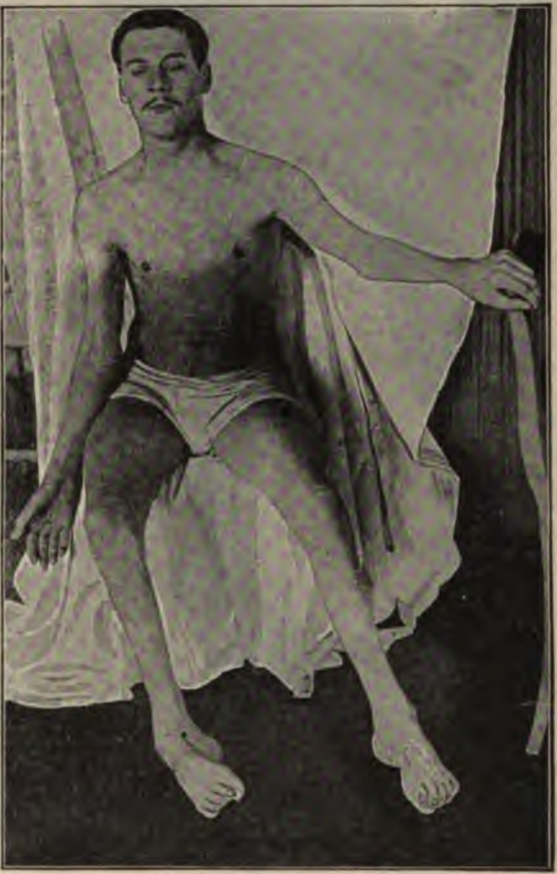

Fig. 145.—Neurotic muscular atrophy. The disease came on in almost the same fashion in three members of one family. (Personal observation).

The mechanical excitability of the muscles is also diminished. The tendon reflexes of the affected muscles are wholly absent or much diminished. They are never increased. The sensibility is never much affected, but it is often not perfectly normal. Paræsthesiæ and painful sensations in particular are often present, and there is sometimes a manifest but slight blunting of the cutaneous sensibility. The functions of the bladder and rectum remain normal.

From the clinical facts just described the conclusion may be drawn that the form of muscular atrophy under discussion must have a neurotic or a spinal origin. The assumption of a neurotic origin is favored not only by the results of the electrical examination and the occurrence of pain and slight sensory disturbances, but also by the results of the anatomical investigations thus far reported, although they are of course still few in number. According to these, the disease seems to be confined mainly to the muscles and the peripheral motor nerves, while the anterior roots and the ganglion-cells in the anterior horns show no perceptible alteration, but a moderate loss of fibers is sometimes found in the region of the columns of Goll.

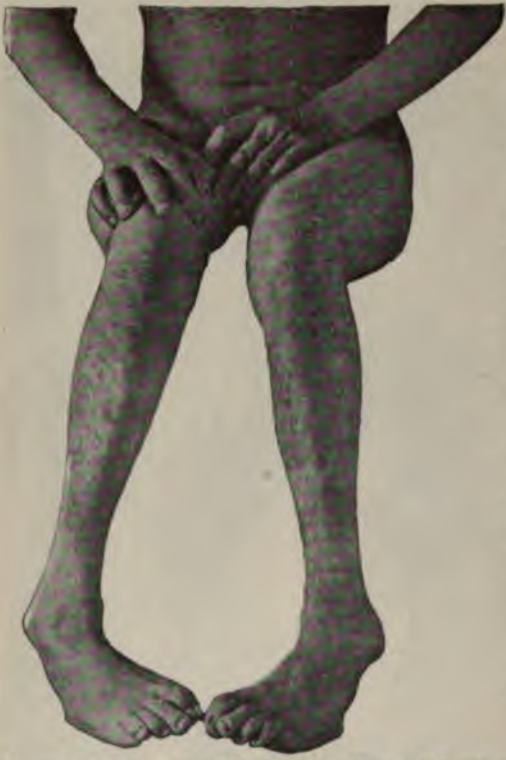

FIG. 146.—Peroneal muscular atrophy. Legs of the brother of the patient shown in Fig. 145.

Further observations and investigations must teach us whether, as is probable, we shall find closer relations and transition forms between neurotic and spinal atrophy; but meanwhile the disease just described is a form of progressive muscular atrophy so well characterized and so readily recognizable in its clinical aspect that a separate description of it is undoubtedly warranted.

5. PROGRESSIVE MUSCULAR DYSTROPHY

(The Myopathic Juvenile Forms of Progressive Muscular Atrophy)

The so-called purely myopathic muscular atrophies are much commoner than the forms of muscular atrophy thus far described, in which, besides the wasting of the muscular fiber, there is a more or less extensive atrophy of the motor nervous system. Myopathic muscular atrophy is the name given to all those forms in which anatomical investigation has thus far found atrophic processes, not in the cord or in the peripheral nerves, but exclusively in the muscles themselves. The old, long contest whether the "seat" of progressive muscular atrophy was to be looked for in the spinal cord or in the muscles themselves, has thus at last been decided: that each of these hypotheses was correct for certain cases. Thanks to persistent clinical and anatomical investigations, we are now in a position to distinguish, easily and positively for the most part, the spinal (and neurotic) from the myopathic cases even during the patient's life. Of course many facts even now seem to indicate that there is no very strict division, and that even here transition forms may occur which can be fully understood only by a still more accurate insight into the nature of the morbid process in question.

In spite of many common peculiarities, the individual cases of myopathic muscular atrophy differ in many respects from one another. These differences relate either to the localization of the muscles chiefly or at least first affected, or to the special variety of change in the muscles (simple atrophy or atrophy with a large interstitial development of fat). These differences were the reason why it was formerly believed that several forms of disease could be recognized, whereas in reality we are dealing only with different types of the same disease. These varieties, of course, have so much individuality and so constantly recur in the same way that we are perfectly justified in differentiating them, from a clinical standpoint; but, on the other hand, there are so many transitions between the different types that their intimate relationship is undoubted. Erb has therefore been perfectly correct in proposing to group together all these different types under one common name—*progressive muscular dystrophy*—in order to attest their kinship. We can then distinguish the different sub-types by special terms.

Before we proceed in what follows to describe these different sub-types in greater detail, we would first mention the most important points which are common to all forms of myopathic muscular atrophy—that is, of so-called muscular dystrophy. They are as follows: 1. All forms of dystrophy occur generically or hereditarily. We therefore see the disease most frequently in brothers and sisters, or more rarely in members of the same family in different generations. On the other hand, sporadic cases are of course not uncommon. If the disease occurs in members of the same family it usually assumes the same form in each of them; but different forms may exceptionally occur in one family—a circumstance strongly suggesting an intimate relation between the different types. 2. The first symptoms of the disease almost always appear in youth, sometimes in childhood, sometimes not until puberty. Only in rare cases does the disease begin between thirty and forty or even later. It is worthy of note that certain forms usually begin in childhood ("infantile" form with implication of the facial muscles, pseudo-hypertrophy), and other forms only at puberty ("juvenile" form with implication of the shoulder and upper-arm muscles). 3. The localization of the muscular atrophy is extremely irregular. The different forms or types are of course distinguished by their special localization; but as the disease advances, or in the frequent transition cases, it often happens that other muscular regions besides those chiefly affected are also involved to a lesser degree. It thus appears that in all forms it is finally the same muscular regions which are chiefly affected with atrophy, while, on the other hand, in all forms certain muscular regions with scarcely an exception remain intact. The groups of muscles most exposed to the disease are: (*a*) Facial muscles, as far as they are innervated by the facial nerve, especially the orbicularis palpebrarum and orbicularis oris. (*b*) Muscles of the shoulder-girdle: pectoralis, latissimus dorsi, serratus anticus major, rhomboidei, trapezius. (*c*) Muscles of the upper arm: biceps, brachialis anticus, supinator longus, triceps. (*d*) Long extensors of the back (erectores spinæ). (*e*) Pelvic muscles (glutæi) and muscles of the thigh. Other muscles, however, are almost always spared in muscular dystrophy: (*a*) Deltoids, supraspinati, and infra-spinati. (*b*) Forearm muscles, and especially the small muscles of the hand and fingers. (*c*) Lower leg muscles, especially the gastrocnemius and the small muscles of the foot. (*d*) Tongue and muscles of deglutition. All these statements are not absolutely without exception, but they show the usual rule, which must depend upon the essential nature of the disease. 4. The anatomical changes in the muscles are essentially the same in all the different forms of dystrophy. They consist first of the most varied changes in volume of the individual muscular fibers. Many fibers show an atrophy which progresses finally to complete disappearance, while, on the other hand, so-called hypertrophic (hypervoluminous) fibers are also almost always to be found. Erb suspects that the hypertrophy is the first stage of the disease of the fiber, and that the atrophy follows this later. Besides these changes in volume, we see regularly an evident increase in the muscle nuclei and a considerable increase in the interstitial connective tissue. If there is pronounced pseudo-hypertrophy the interstitial tissue is permeated with an abundant accumulation of fat-cells. In the muscular fibers themselves the transverse striation is almost always preserved to the last, and we never find granular or fatty, so-called degenerative destruction in any great degree. The peripheral motor nerves and the motor ganglion-cells in the anterior horns—that is, the peripheral motor neurones—remain perfectly normal. Two clinical facts, which are very important in diagnosis, are associated with this circumstance: (*a*) In all forms of dystrophy the fibrillary contractions in the diseased muscles, which are so common in spinal atrophies, are almost invariably absent. (*b*) Distinct electrical reaction of degeneration is almost never found in dystrophy (in contrast to the spinal atrophies). Since the disease is also a motor system disease, like all the other forms of progressive muscular atrophy, it is self-evident that in dystrophy the sensibility, the functions of the bladder, etc., remain perfectly normal.

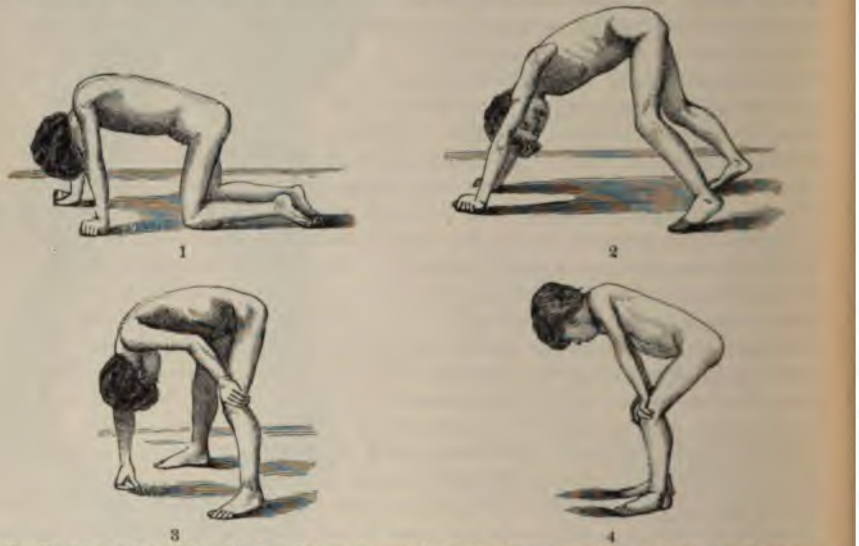

FIG. 147.—Positions of a child with hereditary (pseudo-hypertrophic) muscular atrophy, on rising to an erect attitude. (From GOWERS.)

Since we have mentioned the most important features common to all forms of dystrophy, we will pass to the description of the chief individual clinical types. We would emphasize the fact once more that transition forms between these types are by no means rare.

1. PSEUDO-HYPERTROPHY OF THE MUSCLES (*Lipomatosis luxurians muscularis progressiva* of Heller; *Atrophia musculorum lipomatosa* of Seidel).—Pseudo-hypertrophy is the rarest form of muscular dystrophy, but it was the first to become accurately known, on account of its striking aspect. It was quite fully described in Germany by Griesinger (1864), while in France Duchenne gave in 1868 a very complete clinical description of it. In 1866 M. Eulenburg and Cohnheim had already shown, by the careful anatomical examination of a case, that the nervous system remained perfectly normal, a statement which was confirmed by all subsequent investigations (Charcot, F. Schultze, and others).

Pseudo-hypertrophy develops almost invariably in childhood, somewhere between the ages of five and eight years. It is a pronounced family or even hereditary disease. The male sex is decidedly more disposed to the disease than the female [but the disease seems to be transmitted through the female in the hereditary cases.—K.] Sometimes, but not always, we also find in the affected families some disposition to a nervous taint, such as hysteria, epilepsy, feeble-mindedness, anomalies of the skull, etc.

The disease begins gradually and almost always without a special exciting cause. The parents notice that the child, who had been previously perfectly well and strong, becomes insecure on his legs, so that he can no longer jump or go up-stairs as well as he used to do. This points to the first characteristic peculiarity wherein pseudo-hypertrophy differs from spinal progressive muscular atrophy. It begins, with rare exceptions, in the muscles of the trunk, especially in the muscles of the back and pelvic muscles (glutæi), and in the muscles of the lower extremities, especially in those of the thigh. While the arms and hands are still perfectly normal, walking constantly grows more and more difficult, and the gait very soon assumes so characteristic a type that from this alone the diagnosis can often be made at the first glance. The gait becomes waddling (chiefly on account of the weakness of the glutæi medii), the belly appears very prominent, the vertebral column is arched forward in the lumbar region in marked lordosis, and the whole upper part of the body is balanced on the legs. The legs are raised slowly and with difficulty, and the toes usually droop from paresis of the dorsal extensors. The child's movements, when he tries to raise himself from the floor or pick up any object, are very characteristic, and are alike in almost all cases, as a result of paresis of the extensors of the thigh and leg (glutæi and extensores cruris). Since it is impossible to raise the trunk, the child usually first gets on all fours, and then gradually straightens himself up ["climbs up his thighs"] by leaning his arms on his knees (see Fig. 147). Later on disturbances of motion appear in the upper extremities also, and in general they are very similar to those to be described more fully in the next type.

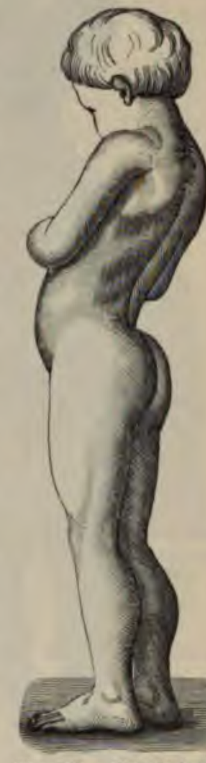
Fig. 148. — Pseudo-hypertrophy of the muscles. (From Duchenne.)

If we inspect the patient more closely we shall usually find at the first glance an extraordinary increase in the volume of single muscles (see Fig. 148). The calves are disproportionately thick, and sometimes the thighs also; the arms are affected later, especially the deltoids, the triceps, etc. This increase of volume is caused by an abnormal interstitial development of fat, "pseudo-hypertrophy." Hence the muscles do not feel firm, but soft and spongy. It is by no means rare, however, that, besides the pseudo-hypertrophy in some muscles, a genuine atrophy develops in others, with a pronounced loss of substance and without any co-existing development of fat. This is seen especially in the upper extremities in the groups of muscles previously mentioned. Finally, there seems to be in addition even a genuine muscular hypertrophy. In several cases we have seen a marked increase in volume in the muscles of the calves, which were capable of quite an extraordinary display of strength. In such cases, however, there is, probably, in our opinion, a sort of compensatory hypertrophy, since the muscles that are still able to work are exerted excessively.

Fibrillary twitchings of the muscles are scarcely ever seen. Electrical examination shows a diminution of excitability corresponding to the atrophy and to the increased deposit of fat, but never reaction of degeneration. The sensibility remains perfectly normal, and also micturition and defecation. The patellar reflex is sometimes absent, because the quadriceps is involved. It is noticeable that the skin, especially in the legs, very often shows a peculiar bluish marbled coloring. Bulbar symptoms apparently never occur. The intelligence in most cases is perfectly normal, but exceptionally children with hereditary muscular atrophy also show manifest signs of mental or even of moral weakness.

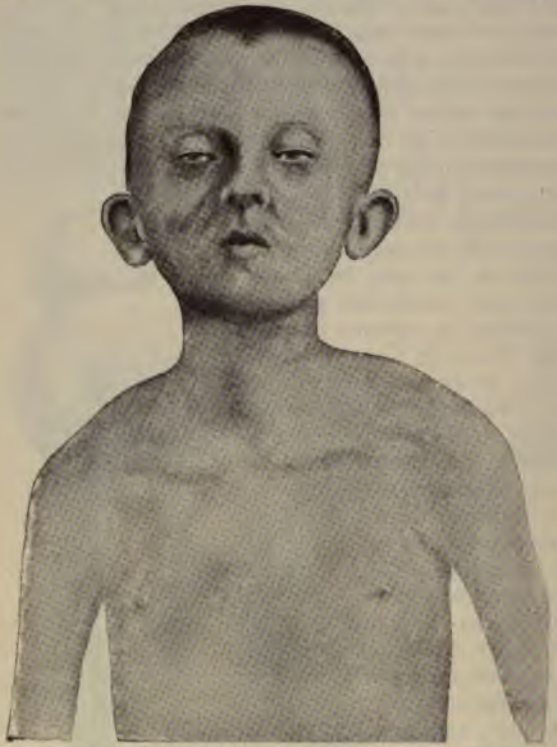

FIG. 149.—Juvenile myopathic muscular atrophy in a ten-year-old child, with marked implication of the facial muscles. Inability to close the eyes or move the lips. Atrophy of the pectorals, etc. (From the Erlangen Medical Clinique.)

The disease advances very slowly but without remissions. Finally the patient can not walk at all; he is confined to the bed, and becomes more and more helpless. Death usually ensues from some intercurrent disease, but sometimes from insufficiency of the respiratory muscles.

2. **The Infantile Atrophic Form of Muscular Dystrophy usually with Implication of the Facial Muscles.**—Simple atrophic forms of progressive muscular wasting develop far more frequently in early childhood than pseudo-hypertrophy. They may be combined with pseudo-hypertrophy, as we have said above, especially in the form where there is lipomatosis in the lower extremities and simple atrophy in the shoulders and arms; but what gives the simple atrophic infantile form a special stamp is the comparatively frequent implication of the facial muscles. These muscles may not be involved, and, on the other hand, they may be affected in rare instances in the juvenile form as well, but marked atrophy of the facial muscles is a peculiarity chiefly of the infantile atrophic form.

Duchenne had already observed that in children there is a form of muscular atrophy which begins in the muscles of the face; but his statements were almost forgotten until, a few years ago, Landouzy and Dejerine called attention anew to this subject, and demonstrated, contrary to previous belief, that implication of the facial muscles is by no means an unusual symptom. If the atrophy is already plainly pronounced, characteristic symptoms ensue: the eyes can no longer be completely closed, and the movements of the mouth in whistling, laughing, and talking become impaired. From the sinking in of the cheeks, the drooping of the under lip, etc., there develops a very characteristic type of countenance (“*facies myopathique*”), by which the practiced eye can recognize the affection almost at once (Fig. 149). There may finally ensue complete rigidity and immobility of the face. At the same time or somewhat later, as a rule, other muscular regions may be affected, and again, as in other forms, chiefly the shoulder muscles, the muscles of the upper arms, the extensors of the spine, and the muscles of the pelvis and thigh. The child's gait becomes as waddling as that of children with pseudo-hypertophy, which we have just described. The spine has a lordotic curvature, the child rises from the floor by the aid of the arms (Fig. 147), etc. The sequence in which the different muscles are attacked by the atrophy is not always the same. The facial muscles, for example, may often be attacked later than the muscles of the shoulder, upper arm, and trunk. In order to detect the slighter degrees of implication of the face, it is advisable to have the patient close his eyes when the head is bent backward; we can then readily detect the fissure

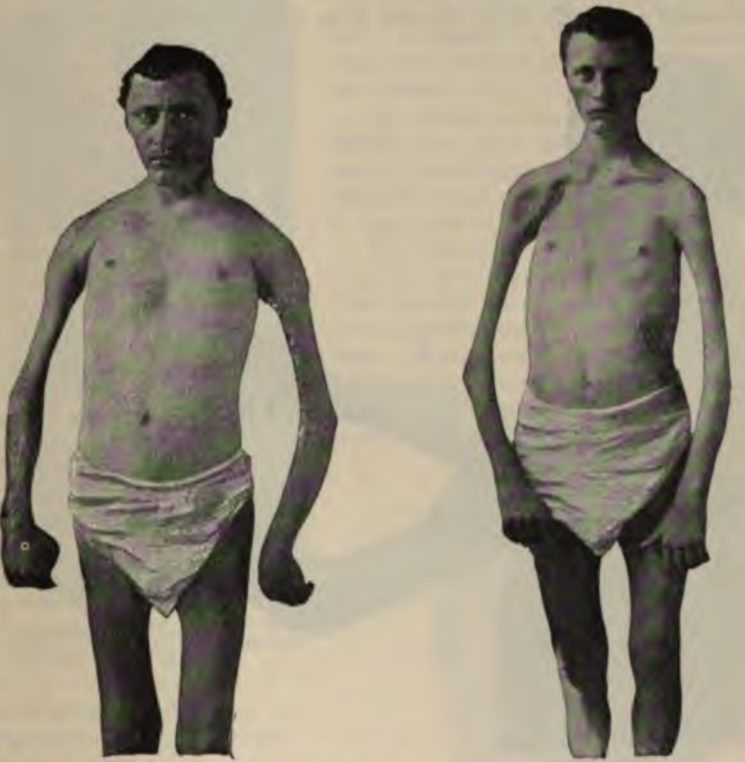

FIG. 150.—Two brothers with juvenile muscular atrophy. (Personal observation from the Erlangen Medical Clinique.)

left between the eyelids (insufficiency of the orbicularis palpebrarum). We can also notice in the early stages that the patient can not protrude the lips to whistle.

The muscles of mastication, the internal ocular muscles, and the muscles of the forearm and hand almost always remain normal. A slight persistent contracture of the biceps is sometimes present and is characteristic. Fibrillary contractions and reaction of degeneration are almost always absent.

3. THE JUVENILE FORM OF DYSTROPHY.—The peculiarities of this form were first carefully studied by Erb. It usually does not begin until after actual childhood, in the years of puberty or sometimes later (between twenty and forty). It occurs sporadically occasionally, but it is also frequently hereditary or generic (that is, occurring in families), and the girls of the family are often attacked

by this form, while the infantile form is seen especially in boys. The disease is also, like pseudo-hypertrophy, sometimes seen to begin in the pelvic muscles and the legs, but more often the shoulders and upper extremities are first attacked. There is also a remarkable regularity in the choice of the muscles attacked, as has been mentioned above. According to Erb, the following muscles are almost constantly diseased in the trunk and the upper extremities: the pectoralis major and minor, the trapezius, the latissimus dorsi, the serratus anticus major, the rhomboidei, the sacro-lumbalis and longissimus dorsi, and later the triceps. The following, however, almost always remain normal; the sterno-mastoid, the levator anguli scapulæ, the coraco-brachialis, the teres major and minor, the deltoid, the supra-spinatus and infra-spinatus, and, as must be mentioned especially in distinction from spinal muscular atrophy, the small muscles of the hand. The muscles of the forearm, too, except the supinator longus, remain intact for a long time, if not permanently; but exceptions also occur: for example, the accompanying illustration (Fig. 150) shows two brothers with juvenile muscular atrophy which in other respects is characteristic, but the muscles on the extensor side of the forearm are also completely atrophied. In the lower extremities the atrophy attacks chiefly the glutæi and the quadriceps, more rarely the peronei and the tibialis anticus, while the sartorius and the muscles of the calf are usually wholly exempt. Fibrillary twitchings in the affected muscles are generally absent, and there is hardly ever any reaction of degeneration.

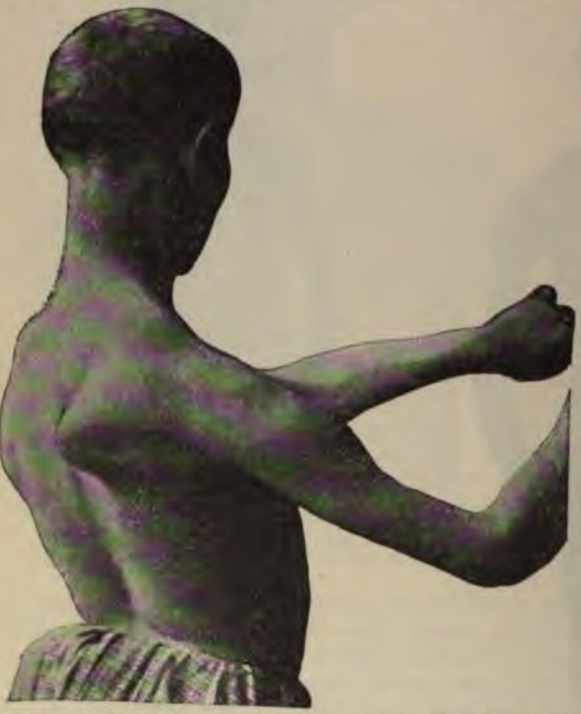

FIG. 151.—Juvenile muscular dystrophy. Prominence of the scapulæ when the arms are raised in consequence of atrophy of the serratus and rhomboidei. (Personal observation from the Erlangen Medical Clinique.)

The disturbances of function due to this condition are self-evident, so that a full description of them may be omitted. The arms usually suffer first, as we have said. The marked projection of the scapulæ, from the paralysis of the serratus, is especially characteristic (Fig. 152). If we try to take the patient under the shoulders and lift him, the shoulders are perfectly limp. They are therefore immediately raised so high that the head comes between the shoulders ("loose shoulders"). The most striking features in the anterior wall of the chest are the flattening in the region of the pectoralis and the peculiar folds of skin on the anterior edges of the axillæ. If the patient walks we can often recognize the disease at a distance at the first glance by the peculiar gait. The upper part of the body is carried backward (lordosis of the lumbar vertebræ), and the gait is waddling, since the pelvis

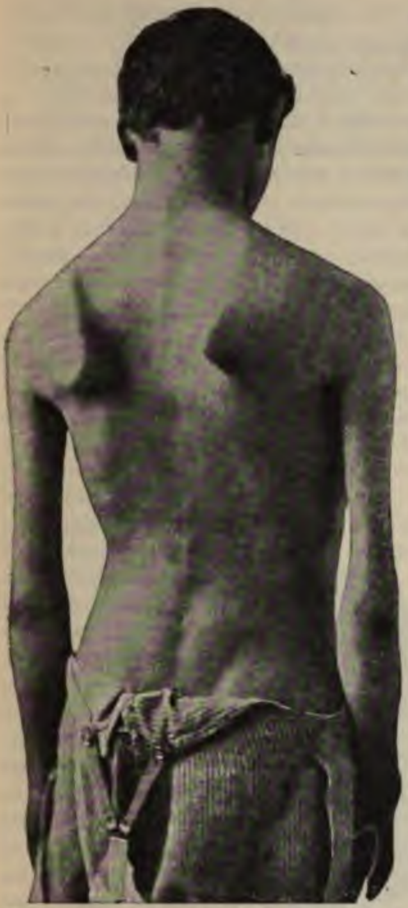

FIG. 152.—Muscular dystrophy. Prominence of the scapulæ when the arms are hanging down. (Erlangen Medical Clinique.)

can no longer be held properly by the weak glutæi. On stooping and rising, the same symptoms are seen as have previously been described (Fig. 147). As a rule, in the juvenile form the face is not involved, but we can sometimes detect slight disturbances of the facial muscles, especially defective closure of the lids and inability to whistle (Fig. 153). We once made the interesting observation that the adult brother of a young girl suffering from juvenile dystrophy showed these (congenital?) anomalies in the face from earliest childhood, without showing any further indications of dystrophy. Bulbar symptoms are as rare as in the other forms. It is of significance that the intercostal muscles and the diaphragm may finally atrophy, and the consequent respiratory disturbance may be the cause of death.

The course of juvenile dystrophy is very chronic, like that of all the other forms. The disease progresses very slowly and often seems almost to stand still for a long time. Many cases have been under observation for twenty or thirty years.

We may mention in conclusion that in some cases of dystrophy the trouble has been seen to begin at a more advanced age—from forty to fifty—but in other respects these cases do not differ from the general type, although we have noticed that, in such cases as begin relatively late in life, the muscles of the pelvis and thighs were chiefly affected, so that the patient could not stand or walk while the arms still performed their functions tolerably well (Fig. 154).

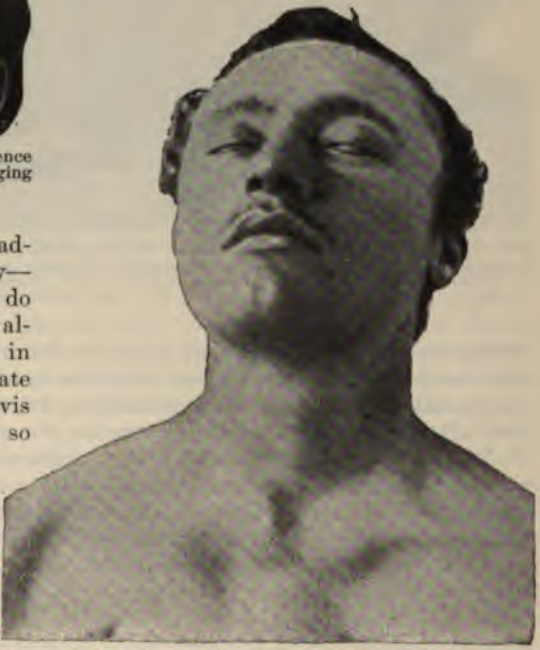

FIG. 153.—Juvenile muscular dystrophy. Insufficient closure of the eyes. Myopathic facies. (Personal observation.)

Diagnosis of Myopathic Muscular Atrophy (Muscular Dystrophy).—Now that the type of myopathic muscular dystrophy has been established in all its details by numerous observations, the diagnosis is usually no longer difficult. The generic occurrence of the disease, its development usually in childhood or youth, the remarkable regularity in the selection of muscles affected, the absence of fibrillary contractions and also of electrical reaction of degeneration in the affected muscles, are the chief points upon which the diagnosis is to be based in the individual case. If we bear in mind these peculiarities, the distinction from the spinal and neurotic amyotrophies or from other similar types of disease (syringomyelia) is usually easy. Only a few cases, which clinically seem to present transition forms, remain for a time uncertain in their anatomical determination. We must, however, indicate one condition which may sometimes give rise to mistakes in diagnosis —namely, *congenital muscular defects.* In some persons we find that certain muscles or parts of muscles are absent from birth, and it is remarkable that it is most frequently the same muscles which are most often affected in muscular dystrophy, especially the pectoralis major, or at least its sternal portion, the serrati, the rhomboidei, the trapezii, etc. Such congenital muscular defects naturally present at the first glance a very similar picture to that of a patient with dystrophy; but we can usually decide correctly as to the condition by careful inquiry, since in case of congenital muscular defects the abnormalities have always been present, and they also show no noticeable increase in the course of years.

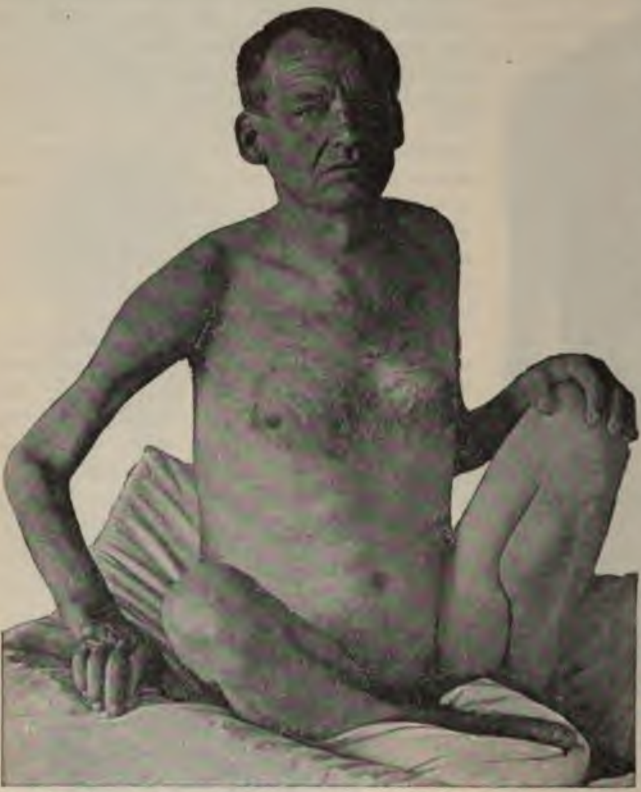

FIG. 154.—Progressive myopathic muscular atrophy of the pectorals, upper arms, and muscles of the pelvis and thigh. Ordinary position of the patient in sitting; he can neither stand nor walk. (Personal observation.)

Treatment.—The treatment of myopathic muscular dystrophy is seldom successful. We may sometimes obtain a certain improvement by persistent treatment by electricity or massage; but at present any considerable alteration in the coure of the disease is beyond the domain of possibility. [Persistent and systematic gymnastic exercises sometimes have a decidedly beneficial effect.—K.]

6. PRIMARY DEGENERATION OF THE PYRAMIDAL TRACTS

(*Primary Lateral Sclerosis. Spastic Spinal Paralysis. Motor Tabes. Spasmodic Tabes Dorsalis*)

While in all the forms of primary disease of the motor system thus far deribed the muscles are involved primarily or secondarily, and muscular atrophy erefore plays a leading part in the clinical picture of all the diseases described ove, in rare cases the degeneration may be limited to the central neurone *PyS* of Fig. 137, page 986) of the motor tract. In such cases a type of disease velops, in which indeed the disturbances are exclusively motor, but in which uscular atrophy is completely absent—a type of disease to which has been given e name of "*spastic spinal paralysis*" (Erb, Charcot). There is no doubt that ch primary, isolated degeneration of the pyramidal tract may occur, without volving the peripheral motor neurone (cell of the anterior horn with its process, e peripheral motor nerve); but we must add that primary degeneration of the ramidal tract is especially apt to be associated with degeneration of other sysms of neurones (lateral cerebellar tract, fibers of the posterior columns), so that e anatomical picture of the disease is not a simple one, but corresponds to a *combined system disease.*"

Type of Spastic Spinal Paralysis.—Three symptoms predominate in the picure of spastic spinal paralysis: motor paresis, hypertonicity of the muscles (muslar rigidity), and the increase of the tendon reflexes—the patellar reflex, and kle clonus. The loss of motor power—we are speaking at present only of the astic paralysis of the legs, which is by far the most frequent and the most early marked—is found in various degrees, from a simple weakness of moveent to a complete and more or less extensive paralysis. The other two sympms, however—the hypertonicity (rigidity) of the muscles, and the increase of e tendon reflexes—give the disturbance of motion its characteristic "spastic" ature. Both symptoms, indeed, are to be regarded as in part identical, for the gidity of the muscles is also of reflex origin. If the tendon reflexes be much ineased, the reflex tension of the muscles comes on even upon the stretching and lling of the tendons, which are excited by the weight of the limbs, or by any tive or passive movements. The reflex muscular tension opposes any attempt motion. The muscles feel rigid and firm, and the legs are often found in most permanent contracture and extended, with the feet in plantar flexion. If e try to flex the leg passively at the knee, or if we try to extend the foot dorsally, e find it almost impossible to do so. The more rapidly and suddenly we try to oduce the motion, the more marked is the muscular resistance, which can arcely be overcome. If, however, we go to work very slowly and cautiously, and oid any sudden tension of the tendons, we can almost always flex the leg witht special trouble. If we put the patient on the edge of the bed, the legs do not ng down laxly, but they usually fall at once into a vigorous extensor tetanus, nce the weight of the leg puts the quadriceps into contraction by the tension on e ligamentum patellæ. A convulsive, reflex tremor of the whole leg often comes at once, similar to ankle clonus. If we examine the patient while in the bath e find the spasms decidedly less, because in the water the influence of the weight f the limb is diminished. It must still remain undecided whether the existence f muscular rigidity in spastic paralysis is to be explained exclusively by the crease in the tendon reflexes. In some muscles there is sometimes so persistent condition of tonic irritation that we are led to think of processes of direct, bnormal motor irritation. Thus the adductors of the thigh, in particular, nd often the dorsal extensors of the great and other toes, etc., show a constant gidity and tension, the reflex origin of which can not be established, at least ith any certainty.

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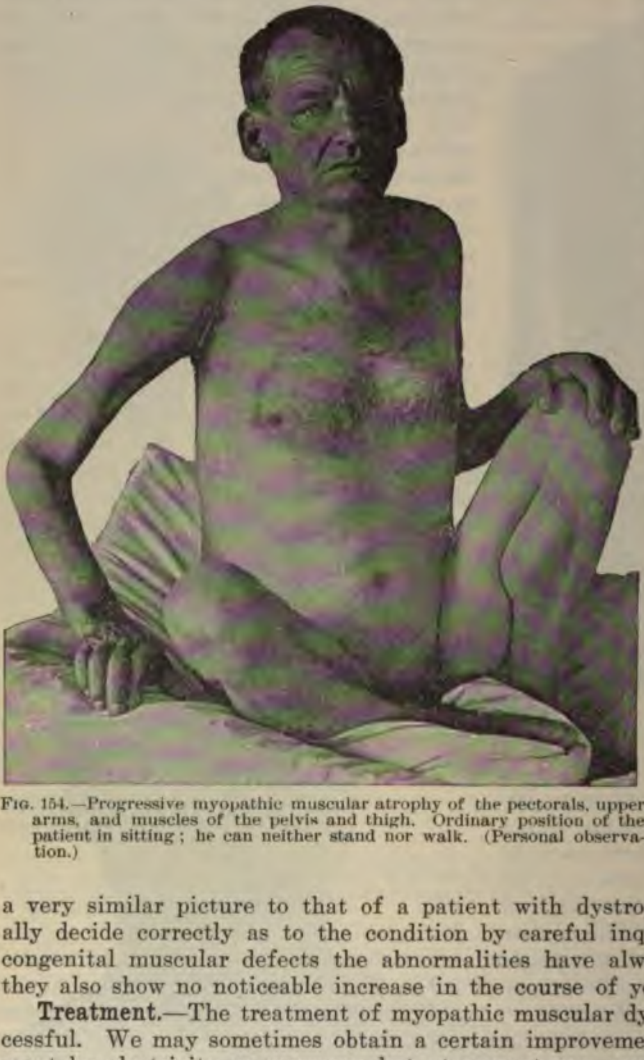

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(Primary Lateral Sclerosis. Spastic Spinal Paralysis. Motor Tabes. Spasmodic Tabes Dorsalis)

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Active motion, as is easily explained, must also be impaired from the inhibitory action of the tonic and reflex spasms. The degree of disturbance of motion is thus increased, and the paresis often seems greater than is really the case. The influence of the muscular tension is especially manifest in the gait of the patient. As long as walking is still possible, we notice very plainly that it is rendered difficult, not only by the muscular paresis, but also by the stiffness of the legs. The patient walks with short and difficult steps, the legs are scarcely flexed at the knee, and the feet are scarcely raised at all. The feet "stick to the floor" and are slowly slid forward, and there is a marked tendency to walk on the toes, from the contraction in the muscles of the calves. The weight of the body alone presses the feet downward. We term this very characteristic form of gait the spastic-paretic gait. It is also characteristic of spastic paralysis of the legs that the patient is unable to execute certain isolated movements. Thus the patient, like a hemiplegic (*vide infra*), often can not dorsally extend the foot by itself; but if he flexes the whole leg and draws it up to the trunk, there always follows a marked dorsal extension of the foot with great tension of the tendon of the tibialis anticus muscle.

The increase in the tendon reflexes may also exist without the presence of any special motor paresis of the muscles at the same time; but since the motion is not a little influenced in such a case by the constant spasms, a disturbance of motility may be produced, which we have called "spastic pseudo-paralysis," or, more properly, pseudo-paresis. In these cases the muscular strength is almost normal, and the patient can walk for quite a long time. Nevertheless, all his movements are stiff and difficult, and the gait shows all the peculiarities of the pure spastic gait. The steps are not very short and they follow one another quite rapidly, but the legs remain perfectly stiff and are scarcely raised from the ground, and the patient walks almost entirely on his toes. In the house the patient walks with a noisy shuffle, and in soft sand we can see the furrows drawn by the feet as they slide along the ground. Since the Achilles tendon is made tense at each step, and since a reflex contraction of the muscles of the calf responds to this tension, each step is often followed by a short lifting of the whole body ("teetering" spastic gait).

Although the symptoms mentioned are the characteristic positive features of "spastic spinal paralysis," yet, in the original sense of the word, the affection is also characterized by the fact that certain other spinal symptoms, especially disturbances of sensibility, disturbances in micturition and defecation, muscular atrophy, and other trophic symptoms, are entirely absent. Only when such was the case have Erb and Charcot claimed that a special anatomical cause must lie at the bottom of the peculiar group of symptoms, which they suspected to be a primary disease of the lateral columns—that is, of the pyramidal tracts particularly. This suspicion was entirely warranted, since the picture of pure spastic spinal paralysis could be explained by no other anatomical theory. A degeneration in the lateral columns explains the motor paresis, explains the increase of the tendon reflexes by the loss of reflex inhibitory influences, and finally it also explains any direct tonic contraction of the muscles by the hypothesis of processes of motor irritation. Actual evidence of the occurrence of primary systemic degeneration in the lateral columns could of course be furnished only by anatomical observation. This has now actually taken place, so that at present there can no longer be any doubt of the occurrence of a "spastic spinal paralysis" in the original sense of Charcot and Erb. If we observe the same morbid picture not only in primary degeneration of the pyramidal tract, but also in other cases, it is due to the fact that, under certain circumstances, other diseases may, at least for a time, involve the lateral columns almost exclusively, and especially the pyramidal tracts.

So far as our present knowledge extends, the affection comes on in two somewhat distinct clinical forms. The first may be brought into the closest relation with amyotrophic lateral sclerosis. As we have seen above (page 989), we often find in typical amyotrophic lateral sclerosis a pure spastic paresis in the legs without any trace of muscular atrophy, while in the arms and the bulbar region there is a more or less marked muscular atrophy besides the spastic symptoms. There are also cases of disease—they occur usually in somewhat advanced life and sporadically, that is, not generically as a family disease—where spastic rigidity and paresis develop not only in the legs, but also in the trunk, the arms, and the face, without a trace of muscular atrophy. In the course of a few years the patient finally becomes almost completely stiff (Fig. 155), without being entirely paralyzed. The stiffness is due solely to the muscular rigidity. The arms also show pronounced hypertonicity on passive movement. Impulsive laughing and crying also occur, just as in amyotrophic lateral sclerosis (Fig. 156). If we have the rare opportunity to make an autopsy on such a case, we find a marked systemic degeneration in both lateral columns, chiefly in the region of the pyramidal tract, *PyS*, but usually involving other bundles of fibers in the lateral columns as well. The degeneration of the pyramidal tract, *PyS*, may be traced upward through the pyramids, the pons, and the crus to the internal capsule. The ganglion-cells in the anterior horns of the spinal cord are entirely normal or they show only insignificant changes. The close relationship between this form of "pure lateral sclerosis" and amyotrophic lateral sclerosis is shown by the fact that the one disease may develop into the other. After the typical affection of lateral sclerosis has continued in its pure form for some years, atrophy may sometimes finally develop in the small muscles of the hand, the tongue, etc.—in short, there is clinical evidence that the peripheral motor neurone has begun to degenerate; but the atrophy, as a rule, remains comparatively slight, although different degrees of transition forms probably occur.

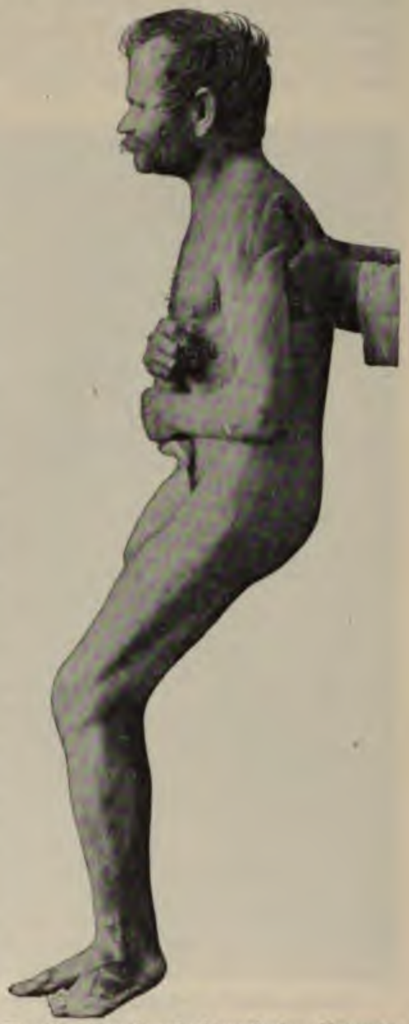

FIG. 155.—Spastic spinal paralysis. Enormous muscular rigidity of the trunk, arms, and legs. When supported from behind, the patient can stand as stiff as a post. (Erlangen Medical Clinique.)

A second well-characterized form of primary degeneration of the pyramidal tract is the hereditary or family spastic spinal paralysis which has been described by us. This shows itself in several members of the same family, especially in brothers and sisters; in this form there very gradually develops, in the course of years and years, a constantly increasing rigidity of the legs with increased tendon reflexes and all the motor disturbances dependent thereon. The gait is pronouncedly spastic, the feet cling to the floor, the legs are pressed firmly together from the contracture of the adductors, and on walking, one foot can pass the other only with difficulty. The patient treads only on the anterior part of the foot; the whole body is projected stiffly forward, so that the patient has to hold a cane in front of himself to keep from falling forward (Fig. 157). For a long time the purely spastic symptoms outweigh the paretic (spastic pseudo-paralysis), and only very gradually does the spastic gait become spastic-paretic, and an actual loss of muscular strength ensue. The general condition, the sensibility, the bladder and the muscular nutrition may remain unaffected for a long time or until the end. The disease usually begins somewhere between twenty and thirty, but some cases apparently occur in children also. There can hardly be any doubt that the disease is due to a congenital morbid predisposition of the pyramidal tract, and that thus it is closely connected, ætiologically, with the other forms of hereditary systemic disease, especially with hereditary muscular atrophy; but the anatomical examination of the spinal cord, which has been possible thus far in only a few cases, has shown disease in the lateral cerebellar tract (*KlS*) and the columns of Goll, besides degeneration in the pyramidal lateral tract (*PyS*)—that is, in the territory of the neurones of the lumbar spinal ganglia. Anatomically, therefore, hereditary spastic spinal paralysis must also be termed a combined systemic disease.

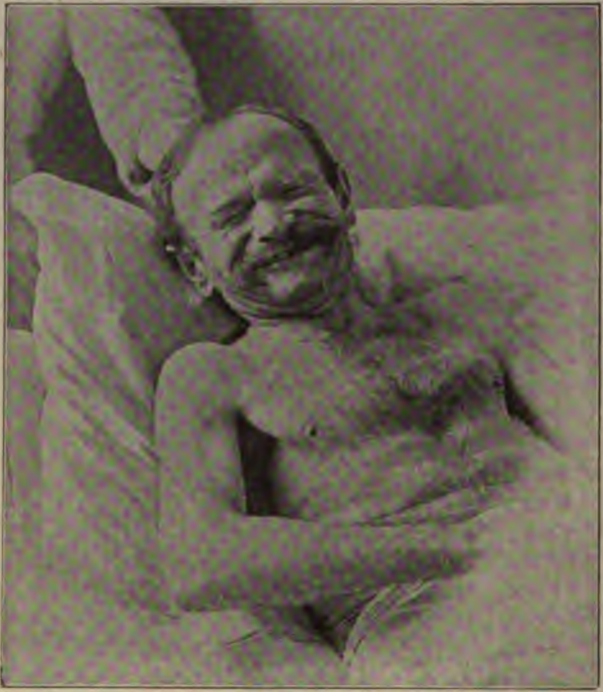

FIG. 156.—Spastic spinal paralysis. Impulsive laughing. The same patient as Fig. 155. Symptoms of amyotrophic lateral sclerosis came on later. (Erlangen Medical Clinique.)

Besides the two forms of primary degeneration of the pyramidal lateral tracts (*PyS*) already mentioned, which in our opinion are perfectly well established clinically and anatomically, there are in reality several other forms of disease whose clinical symptoms are in the main those of spastic spinal paralysis, while the anatomical changes consist of a primary, more or less strictly systemic disease of the lateral columns alone, or of the lateral and posterior columns. In the latter case ataxia, bladder disturbances, slight disturbances of sensation, etc., may at times be added to the spastic symptoms; but since we have very little definite knowledge of these rare forms of disease, we can not at present give any suitable description of them. Many of these diseases seem to be exogenous in origin and to be especially connected with previous syphilis. Other cases are related ætiologically to severe general constitutional diseases, especially pernicious anæmia (page 723). We may also mention that a pure type of spastic spinal paralysis may be produced by chronic poisoning with lathyrus seeds (lathyrus sativus and lathyrus cicera, the so-called vetches, which are used to make bread in southern countries). Systemic degenerations of the spinal cord also occur in pellagra, which is common in northern Italy from the continued use of diseased or fermented maize. This sometimes causes especially marked spastic symptoms, besides, of course, many other spinal and mental symptoms (Tuczek).

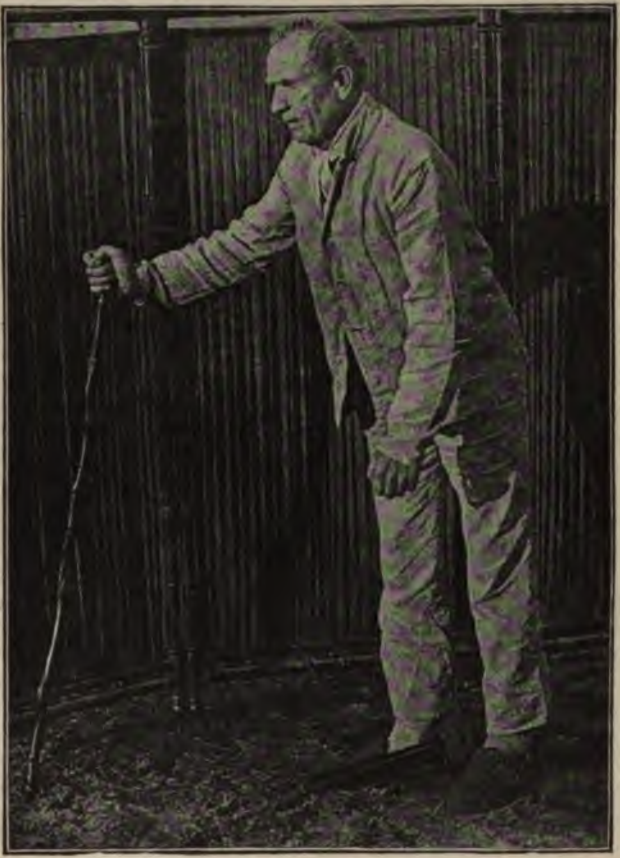

FIG. 157.—Family spastic spinal paralysis. (Erlangen Medical Clinique.)

[Gowers has given the name of *ataxic paraplegia* to a class of cases of combined degeneration of the lateral and posterior columns, which are by no means rare. The disease comes on gradually in middle life, usually without any definite cause. Men are much more frequently affected than women. The early symptoms resemble those of spastic paraplegia, with the addition of ataxic unsteadiness, which may be at first the most prominent symptom. There are very rarely any lightning pains, but there is at times a dull pain in the legs or the sacrum. There is rarely any impairment of sensation. The tendon reflexes are usually much exaggerated. Sexual power may be lost early, and the bladder and rectum are often affected. Reflex immobility of the pupils and optic atrophy are rare, but nystagmus is occasionally noticed. The arms are usually involved later. Mental symptoms are rare. The disease is slowly progressive, but it rarely proves fatal, death being usually due to some intercurrent disease. The extent of the degeneration in the cord varies, but it is never so well marked in the posterior columns as it is in true tabes. Putnam has also described a type of degeneration occurring also in adult life, but much more frequent in women. In his cases the trouble seemed to be not infrequently associated with profound anæmia or other forms of malnutrition. Paræsthesia is a very prominent symptom of the earlier stages of the disease, and it may come on suddenly or rapidly increase. Its appearance in an anæmic person of middle age is therefore suggestive of the onset of this form of disease. Disturbances of micturition are also very common in the early stages. Inco-ordination and muscular weakness develop later, and the weakness may increase to complete paraplegia. The reflexes are usually exaggerated, but they may be absent. The disease is apt to terminate fatally within four or five years. The pathological changes are of a degenerative nature, and are most marked in the posterior and lateral columns, but they are quite diffuse, so that this type of disease is more closely allied to diffuse chronic myelitis than to a systemic degeneration. In some cases systematic gymnastic exercises, by Fränkel's method, prove of decided benefit. The treatment otherwise is the same as that of chronic myelitis.—K.]

Diagnosis.—Since the symptom-complex of spastic spinal paralysis must always arise if the pyramidal tracts are affected by any disease in any part of the two lateral columns of the spinal cord or even higher up, it is easily seen that it may often be very difficult or even quite impossible to decide in a given case whether we have to do with a primary systemic degeneration or with some other circumscribed disease of the pyramidal tracts. The chief weight in diagnosis should always be laid on the general development of the morbid condition, the ætiological conditions (several in a family affected), and the strict limitation of the symptomatic picture (absence of any sensory or bladder disturbances, etc.).

The morbid conditions chiefly to be considered in differential diagnosis are the following: 1. *Multiple sclerosis.* As previously stated (page 956), this may for some time resemble very closely the type of pure spastic spinal paralysis. 2. *Syphilitic disease of the upper dorsal cord,* the so-called *syphilitic spastic spinal paralysis.* We will consider this important disease more fully in a subsequent chapter on syphilis of the spinal cord. 3. *Chronic hydrocephalus* (*vide infra,* the section on diseases of the brain). 4. *Subacute and chronic myelitis* following other acute diseases (typhoid fever, influenza, etc.). This can, as a rule, be easily distinguished from genuine spastic spinal paralysis by the mode of origin, and also by the clinical symptoms. 5. Slight *compression of the spinal cord,* from vertebral caries, tumors, traumatic affections of the vertebræ, etc., may for some time give rise to typical symptoms of spastic spinal paralysis, but with careful attention and longer observation there will only rarely be any mistake.

Prognosis and Treatment.—The prognosis of genuine spastic spinal paralysis is naturally as unfavorable as that of any other systemic disease. The treatment can therefore be merely symptomatic. The spastic symptoms, which are often very troublesome, are most readily alleviated by the use of long-continued warm baths. In other respects the treatment follows the rules that obtain in all chronic affections of the spinal cord.

APPENDIX

CONGENITAL SPASTIC PARAPLEGIA IN CHILDREN

(*Spastic Cerebral Paralysis. Infantile Spastic Diplegia. Little's Disease*)

In this place we would also speak briefly of a morbid condition, first described by J. Heine and the English physician Little, which is not very rarely observed in children. The symptoms in these cases are probably usually congenital, or at least they arise during birth (*vide infra*), but from reasons that are easy to understand they are not noticed until later. When the children come under medical observation they are usually from three to six years old, or even older.

The most striking symptom is that the child can not walk at all, or he walks only with difficulty and with assistance. If we examine the legs carefully we usually find that they are quite stiff. The thighs are usually somewhat flexed and firmly adducted, and also rotated inward. The knees are also slightly bent, while the feet are often in a position of equinus or equino-varus (Fig. 158). From the contracture of the thighs in adduction it readily happens that the legs cross each other when the child lies down, or especially when he tries to walk. Passive movements of the legs can be performed only by overcoming more or less muscular resistance. The tendon reflexes, especially the patellar reflex, are much increased, and there is often a persistent ankle clonus. The sensibility and micturition are normal. The arms, as a rule, are readily moved, but slighter spastic symptoms may also occur in them. Many of these children are mentally perfectly normal, learn to talk and write well, etc.; but in other cases cerebral disturbances are also plainly manifested, feeble intelligence, defective speech, epileptic attacks, strabismus, etc. As the child's physical development proceeds the symptoms often improve, but in general the condition is stationary and not progressive.

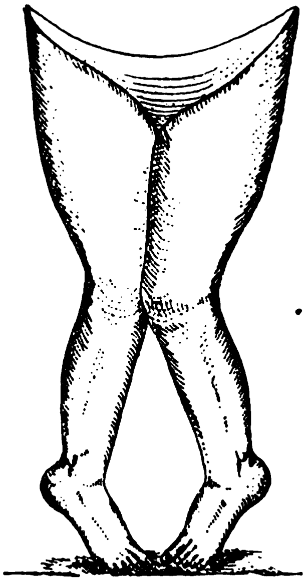

FIG. 158.—Position of the legs in congenital spastic cerebral paralysis. (Sketch from personal observation.)

Our knowledge is still very defective as to the causes of these congenital spastic paraplegias and the anatomical changes which underlie them. First of all we must make a fundamental division of the cases into two groups. In the one we have to do with children who are born with great difficulty and with artificial aid (forceps, etc.). In this group we can probably assume injuries of the brain in parturition from hæmorrhage, etc., which result in permanent disturbances of function. In the other group, where the history affords no such evidence (except that often the children are born prematurely), we usually have to assume some congenital defect (so-called porencephaly in the cerebrum, congenital aplasia of the motor centers or of the whole pyramidal tract). This congenital form of spastic paraplegia may also occur generically, but it is to be distinguished from the hereditary spastic paralysis above described, which develops slowly and only in later life and is progressive.

In the way of treatment, we may expect most from regular gymnastic exercises. Surgical interference (tenotomy, transplantation of tendons) and orthopædic appliances may sometimes considerably improve the child's ability to walk.

CHAPTER X

ACUTE AND CHRONIC POLIOMYELITIS

1. Spinal Paralysis of Children

(Acute Poliomyelitis in Children)

Ætiology and Pathological Anatomy.—In children there occurs quite frequently a definite and well-characterized form of paralysis, for the first accurate knowledge of which we must thank Jacob von Heine in 1840. Although Heine subsequently, in 1860, expressed the opinion that a disease of the spinal cord formed the basis of the paralysis, the first actual confirmation of this opinion was furnished by Prévost and Vulpian, Charcot and Joffroy, and others, so that at present we are justified in exchanging the old term "essential paralysis of children" for the name of "spinal paralysis of children."

As the name indicates, the affection occurs chiefly, if not exclusively (*vide infra*), in children, and is most frequent in the earlier years, somewhere between one and four. An exciting cause, such as taking cold, has hardly ever been made out. The children are almost always perfectly healthy* previously, and come of healthy families without any neuropathic predisposition. The whole course of the disease makes the hypothesis very probable that we have to do with an acute infectious disease—with an infectious process, which often causes first a mild or severe general infection of the body, and then is localized chiefly in a circumscribed portion of the spinal cord. It also, perhaps, bears some relation to this hypothesis that most of the cases occur in warm weather. The fact should also be mentioned that small and even extensive epidemics of spinal paralysis of children have been repeatedly observed. We have ourselves seen three children fall ill in a little village in a few days with acute poliomyelitis.

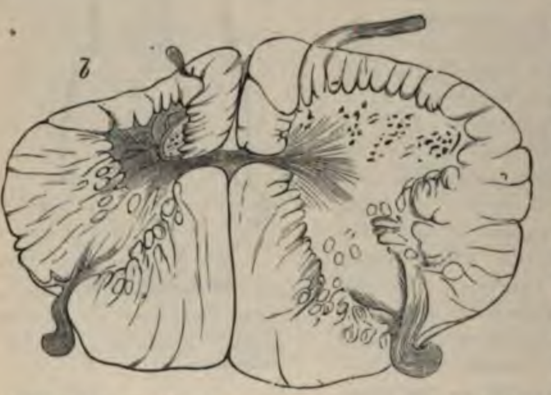

Fig. 159.—Section through the cervical enlargement in anterior poliomyelitis; the left anterior column is very much contracted and is without ganglion-cells. (From Charcot and Joffroy.)

With reference to its anatomy, the disease may be defined as an acute inflammation, which affects chiefly a definite portion of the anterior gray matter of the spinal cord, usually attacking only the anterior gray horn of one side; yet it does not always limit itself strictly to this, but it may involve the white matter in the vicinity somewhat, although, of course, to a lesser extent than the gray matter. Although fresh cases have so far been examined in very scanty numbers, still we can sometimes make out clearly the signs of inflammation in the older centers of disease. The lesion which is most frequently found in old cases consists of a considerable atrophy of one anterior horn, which is cicatrized and shriveled and changed to a dense sclerosed tissue, often pierced by dilated and thickened vessels, and containing scarcely a single normal ganglion-cell. If the paralysis affects one arm, the corresponding anterior horn in the cervical enlargement is atrophied (see Fig. 159); if the leg be paralyzed, the process is seated in the lumbar enlargement. In bilateral paralysis we must think of an affection of both anterior horns at the corresponding level of the cord.

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* The paralyses arising after acute diseases—such as measles, scarlet fever, small-pox, etc.—are perhaps partly of spinal origin, but they can not be identified with the idiopathic spinal paralysis of children.

This inflammation of the anterior horn, the poliomyelitis, is to be regarded as the primary focus of disease, dependent upon the local action of the excitants of inflammation. From this point, as in every severe lesion of the motor ganglion-cells there situated, there develops a secondary degeneration, which, extending to the periphery, affects the corresponding anterior roots, and later their appropriate motor nerves and the muscles supplied by them. In the paralyzed muscles and nerves we accordingly find a high degree of degeneration and atrophy, such as we have learned to recognize in severe peripheral paralyses.

Although at present the spinal origin of the atrophic paralysis of children is regarded as sufficiently certain, we must mention that it is not improbable, at least in many cases, that the peripheral nerves are also primarily involved; the poliomyelitis may be combined with a multiple neuritis, produced by the same causes and thus co-ordinate. Under this heading should be classed those cases where there is at first great pain in the extremities, especially on pressure and passive movement. The rapid disappearance of many of the initial paralytic symptoms may perhaps be referred in part to the presence of co-existing neuritic processes. We shall also see later that there are perhaps ætiological relations between spinal and cerebral infantile paralysis.

Clinical History.—The disease almost always begins suddenly. A child who was previously perfectly well and lively is all at once attacked with severe fever, often reaching 105° or 106° (40°–41° C.), and quite severe general symptoms are also associated with the fever even from the beginning. The child complains of headache, and sometimes of pain in the loins and in the limbs (*vide supra*), and is decidedly stupid and somnolent. Very often still more marked cerebral symptoms develop: complete loss of consciousness, single twitchings in the face or the extremities, or general convulsions. The eclamptic attacks, turning of the eyes, and clonic contractions in the face and extremities, sometimes appear even at the beginning of the disease. On the part of other organs we see most frequently gastro-intestinal symptoms, especially vomiting, more rarely diarrhœa. All the initial symptoms, whose intensity varies very much in the different cases, sometimes last only a very short time—a day or two—although they often continue for a week or two. Indeed, we even know cases in which, as the mothers have assured us, the children are said to have "lain in spasms," almost uninterruptedly, even for four or five weeks before the beginning of the paralysis—that is, before it became noticeable. On the other hand, however, it may happen that the initial symptoms, especially the severe cerebral symptoms, are entirely absent or greatly modified.

After the initial period of the disease just described has passed away, the parents usually notice that the child is attacked by a more or less extensive paralysis. If its development can be followed closely, we always find that it spreads rapidly, often in single spurts which quickly follow one another, so that it usually reaches a considerable extent in a short time. Either both legs, or the legs and one arm, or all the extremities, and even the muscles of the trunk, are affected. In some cases one oculo-motor or facial nerve has been observed to be affected at the beginning, a symptom which we would most readily refer to a neuritic process. The paralysis scarcely ever remains permanently as extensively distributed as at first; it diminishes rapidly, and soon is limited to a definite muscular region, which remains permanently paralyzed. In some cases the paralysis may entirely disappear, but as a rule a complete paralysis is left in one extremity, or at least in a portion of it; most frequently in one leg, especially in the peroneal muscles; somewhat more rarely in the arm, chiefly in the deltoid; sometimes in both legs; or, very rarely in spinal paralysis, in one arm and leg on the same side or on opposite sides. Meantime the child's general health has been completely restored. He is well and vigorous, has an excellent appetite, never shows any permanent cerebral disturbance—only the painless, flaccid paralysis, the inability to use the affected extremity, is left behind. In the following weeks and months a further, slower improvement in the power of motion often becomes noticeable, but, as a rule, a permanent and more or less complete paralysis of certain muscles remains.

In regard to the more intimate peculiarities of this remaining paralysis, it may invariably be characterized as a flaccid atrophic paralysis. A marked atrophy of the paralyzed muscles shows itself a few weeks after the beginning of the paralysis. This atrophy gradually spreads, and it may finally attain the highest degree. The atrophy is often, but not always, partly concealed by a more abundant development of fat tissue. The changes in the electrical excitability of the paralyzed nerves and muscles come on still more rapidly than the visible atrophy. Since we have to do with a complete degeneration of the nerves, as follows from the anatomical basis of the disease, a pronounced electrical reaction of degeneration must necessarily develop in the affected parts. Duchenne found that usually the faradic excitability of the nerves and muscles is completely lost after a week or two. On galvanic examination, we can at first detect an increase of excitability in the muscles with a predominance of slow anodic closure contractions (AnSZ), while later, after two or three months, the galvanic excitability also sinks very considerably; but the muscular contractions preserve their qualitative peculiarities characteristic of reaction of degeneration (see page 847). Very often the whole affected extremity remains backward in its growth, so that later the bones may show a shortening of several centimetres (Fig. 160). The parallelism between the muscular atrophy and the stunted growth is not, however, present in all cases, as Volkmann, in particular, has stated.

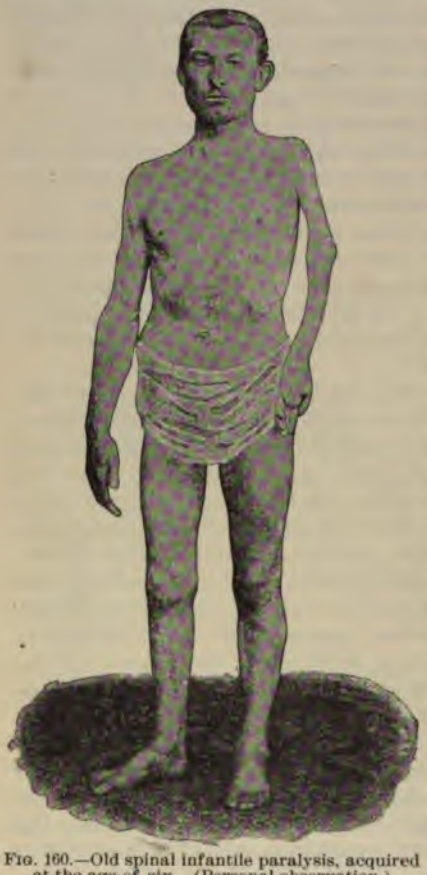

FIG. 160.—Old spinal infantile paralysis, acquired at the age of six. (Personal observation.)

Passive motion of the paralyzed extremity is at first, on account of the absence or diminution of the muscular tonus, perfectly free, and so remains except for the contractures that finally set in (*vide infra*). Many joints are so flaccid that we can actually make flapping movements with the paralyzed limbs and give them the most extraordinary positions. The tendon reflexes are invariably completely absent in the paralyzed extremities, and so almost always are the cutaneous reflexes—a condition which is to be referred to the interruption of the reflex arc in the anterior horns, which may sometimes be of diagnostic significance. The skin often shows certain trophic disturbances (atrophy, anomalies in the growth of the hair and in the secretion of sweat); it almost always feels cool and has a cyanotic appearance. Its sensibility, however, is completely retained in all cases. Micturition is sometimes a little disturbed at the beginning of the disease, but in most cases this disturbance completely disappears later.

After the paralysis has existed for a time, certain secondary contractures almost always develop in the paralyzed parts, some of which are of a very characteristic type. In the legs especially the "paralytic club-foot" (*talipes varo-equinus*) is a condition long known. It is due to the fact that, from the paralysis of the peronei muscles and of the tibialis anticus, the point of the foot constantly droops, and that a contracture is gradually developed in the antagonistic unparalyzed muscles of the calf, whose points of insertion are permanently approximated. In paralysis of the muscles of the calf there arises, on the other hand, a moderate degree of calcaneus from the contracture of the antagonists. In the arms and in the vertebral column, in paralyses of the spinal muscles, the most manifold and sometimes very considerable contractures and deformities may also arise, which are in the main always to be referred to the contracture of unparalyzed antagonists and to external mechanical conditions, such as weight and pressure.

In conclusion, if we simply compare the type of disease sketched with its anatomical cause, the general agreement of the two may at once be seen. The affection of the anterior gray cornua must have as a result a paralysis with a subsequent atrophy and reaction of degeneration, in which the reflexes must be lost by the destruction of the reflex arc, but the sensibility must remain perfectly normal from the persistence of the sensory conduction (the posterior columns and the posterior gray cornua), and the vesical functions must also remain normal. The subsequent paralysis is the result of the permanent destruction which the morbid process, in itself completely ended, has caused in the spinal cord.

We must also mention the peculiar fact which has been observed in some cases, that progressive muscular atrophy may come on in later life in persons who long before have had spinal infantile paralysis. The cells of the anterior horns once injured by the acute disease seem never to have become equal to the functional demands made upon them, and therefore they succumb to a premature atrophy.

Diagnosis.—The diagnosis of the spinal paralysis of children is almost always easy to make correctly, if we hold strictly to the definition and peculiarities of the disease, and do not reckon as spinal paralysis every paralysis appearing in a child. We should consider chiefly the acute beginning, the subsequent flaccid paralysis with atrophy and reaction of degeneration, with the loss of the reflexes, but with retained sensibility. If we observe these features, we are sufficiently protected against confusion with cerebral diseases and other diseases such as spondylitis, hereditary muscular atrophy, or spastic cerebral and spinal paralysis. We have already mentioned above that transitional forms between acute poliomyelitis and acute polyneuritis probably occur; but we would constantly emphasize one principal distinction between polyneuritis and genuine, circumscribed acute poliomyelitis—polyneuritis has a general, hæmatogenous, toxic or toxic-infectious origin, while acute poliomyelitis seems to be a true local inflammation, dependent upon the local presence of the (organized?) excitants of inflammation. In the clinical differential diagnosis we should consider chiefly the existence of any initial pains and any initial sensitiveness of the peripheral nerves and muscles. The further course of the disease may also be decisive, since the neuritic paralyses afford a much better prognosis than the paralyses of poliomyelitis.

Prognosis.—It is not impossible, but it is not yet proved, that many of the cases where children die speedily with convulsions are to be regarded as the initial stage of acute poliomyelitis. If, however, the first stage of the disease be past, the prognosis as regards life is entirely favorable, since the rest of the child's physical development is no further affected in any way. The prognosis as regards the complete restoration of the disturbance of function is, however, much more unfavorable. Although we should not forget that the paralysis is at first much more extensive than it is later, nevertheless this lessening of the paralytic symptoms occurs only in the first weeks or months. A limb which has not recovered in six to nine months usually remains paralyzed for life. Nevertheless, this should not restrain us from persevering in treatment, at least in the first years, since sometimes a very noticeable improvement in the functions of the paralyzed parts is thus procured.

Treatment.—If we have an opportunity, even during the initial stage of the disease (when, of course, the diagnosis can not usually be made with certainty), to attack the disease by our treatment, we may prescribe cold compresses or an ice-bag to the head, and eventually, when there is high fever or great stupor, a tepid bath with cool affusions. We are but rarely led to try blood-letting by leeches behind the ears or on the temples, when there are signs of marked cerebral hyperæmia. Internally we usually prescribe a mild "intestinal derivative," such as half a grain or a grain (gramme 0.03–0.05) of powdered calomel every two or three hours, infusion of senna, etc.

After paralysis appears, we may expect the most success from electrical treatment, kept up consecutively for months, and, with interruptions, for years. We put a large, broad electrode on the vertebral column at the spot which corresponds to the place of the lesion in the spinal cord—on the cervical vertebræ in paralysis of the arm, and the lower dorsal in paralysis of the leg—while the other electrode serves for peripheral application to the paralyzed nerves and muscles. In this way we apply a moderately strong constant current, reversing it occasionally, for two or three minutes, partly stabile and partly labile, passing the cathode, or eventually the anode, slowly over the paralyzed muscles and nerves. We may also employ occasional interruptions and reversals of the current. Duchenne has also found persistent treatment by the faradic current of advantage. The sittings should take place three or four times a week, and later even oftener, if possible.

Besides electrical treatment, methodical gymnastic exercises of the muscles, that can still be moved to some degree actively, may also be of distinct advantage. Regular and persistent massage of the muscles is also to be recommended in the later stages. In practice we can not avoid prescribing certain embrocations, such as spirit of camphor, spirit of mustard, or spirit of formic acid. Passive motion is very important to guard against contractures, and to improve the already existing deformities.

The use of baths, of brine or ferruginous waters, is to be recommended, although, of course, they must not be overvalued. They may be given at home. If circumstances permit sending the child to a bath during the summer months, we should chiefly consider the brine baths at Reichenhall, Kreuznach, Kösen, and Colberg; the acid salines at Rehme, Nauheim, and Soden; and eventually, with weak and anæmic children, the use of the iron baths at Driburg, Pyrmont, or Schwalbach. Good results are sometimes obtained at the indifferent thermal baths at Teplitz, Wildbad, Ragatz, or Gastein, but these must be used only with caution. We also obtain good results, especially with older children, at the cold-water cures.

Very little is to be expected from the use of internal remedies. Iodide of potassium and strychnine are recommended, the latter in the form of subcutaneous injections, $\frac{1}{60}$ to $\frac{1}{20}$ grain (gramme 0.001–0.003) daily.

When the acute process is wholly past and we can no longer count on any further regenerative process, the mechanical surgical treatment of the patient may often be of material service. In the first place the power of movement may be improved by orthopædic appliances, and troublesome contractures may also be relieved by tenotomy. The therapeutic attempts to join the tendons of muscles which can still contract to the tendons of the paralyzed muscles, in order to render it once more possible to obtain the effect of movement of these muscles (Nicolodoni, Vulpius, and others), are of especial interest. The symptomatic benefit thus obtained is very considerable. Details as to all the orthopædic and surgical methods of treatment are to be found in the special treatises. When we can no longer hope for any further improvement in the paralysis worth mentioning, the treatment is limited to improving the patient's general condition and strengthening him as much as possible by proper food and good air.

2. ACUTE POLIOMYELITIS OF ADULTS

(Acute Atrophic Spinal Paralysis of Adults)

Although it had been believed for a long time that the form of acute atrophic spinal paralysis, just described, occurred only in children, later observations by Moritz Meyer, Duchenne, Erb, F. Schultze, F. Müller, and others, have established the fact that precisely analogous cases of disease may also develop, although far less frequently, in adults, especially in young persons under thirty. There is no longer any doubt of this fact, especially if we consider an authentic anatomical lesion found by F. Schultze. We have, however, once before had to say that for a long time we have regarded the diagnosis of acute, and, as we shall soon see, of chronic poliomyelitis also, as too lightly made, and that certainly very many of the cases diagnosticated and published as poliomyelitis are to be classed as primary neuritis (see page 885). Since we know that primary degenerative processes may develop acutely and subacutely in the motor nerves, and may also lead to an atrophic paralysis, a greater part of the teaching on poliomyelitis needs new and careful revision in order to exclude what does not belong to it.

The type of acute poliomyelitis of adults, so far as it has been established by definite observations, which at present are not numerous, is not materially different from the type of the spinal paralysis of children.

We often can not make out any ætiological conditions; sometimes exposure to cold, over-exertion, etc., seem to favor the development of the disease. Cases are seen more frequently in men than in women.

The disease likewise begins with quite severe initial symptoms, fever, headache, somnolence, delirium, and vomiting, which may last from a few days to a week or two. The violent spontaneous pains which are very often reported in the extremities usually belong probably to those cases in which a primary neuritis, but not a poliomyelitis, is the chief anatomical lesion; but severe pain in the back may also occur in genuine poliomyelitis in the first acute stage of the disease. After the end of this first stage the paralysis appears. This develops with varying distribution, usually in single spurts, but always rather rapidly. The paralyzed muscles are perfectly flaccid, the cutaneous and tendon reflexes are wholly absent, and very soon a pronounced atrophy and reaction of degeneration appear, while the sensibility and the vesical and sexual functions remain normal.

The distribution of the paralysis shows certain peculiarities, which must be briefly described here, since they can be studied much better in adults than in children. The paralysis may be very extensive, it may affect all four extremities, or it may occur in the form of paraplegia, or even of monoplegia. In the extremities we very often find certain groups of muscles paralyzed, to which E. Remak first called attention. Since the muscles that are paralyzed at the same time are not supplied by the same peripheral nerves, but usually are connected in their functions, we may suppose that the corresponding ganglion-cells in the anterior cornua of the spinal cord also lie together, without regard to the distribution of their peripheral processes in the different motor nerves. Thus, for example, it is worthy of note that, in paralysis of the crural region, the sartorius often remains entirely free; that in the leg the tibialis anticus, on the one hand, and the peronei and the extensor digitorum on the other, may be separately diseased; that in the forearm the supinator longus, supplied by the radial nerve, remains free, while all the other muscles on the extensor side of the forearm are paralyzed ("forearm type" of E. Remak); and that, on the other hand, the supinator may be paralyzed alone or together with the biceps, brachialis anticus, and deltoid ("upper-arm type" of E. Remak). From the group of muscles paralyzed we can determine with some degree of accuracy the location of the disease in the cord according to the statements previously made in regard to the position of the spinal motor centers (page 921).

In regard to diagnosis, especial attention must be paid to the distinction between poliomyelitis and neuritis. The greatest stress is to be laid on the initial pains and any other slight disturbances of sensibility. In other respects, the course of the two diseases is so similar that we can indeed imagine that they are closely allied in their ætiological relations, and exhibit merely different forms of localization of the same (probably infectious) morbid agency. It always seems to us, however, as we have said above, that the principal distinction is that in genuine circumscribed poliomyelitis there is an actual local focus of inflammation, but in polyneuritis there is a toxic (hæmatogenous) degeneration of the peripheral motor neurones. In the latter case, of course, the motor ganglion-cell itself may also be destroyed if the disease be severe, but this would always be a different process from the local poliomyelitic inflammation.

The prognosis is not wholly unfavorable, as sometimes a complete recovery has been observed, although only after months. Of course, it is very possible that such cases are multiple neuritis. On the other hand, however, the same permanent paralyses as in spinal paralysis of children may be left, with atrophy and contractures.

The treatment follows precisely the same rules that we have mentioned in the spinal paralysis of children. The internal or subcutaneous use of ergotine may be added on the recommendation of some physicians. F. Müller recommends a solution of two and a half drachms of ergotine (grammes 10) with a third of a grain of sulphate of atropine (gramme 0.02) in five drachms of water (grammes 20), of which he injects seven to fifteen minims twice a day.

3. SUBACUTE AND CHRONIC POLIOMYELITIS

(*Subacute and Chronic Atrophic Spinal Paralysis. Paralysie générale spinale antérieure subaigue* [DUCHENNE])

While the anatomical basis of acute poliomyelitis in adults still leaves much to be desired, our anatomical knowledge of the occurrence of a subacute and chronic poliomyelitis, in the sense of the authors who have described it, is still more defective. Confusions with multiple neuritis are also undoubtedly very common here, and the diagnosis is not incontestable in all the cases published under the name of "subacute poliomyelitis"; but there can be no doubt, from some recent and perfectly accurate observations (Oppenheim, Nonne, and others), that extensive subacute and chronic diseases may occur in the anterior horns of the cord and lead to the development of extensive paralysis. The ætiological and general pathological conception of these cases is of course still wholly unsettled.

In the cases classed under this heading a paralysis, first of both legs and somewhat later usually of both arms, develops in a comparatively short time—in the course of some days, or weeks at most. It usually has no special cause or any severe initial symptoms. The patient complains at first of weakness in the legs; he can no longer walk, and he is confined to the bed. A short time later the same disturbances appear in the arms, and lead to a more or less complete paralysis. More rarely the opposite condition obtains, that the arms are affected first and the legs later, but the sensibility always remains wholly unaffected, except at most for slight paræsthesia, and there is also no pain, either spontaneous or occurring on pressure. Soon after the paralysis an equally extensive atrophy develops, and a distinct loss of electrical excitability, running parallel to it; which passes over into a partial or, in all severe cases, a complete reaction of degeneration. The cutaneous and tendon reflexes are very much diminished and often entirely lost. The bladder and rectum, however, remain intact, and bed-sores never develop if the patient has proper care. We sometimes notice a striking diminution of the sweat secretion. In rare cases the muscles of the neck, the lips, the tongue, and the pharynx are attacked by the disease.

After the paralysis has reached its greatest extent there is usually a pause. The condition remains stationary for months sometimes, and then a gradual improvement begins, which perhaps may go on to complete recovery; but often, of course, the recovery remains incomplete, so that the patient has a more or less marked disturbance of function for life. The "middle form of chronic poliomyelitis" described by Erb, in which there is only a partial reaction of degeneration in the paralyzed muscles, almost always gives a good prognosis. The disease takes an unfavorable course, if the respiration be impaired through involvement of the respiratory muscles, or, if the patient can not swallow, through involvement of the muscles of deglutition, or if inhalation pneumonia set in. The autopsy then shows extensive degenerative inflammatory changes in the anterior gray columns of the cord with a great destruction of ganglion-cells, while we can usually discover only very slight changes (secondary degeneration) in the peripheral nerves.

As a consequence of what has been said, we shall be compelled to make a diagnosis of chronic poliomyelitis when extensive paralysis comes on with subsequent atrophy and electrical reaction of degeneration and without any neuritic symptoms (pain!), and without disturbances of sensation or of the bladder. It is distinguished from "spinal muscular atrophy" proper, by the fact that in this there is a slow atrophy, attacking fiber after fiber, so to speak, and that the atrophy and muscular weakness go absolutely parallel, while in chronic poliomyelitis the paralysis precedes the atrophy, and whole muscles or muscular regions are affected from the start.

The treatment is not absolutely hopeless, as is shown by the above description. A persistent electrical treatment is always most advisable.

CHAPTER XI

THE SO-CALLED ACUTE ASCENDING SPINAL PARALYSIS

(*Paralysis Ascendens Acuta. Landry's Paralysis*)

In the year 1859 Landry described a disease under the name of "*paralysie ascendante aigue*," which is chiefly characterized clinically by the fact that first the lower and soon after the upper extremities, and finally a number of the muscular regions supplied by the medulla, are attacked by a rapidly advancing paralysis, while the sensibility and the functions of the bladder and rectum remain normal. In many cases the disease terminates fatally. Examination of the nervous system has so far, however, shown no lesion which can be regarded with certainty as the anatomical cause of the disease. Considering the continued and quite numerous observations of the disease, it seems questionable, at any rate, whether we can establish a uniform anatomical basis for it (*vide infra*); but the clinical picture of Landry's paralysis is so striking that a comprehensive description of it seems demanded.

General Symptomatology.—Acute ascending paralysis attacks chiefly persons previously strong and healthy, in youth or middle life, somewhere between twenty and thirty-five years of age. Some cases also have been seen in children and older people. The disease seems to be more frequent in men than in women.

The affection almost always begins with certain prodromata. These consist of general malaise, moderate febrile symptoms, headache, loss of appetite, and quite frequently of dragging and tearing pains in the back and the extremities. After these symptoms have lasted some days, or more rarely some weeks, during which they are either comparatively slight, or so severe that many patients are already confined to the bed, there usually comes on quite suddenly, or sometimes more gradually, a paresis, first of one leg, but very soon of both legs, which rapidly increases, and usually in a few days leads to an almost complete motor paraplegia.

The paralysis is flaccid in almost all cases. The legs may be moved passively without any muscular resistance, and the muscles show neither active nor reflex tension. Their electrical excitability remains perfectly normal in many cases, but there is sometimes a rapid loss of faradic muscular excitability. It is not yet proved whether complete reaction of degeneration occurs. The reflexes, both cutaneous and tendon reflexes, seem to be diminished or wholly lost in the majority of cases, but some exceptions to this rule have been known.

Sensibility is sometimes perfectly intact, but slight alterations do occur, and quite rarely there may be even marked anæsthesia. At times a noticeable delay of sensation is observed. We find no changes in the nerves of special sense. There is occasionally a slight œdema in the legs, which is perhaps to be regarded as a vaso-motor disturbance. The marked sweating, from which many patients suffer, is also worthy of mention. The bladder and rectum in most cases are not at all affected, or they present merely slight and temporary disturbances.

A short time after the legs are attacked the arms also begin to be paretic. A marked motor weakness appears first in one, then in the other arm, and this may also increase to almost complete paralysis. The sensibility, the reflexes, and the electrical excitability show conditions like those in the lower extremities. The muscles of the trunk are also affected at the same time as, or still earlier than, the arms. The patient can no longer sit up in bed, turn on his side, or make similar movements. In some cases a paralysis of the muscles of the neck has also been observed.

The third and last stage of the disease is characterized by the appearance of respiratory disturbances and bulbar symptoms. Manifest signs of a beginning respiratory paralysis appear; the respiration is labored and difficult, the movements of the diaphragm are impaired, and the paroxysms of coughing grow weaker. Disturbances in swallowing, difficulty in articulation, and paresis of the soft palate and the lips may set in. In a few cases a facial paralysis and disturbances of the ocular muscles have been observed. The condition grows worse acutely, and, as we have said, in many cases death ensues.

Besides the symptoms thus far mentioned, referable to the nervous system, we find certain other symptoms in almost every case, which are less striking, but yet of greater significance in judging of the disease. The first of these is fever. The temperature is usually elevated from the beginning; it may temporarily show a considerable increase, up to 104° (40° C.), and later it varies between 100° and 102° (38°–39° C.), but between times it may even sink to normal. Of the internal organs the spleen shows the most frequent changes. It is usually swollen moderately, but appreciably. There is also sometimes a slight albuminuria.

In the cases with a fatal termination the whole duration of the disease is sometimes only a few days, and as a rule a week or two, or rarely more. Fortunately, however, all cases do not terminate fatally. The disease may come to a standstill at any time, even if the most threatening symptoms be present. Then the paralysis shows no further advance, the disturbances present disappear, and recovery ensues after a course of several weeks. It is, of course, usually quite a long time before the patient again feels himself in possession of his full powers.

Pathological Anatomy and Pathogenesis.—If we consider the whole picture of acute ascending paralysis, the idea is necessarily forced upon us that we have to do here with an acute infection or auto-intoxication of the body, with a predominating localization in the motor nervous system. The beginning of the disease with general malaise corresponds perfectly to the prodromal stage of many other acute infectious diseases. The fever, the acute splenic tumor, and the occasional albuminuria also can scarcely be explained in any other way, according to our present views, except by the above hypothesis. There are, of course, no definite and positive data as yet in support of this hypothesis. A notable case, published by Baumgarten, in which many bacilli, like those of splenic fever, were found in the spinal cord, is at present wholly unique. Further investigations as to the anatomical changes in "acute ascending spinal paralysis" are still very desirable. In the earlier observations there is usually the statement of an "absolutely negative anatomical examination"; but these statements usually refer only to the spinal cord. The peripheral nerves were formerly not examined at all; and yet, as we have long proclaimed and as is now quite generally accepted, it is extremely probable from the clinical symptoms (pain, loss of electrical excitability, condition of the reflexes) that most cases of so-called Landry's paralysis are multiple neuritis (*q. v.*), and are nothing more than the most acute form thereof. Some recent anatomical investigations have already confirmed this hypothesis.

It would, however, be distinctly false at present to identify acute ascending paralysis completely with multiple neuritis, for in some observations (R. Schulz and F. Schultze, von den Velden) extensive acute myelitic changes have also been found in the motor portions of the spinal cord (lateral columns, anterior gray columns). We must therefore wait for many more observations and not decide prematurely upon the nosological position of the disease. [Recent investigations show that Landry's paralysis is an affection of the whole peripheral motor neurone, but that the ganglion-cells in the anterior horns show, as a rule, greater changes than the peripheral nerve-fibers.—K.]

Diagnosis and Prognosis.—In every paralysis of the lower extremities beginning acutely and accompanied by general symptoms and fever we must consider the possibility of an acute ascending paralysis, but only the further course of the disease can decide the question. Inasmuch as merely a well-characterized clinical group of symptoms is meant by the above term, the diagnosis is always easy to make, with attention to the peculiarities given above. It is more difficult, however, to decide accurately whether the case corresponds rather to the type of an acute multiple neuritis or to the type of an acute ascending spinal paralysis. We can judge as to this point only by careful attention to the single symptoms, especially the condition of the sensibility (pains, anæsthesia), of the reflexes, and of the electrical excitability.

The prognosis must at first be made with great reserve, and we must especially bear in mind the possibility of a rapidly fatal termination. If the first acute stage pass off fortunately and there be a decided cessation in the extension of the symptoms of paralysis, the prognosis is quite favorable, for we may then expect that the patient will be completely restored.

Treatment.—We can not be certain whether an energetic "derivative treatment" is of advantage in the beginning of the disease. Dry cups along the vertebral column are recommended, and even the use of the hot iron to the back. We would hardly advise the latter. It may be advisable, however, to prescribe inunctions with mercurial ointment, thirty to forty-five grains a day, as in antisyphilitic treatment. Of internal medicines, we should try at the beginning salicylic acid or salicylate of sodium in large doses (30 grains, grammes 2, of salicylate of sodium three times a day), and also antipyrine. We may also try extract of ergot (ergotine) and perhaps iodide of potassium. It also seems to be a good plan to begin galvanic treatment early, applied to the spine and peripherally. If alarming attacks of respiratory insufficiency occur, electrical excitation of the phrenic nerve and of the respiratory muscles sometimes affords relief to the patient.

If the symptoms be arrested, electrical treatment and the use of baths may do most to hasten convalescence.

CHAPTER XII

NEW GROWTHS OF THE SPINAL CORD AND OF ITS MEMBRANES

Pathological Anatomy.—Tumors of the spinal cord in the strict sense of the word are rare. The commonest primary new growth is the glioma, which arises from the neuroglia, and forms a cellular and vascular tumor. This is situated most frequently in the cervical or upper dorsal cord. It often develops at the beginning in the posterior half of the cord, but later it may attain a considerable length and a transverse diameter of several centimetres, so that the spinal cord in the part affected may have a considerable tumor-like thickening, or it may even be changed throughout almost its whole length into a thick, shapeless mass. Inside the glioma we sometimes find secondary hæmorrhages and (myxomatous) softenings. Central gliosis is distinct from real glioma. The former never leads to any special enlargement of the cord (tumor formation), but to a gliomatous degeneration, and especially to an abnormal cavity formation in the cord, particularly in a longitudinal direction. We will speak of this important and by no means very rare disease more fully in a subsequent chapter.

Of other rare new growths in the spinal cord we may mention solitary tubercles, syphilomata, and sarcomata. Solitary tubercle usually begins in the central gray matter. Cysticerci have also been repeatedly observed in the spinal cord.

In the spinal meninges have been found sarcomata, fibromata, lipomata, myxomata, and syphilomata. Sarcoma of the dura mater is the commonest and practically the most important of these growths. It almost always seems to start from the endothelium on the inner surface of the dura, and it is then sometimes called endothelioma. Sarcoma of the dura has a great tendency to spread as a flat growth, so that it may finally surround a considerable portion of the cord with its mass of growth. Sarcoma is often multiple; thus it may co-exist on the spinal dura mater and at the base of the brain, etc. As a rule, it does not invade the pia mater or the substance of the cord itself. Lipoma develops on the outer surface of the dura, starting from the fatty tissue between the vertebral canal and the dural sac. A carcinoma arising from the vertebræ may also reach the spinal meninges by direct invasion.

We know practically nothing of the ætiology of most new growths in the spinal cord. In all probability there is an abnormal congenital predisposition of the tissues, which furnishes the germ for the subsequent development of the new growth. It is worthy of note that the onset of the first symptoms is very often preceded by an injury (fall on the back, etc.), which thus seems sometimes to give the first impetus to the formation of a tumor.

Symptoms and Course.—New growths in the spinal meninges, especially sarcoma of the dura mater, are apt to cause at the beginning pronounced root symptoms, and usually at first symptoms of irritation of the posterior roots. The first symptoms of the disease are therefore radiating pains, either persistent and burning or paroxysmal and neuralgic, and paræsthesiæ in certain root territories. There may also be local pain, a feeling of stiffness and pressure in the back, etc. There is rarely any characteristic tenderness over the spine. Anæsthesia and paralysis may also arise as pure root-symptoms, but only when the disease is extensive, since, as was mentioned above (page 921), the complete division of one sensory or motor root does not cause complete anæsthesia or paralysis in any region. Extensive paraplegic symptoms therefore point usually to an implication of the cord. The cord itself also suffers, as we have described more fully in the account of pressure paralysis from tuberculosis of the vertebræ (page 935). Only exceptionally does the growth itself invade the cord. The symptoms of spinal compression are exactly the same as in the other transverse diseases of the cord. The differential diagnosis between meningeal tumors and compression of the cord due to vertebral caries is therefore often very difficult, if we can detect no changes in the spine itself (Pott's boss). We must then pay chief attention to the general symptoms of tuberculosis (heredity, habitus, fever, other tubercular affections). If a meningeal sarcoma occurs as a metastasis or as one manifestation of multiple sarcomatosis, the discovery of a sarcoma in some other place (such as the skin) may put us on the right track in our diagnosis.

In glioma of the cord itself the initial symptoms of sensory irritation may be almost wholly absent, or at least they may be only slight. A complicated type of spinal disease gradually develops, in which all the spinal symptoms may occur in the most varied combinations. The disease sometimes exhibits from the beginning the ordinary type of a transverse spinal paralysis. In such cases it is hardly possible to distinguish between a tumor and chronic myelitis. In other cases, however, certain peculiarities in the type lead us to suspect the possibility of a tumor. Among them especially is the early asymmetry of the symptoms on the two sides. Since a tumor may at first be confined to one half of the spinal cord (which scarcely ever happens in myelitis), the signs of a unilateral lesion of the spinal cord (*vide infra*, Chapter XVII) are often observed in tumors in a more or less pronounced fashion. Only later in the course of the disease does the clinical type of unilateral lesion gradually change to the ordinary transverse paralysis. A certain alternation in the symptoms, improvements, and new and quite sudden changes for the worse, are also noticed at times with tumors, a condition which is probably to be referred to changes in the fullness of the vessels, or to hæmorrhages in the substance of the tumor. The manner in which the disease progresses is also to a certain degree characteristic. If we find on repeated examinations evident indications that the spinal affection is slowly extending upward, especially if the cutaneous anæsthesia is moving upward and the paralysis is invading higher muscular territories, this condition is decidedly in favor of the diagnosis of tumor, since it is far less frequently observed in other forms of disease. We may also consider in diagnosis the fact that glioma often develops in the cervical cord, or at least gradually invades the cervical cord; consequently sensory disturbances and paralysis occur in the upper extremities in addition to the paraplegia of the legs. This is of rare occurrence in chronic myelitis, which is usually situated in the dorsal cord. The diagnosis of a tumor of the spinal cord, however, can at best be merely probable.

The accurate determination of the seat of the tumor, the "segmental diagnosis," is of great practical significance, if there is any question of operative treatment. It is evident that only new growths of the spinal meninges can be the subject of surgical interference. Such new growths have in a number of cases of late been operated upon successfully, but of course the number of cases in which a correct diagnosis can be made and an operation can be successfully performed is limited. We need not repeat here the details of a segmental diagnosis; they follow the general rules which hold for all lesions of the spinal cord (page 920); but we must once again remind the reader that total anæsthesia of a given cutaneous territory means not only an involvement of the corresponding posterior root as given in the diagram, but also an affection of the neighboring roots, especially the two roots above. After determining the upper boundary of the anæsthesia we should therefore always suspect that the upper boundary of the lesion in the cord is one or two segments higher than would correspond to the diagram, because the individual cutaneous territories are always in relation with several spinal roots. The same rule also holds when the segmental diagnosis is based upon the motor symptoms. The disregard of this relation, which was first clearly established by Sherrington, is the reason why formerly the location was usually put too low, even when the tumor itself had been correctly diagnosticated. In determining the upper boundary of the tumor, furthermore, we should consider not only the anæsthesia, but also the symptoms of sensory irritation. These very often manifest themselves above the anæsthetic zone and then point with considerable certainty to an irritation of the corresponding posterior roots by the upward growth of the tumor. The accurate determination of the position of the different spinal segments and roots in relation to the bodies and spinous processes of the vertebræ is also of great practical importance. In regard to this, we will refer to Fig. 125, page 928.

A tumor of the spinal cord may last for many years. In new growths of the meninges the symptoms of sensory irritation alone may continue for several years before there are any well-developed transverse symptoms. Other cases, of course, run a much more rapid course. The prognosis of tumors of the cord is wholly unfavorable unless there is a possibility of operative treatment; but, as we have said above, this presents itself in but few cases. Otherwise the treatment is merely symptomatic. If there is a suspicion of previous syphilis we should necessarily try inunction and the exhibition of iodide of potassium. In sarcoma arsenic in large doses is perhaps of some service.

CHAPTER XIII

THE SYPHILITIC AFFECTIONS OF THE SPINAL CORD

Ætiology and Pathology.—The affections of the spinal cord provoked by syphilis must be divided into two principal groups. The first group is formed by diseases in which a slow degeneration of certain systems of neurones develops in a somewhat obscure fashion under the influence of previous syphilitic infection (probably from the action of some toxine). These diseases are called most properly "*metasyphilitic*" (so-called metasyphilis). Chief among them, as we have already seen, is tabes dorsalis (the metasyphilitic degeneration of the posterior root neurones), and also, probably, certain rare cases of degeneration of the lateral columns, and of combined systemic disease in the lateral and posterior columns (PyS, KlS, and the columns of Goll). The second group of syphilitic diseases is to be classed as genuine tertiary syphilis in distinction from metasyphilis. These affections are characterized by the development of a specific gummatous new growth in the cord, especially in the membranes (*meningitis gummosa*) and in the blood-vessels (*endarteritis syphilitica*). This is usually quite extensive and not infrequently associated with corresponding syphilitic disease in the brain. Since metasyphilis of the cord has already been discussed in another part of this work, there remains to be considered here only the specific syphilitic (gummatous) affections of the cord.

Indications of syphilis of the cord may appear in six months to a year after the infection; but they develop more frequently in from two to five years after the infection, or even later. In only a few cases does a circumscribed gumma form, which then causes the symptoms of an intramedullary tumor. Diffuse gummatous meningitis is far more common; this almost always arises from the spinal pia mater, and only exceptionally from the dura mater (page 917). In such cases the spinal cord seems surrounded, in an irregular fashion, but to a considerable extent, by a newly-formed, vascular granulation tissue several millimetres thick. The meningeal blood-vessels are also almost always much diseased, their walls are thickened by endarteritic new formations, and their lumen obliterated or filled with thrombi. The posterior border of the cord is usually most affected. The disease usually has a considerable longitudinal extent. The region of the dorsal cord is often most affected, but in other cases it may be the cervical or lumbar cord, or sometimes the region of the conus medullaris and the cauda equina. The spinal roots especially are often surrounded by a thick sheath of newly-formed gummatous tissue. Rootlets of newly-formed tissue may extend from the pia mater into the substance of the cord, or the syphilitic new growth may extend to the vessels of the cord itself; in such cases secondary tissue-changes (destruction of nerve-tissue, proliferation of neuroglia) develop as a result of the defective blood-supply of the tissue. In many cases certain secondary sequelæ also develop in the tissues of the cord, similar to those we have become acquainted with in compression of the cord from tubercular new formations on the dura mater (*vide* page 936). It is also worthy of mention that in a few cases genuine gummatous disease may apparently be associated with metasyphilitic degeneration.

Symptoms and Course.—It is evident that syphilis of the spinal cord can not afford any single definite type of disease on account of the multiple localization of the anatomical changes. We may say rather that syphilis of the spinal cord is characterized by the irregularity and the often unusual combination of its symptoms, and its changeable and varying course. From the different combinations of symptoms that occur we may best distinguish, relatively speaking, the following two forms of the affection:

1. Chronic Gummatous Spinal Meningitis and Meningo-myelitis.—The disease at first shows definite root symptoms, especially symptoms of irritation of the posterior roots: paræsthesia, hyperæsthesia, shooting pains, neuralgia. Associated with these we sometimes find well-defined local pain in the back or in the sacral region, pain on pressure and on movement of the vertebral column. Motor symptoms appear somewhat later as a rule, sometimes motor-root symptoms (atrophic paralyses in definite muscular territories, for example, in the muscles of the forearm and hand on one side or in the sacral distribution, etc.); or more frequently motor symptoms from implication of the cord itself; spastic paretic

symptoms in one or both legs, sometimes, in disease of the cervical cord, in both legs and one arm. Associated therewith are various, but usually slight, disturbances of sensibility, vesical disturbances, etc. If the region of the posterior columns and posterior roots be chiefly affected, affections resembling tabes may develop (*syphilitic pseudo-tabes* of Oppenheim). Not very rarely unilateral disease may also present the more or less clearly-developed picture of Brown-Séquard's unilateral lesion. It is of course impossible to describe exhaustively all the combinations of symptoms that may be observed.

The whole course of the disease is usually chronic and, as has been said, very variable. In mild cases we may sometimes see almost complete recovery under appropriate treatment, but this is unfortunately a rare event, since the secondary tissue necroses, the results of compression, obliteration of vessels, etc., are incapable of complete restoration. We see improvement quite frequently, especially with timely treatment, which finally passes into a stationary condition. We must be prepared for relapses, fresh exacerbations, and the onset of new symptoms, which may also be cerebral. The disease may last for many—even twenty—years. The affection becomes directly fatal through the occurrence of dangerous complications (pyelo-cystitis) or severe attendant symptoms (apoplexy), etc.

2. SYPHILITIC MYELITIS AND SYPHILITIC SPASTIC SPINAL PARALYSIS.—In many cases syphilis of the spinal cord exhibits from the outset the symptoms of a myelitic affection. This form probably always arises from the vessels of the cord. The syphilitic endarteritis of the smaller vessels leads to a rapid or slow necrosis of tissue and the symptoms of spinal disease which necessarily result therefrom. The disease often develops as rapidly as an acute myelitis. A more or less complete paraplegia comes on within a few days. Only on careful inquiry do we sometimes learn that it has been preceded for some time by mild precursory symptoms (weakness, paræsthesiæ, etc.). In other cases the paraplegia develops more slowly, but yet within a few weeks or months. Since the anatomical lesion is very often in the dorsal cord, the paraplegia far most frequently shows the signs of spastic paralysis. Erb has therefore grouped certain cases under the name of *syphilitic spastic spinal paralysis.* This form of spinal syphilis, which in our experience is in fact very characteristic, is found especially in young persons of the male sex. Weakness, stiffness, and often a lively tremor (ankle clonus!) occur first in one leg and soon after in the other. The gait constantly grows more laborious, slower, and decidedly spastic-paretic. The sensory disturbances are at first very slight; and there is no pain. Later, on careful examination slight changes in sensibility are, of course, often to be found. Disturbances of the bladder are also rarely very prominent, but they are often plainly existent in a slight degree from the beginning. The condition may improve, especially with timely treatment, but, for reasons already mentioned, a morbid condition may often remain stationary for years, or new symptoms even may ensue.

Diagnosis.—So far as any diagnosis of syphilis of the spinal cord is possible, it is evident from what has been said. The chief rule is to consider the possibility of syphilitic disease in all spinal affections that are atypical from the beginning. If a previous infection is certain, or if, at least, it is rendered probable by the special antecedents, the diagnosis is the more warranted. Of course a previous infection often enough can not be established, even when there is certainly syphilis of the cord, and, on the other hand, other forms of spinal disease may, of course, occur in those who have been previously infected. The most characteristic features of syphilis of the cord have been mentioned above. Similar types of disease may be produced by multiple sclerosis, by sarcomatosis of the spinal meninges, by tuberculosis of the vertebræ, etc. We must therefore consider these affections chiefly in the differential diagnosis. Sometimes the success or failure of specific treatment may be of significance in diagnosis.

Treatment.—In every case, even where there is merely a suspicion or a possibility of syphilitic disease of the spinal cord, we should institute at once a thorough antisyphilitic treatment. We think the best treatment is inunction with mercurial ointment (grains xlv–lxxv, grammes 3–5, daily). Other physicians recommend subcutaneous injections of corrosive sublimate, salicylate of mercury, etc. The inunction is almost always combined with the internal exhibition of iodine preparations, especially iodide of potassium (at first grains xx–xxx, grammes 1.5–2, and later still larger doses daily), iodide of sodium, or one of the newer iodine preparations, of which iodalbacid seems to us the best. [It is often necessary to give iodide of potassium in much larger doses. If the diagnosis of syphilis be probable, it is advisable to begin with doses of half a drachm (grammes 2) or more three times a day, which should be increased rapidly until the patient is taking five or six drachms daily (grammes 20–25), and sometimes, as in cerebral syphilis, it may be necessary to push the dose to one or two ounces a day (grammes 30–60).—K.] If circumstances permit, it is well to combine inunction with a course at some bath (sulphur baths in Aix, Nenndorf, also Tölz, Hall in Austria, Wiesbaden, etc.). Every antisyphilitic treatment must be kept up a sufficient length of time and repeated after certain intervals, as is the general custom in the treatment of syphilis. Besides the specific treatment, the other therapeutic methods which are efficacious in chronic spinal affections also have their appropriate use (warm baths, mineral baths, galvanization). Individual symptoms (neuralgic pains, vesical troubles) require special attention.

The properly conducted treatment of spinal syphilis may give results which are sometimes excellent and often at least satisfactory. We can understand from what has been said why our hopes are naturally only too often disappointed.

CHAPTER XIV

SYRINGOMYELIA AND HYDROMYELUS

Pathological Anatomy and Pathogenesis.—The formation of abnormal cavities in the spinal cord is due either to a dilatation of the central canal (hydromyelus) or to a peculiar process, which is called central gliosis, with the secondary formation of cavities (syringomyelia proper). The cases of genuine hydromyelus are recognized by the fact that the cavity is found in the middle of the cord, corresponding to the position of the central canal, and that its walls are covered by cylindrical epithelium. Slight degrees of hydromyelus, in which the dilated central canal has a diameter of a millimetre or a millimetre and a half, are not infrequently found. The dilatation usually extends over only one segment of the cord. Greater degrees of hydromyelus, where the central canal is dilated to a diameter of half a centimetre or a centimetre and the longitudinal extent is greater, are much rarer.

In regard to the origin of *hydromyelus*, we may assume, according to the teaching of Leyden, that the cause is quite generally some anomaly of development in the formation of the central canal. The hypothesis of Langhans, that processes of stasis in the central canal, such as compression of the upper end by a tumor in the posterior cranial fossa, may lead to hydromyelus, is certainly not applicable to the great majority of cases.

The conditions are different in *syringomyelia*, which is of much greater practical importance. In this affection, according to many investigators (Westphal, Simon, F. Schultze, J. Hoffmann, and others), the whole process apparently begins with a gliomatous new formation or transformation of tissue in the central portions of the spinal cord. This peculiar process, which probably starts in the ependymal cells about the central canal, is called central gliosis. It consists of a replacement of nerve-tissue by fibrous glia-tissue. The central gliosis, which extends toward either the posterior or the anterior horns, has a decided tendency to break down and thus to form secondary cavities, which unite with the central canal or remain independent, and these cavities may extend longitudinally in the central portions of the cord with numerous ramifications and projections. To this secondary formation of cavities we give the name of syringomyelia. In this condition the central canal itself hardly ever remains normal; it is usually dilated and shows many projections, reduplications, and the like, circumstances which clearly indicate that we must consider disturbances of development as factors in the origin of syringomyelia and central gliosis. We do not find any special enlargement of the cord in the nature of a tumor. Gliosis is thus distinct from true glioma formation (gliomatosis), but in some cases the distinction between gliosis and the formation of cellular glioma is ill-defined. In ordinary gliosis with syringomyelia, however, the cord appears normal externally, or, when the syringomyelia is extensive, it may appear flabbier, saccular, and flattened in the parts affected. Only on section do we recognize, even with the naked eye, the cavity formation in the center or to one side in the gray matter and extending into the horns of the cord. Under the microscope we find about the cavity the newly-formed tissue, still proliferating peripherally and disintegrating toward the edge of the cavity. This tissue contains many newly-formed ependymal cells in its central portion; toward the periphery the cells are fewer, and the tissue consists chiefly of a mesh-work of fine glia-fibers. We can not say definitely to what circumstances we may refer the destruction of tissue and the cavity formation. Changes in the vessels, which are often found dilated, may perhaps play some part (hæmorrhage, thrombosis, etc.). In most cases central gliosis and syringomyelia begin in the cervical cord and there attain their greatest extent; but they may extend downward in a lesser degree into the lumbar cord and upward into the medulla oblongata.

We must probably always look for the final cause of gliosis and syringomyelia in a defective congenital constitution of the spinal cord. According to F. Schultze, injuries at birth in cases of difficult labor may perhaps lead to hæmatomyelia and afford a basis for the later anomaly. Injury in later life is also frequently accused of producing the trouble.

Clinical Symptoms.—Slight degrees of hydromyelus or of syringomyelia may be met with at autopsies as accidental findings which have given rise to no clinical symptoms whatever. Other cases of extensive cavity formation give rise to various complex forms of spinal disease, which usually escape correct interpretation during life. Thus we recall, for example, Späth-Schüppel's well-known case of general anæsthesia, a case under our own observation which presented the apparently pure type of spastic spinal paralysis, etc.

Opposed to such isolated cases, however, the great majority of cases of central gliosis and syringomyelia of the cervical cord present an unusually characteristic picture, so that we can often make a diagnosis with absolute certainty. Since the investigations of F. Schultze and Kahler we know that this type is by no means rare. It is therefore certain that it was often seen in the past, but it was usually falsely interpreted. The fact that the gliosis begins as a rule in the center of the cervical cord and that, as it progresses, it must always involve at first the gray matter of the posterior and anterior horns on one or both sides, is the reason why the group of symptoms always occurs in a way that is generally very characteristic, although it may show some variations. It is seen especially in men in youth and middle age (between twenty and forty).

The clinical symptoms of syringomyelia of the cervical cord develop very gradually, and almost always begin in the upper extremities—one arm usually being affected before the other. The symptoms observed in such cases may best be classed in three groups: 1. Motor weakness and muscular atrophy. These symptoms are dependent upon the involvement of the anterior horns in the process. The atrophy affects with especial preference the small muscles of the hand (interossei, thenar muscles), and also the muscles of the forearm, the deltoid, etc. Fibrillary twitchings and electrical reaction of degeneration are often found. The tendon reflexes are lost or else exaggerated, which may be easily explained from the special localization of the process. 2. Disturbances of sensibility are almost invariably associated with this muscular atrophy. The chief cause of these is always the involvement of the posterior horns in the process. The disturbances of sensibility almost always have this peculiarity, that the sensibility for pain and the temperature sense are at first always affected, while the simple sensibility for touch and the pressure sense may remain normal for a long time or throughout the disease. The fact that the patient is insensible to the influence of pain or heat is therefore of special significance, because it may frequently lead to wounds, burns, etc., on the hands and forearms, which the patient may often never notice, and which therefore may heal badly. The disturbances of the temperature sense may affect either the sensibility to cold or the sensibility to heat, or both at once. 3. The third group of symptoms is made up of peculiar trophic changes. The above-mentioned external injuries, caused by the analgesia, naturally play a large part in this connection (felons, bullæ, phlegmon, etc.);* but we must also assume special trophic disturbances to explain many processes, especially the peculiar thickening of the bones sometimes observed, the atrophy of the fingers, etc. In many patients there may thus develop a very peculiar and characteristic deformity of one or both hands, of which the accompanying cut (Fig. 161) gives a good idea. The hand and fingers are thickened, and some fingers are shortened by atrophy of the phalanges. The most striking abnormal vaso-motor and secretory disturbances may also occur.

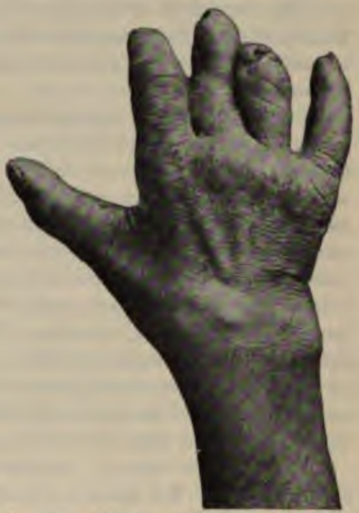

FIG. 161.—Syringomyelia of the cervical cord. Deformity of the hand, atrophy of the interossei and of the adductor pollicis. (Personal observation.)

In many cases the symptoms remain confined almost entirely to the upper extremities. They progress so slowly that many patients are able to do a certain amount of work for a long time; but finally changes may also appear in the legs, either simple paresis and spastic conditions or symptoms similar to those in the arms already described. These arise only when the process involves the gray matter of the lumbar cord. On the other hand, the disease may extend upward from the cervical cord, and then very characteristic bulbar symptoms may ensue. The commonest bulbar disturbances are: partial loss of sensation in the region of the trigeminus, unilateral paralysis of the hypoglossus with unilateral atrophy of the tongue, paralysis of one vocal cord, unilateral paralysis of the accessory (weakness and atrophy of one trapezius and sterno-cleido-mastoid), paralysis of the soft palate, facial paralysis, abducens paralysis, etc. In many cases such bulbar symptoms are much more prominent than the spinal symptoms. Abnormalities in the pupils and the fissura palpebrarum are seen in a few cases, even where the gliosis is limited to the cervical cord, and are to be explained by an involvement of the so-called cilio-spinal center.

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* The so-called *Morvan's disease* is nothing but syringomyelia, in which these trophic and inflammatory changes are especially marked. [The large number of cases coming under Morvan's observation in a small district in Brittany suggest, however, that Morvan's disease may be rather an infectious neuritis, possibly due to leprosy, and therefore distinct from a syringomyelia with marked trophic disturbances.—K.]

It is a remarkable fact that syringomyelia is very often associated with a more or less pronounced kyphoscoliosis of the spine. We are of the opinion that the kyphoscoliosis is not due to the syringomyelia, but that it is also an expression of abnormal conditions of development and growth in the vicinity of the medullary tube.

The course of syringomyelia is very chronic, yet always unfavorable. Death finally ensues from some intercurrent disease, general weakness, pyelo-nephritis, or, as we have seen, from amyloid disease of the kidneys due to many suppurative processes.

The diagnosis of typical cases of syringomyelia of the cervical cord is not difficult if we once know the type. A careful examination of the sensibility prevents us from mistaking it for spinal muscular atrophy. Any combination of muscular atrophy in the hands with analgesia and disturbances of the temperature sense must awaken the suspicion of syringomyelia, as must all unusual trophic disturbances occurring in the hands, etc. If the three groups of symptoms mentioned above co-exist, we can be almost sure of our diagnosis. Only exceptionally can other processes in the cervical cord (such as compression following caries, as we have ourselves seen) produce a similar picture and give rise to confusion. The differential diagnosis between syringomyelia and leprosy may be more difficult, as the latter sometimes gives rise to a very similar type of disease; but in such cases the special endemic conditions, the peculiar changes in the skin, and finally the discovery of the specific bacillus, may enable us to make a correct decision.

There is no special treatment for syringomyelia. The treatment must be symptomatic. We should remember, when there is analgesia, to warn the patient of the possibility of injury.

CHAPTER XV

SPINA BIFIDA

(Hydrorrhachis. Myelocele. Meningocele)

We give the name of spina bifida to a congenital fissure-formation on the posterior side of the vertebral arches, due to anomalies of development, and associated with a hernia-like protrusion of the sac of the dura. The most frequent seat of the malformation is in the sacral and lumbar regions. Only rarely is the tumor so great as to hinder the birth of the child. Children afflicted with spina bifida are usually born normally, and not till afterwards do we find the tumor in the sacral region; its size may be from that of a small nut to that of the fist or larger. The skin above the tumor is sometimes entirely normal, but in other cases very tense and reddened. If we have an opportunity to examine the tumor carefully post mortem, we usually find beneath the skin the protruded sac of the dura, and beneath it the arachnoid. Only rarely is the dura also fissured, so that the sac is formed exclusively of the arachnoid. It is filled with a clear fluid which is precisely identical with the cerebro-spinal fluid. In rare cases there is also a dilatation of the central canal, hydromyelus; then the substance of the cord is atrophied to a greater or less extent, and the central canal communicates directly with the cavity of the spina bifida. In other cases the condition of the cord is normal; sometimes its lower end is adherent to one spot of the sac. We must refer to the text-books of pathological anatomy in regard to the many further details of the anatomy and the history of development.

In regard to the clinical symptoms of spina bifida, the condition of most children at first is perfectly normal, apart from the malformation. The tumor itself usually feels tense. If we exert pressure on it with the hand, we can often force part of its contents back into the vertebral canal. This causes an increase of the cerebral pressure, and we notice, besides the lessening of the spina bifida, a marked expansion of the fontanelles, and also the appearance of somnolence, contractions, and changes in the pulse and respiration, which demand a speedy interruption of this rather dangerous experiment. If such symptoms do not appear at all, we can conclude that the sac is completely constricted and closed.

Only rarely, however, does the child's condition remain normal. The tumor usually shows a slow growth, and the results of pressure on the spinal cord or on the cauda equina gradually appear. Paralysis, anæsthesia, vesical disturbances, bed-sores, etc., develop, and these symptoms finally lead to death. Still more frequently the sac bursts, or its walls inflame, and this becomes fatal from the onset of a purulent meningitis.

The prognosis of most cases of spina bifida is accordingly to be regarded as unfavorable, unless we succeed in curing the disease by surgical treatment. Recovery has been brought about in many cases by methodical compression of the sac, and by puncture, with evacuation of the fluid and a subsequent injection of a solution of iodine to obtain an obliteration of the sac; but, on the other hand, the operative treatment of spina bifida is attended with many dangers, including liability to meningitis, so that we note frequent bad results as well as the favorable ones. We can not here go into the details of the surgical methods for the cure of spina bifida; they can be found in full in the text-books of surgery.

CHAPTER XVI

SECONDARY DEGENERATIONS IN THE SPINAL CORD

ALTHOUGH the secondary degenerations in the spinal cord are chiefly interesting merely from an anatomical point of view, we must briefly describe them, because, in the first place, a clinical significance has been ascribed to them in certain quarters, and also because the study of secondary degeneration has been the starting-point of all our present knowledge as to the systemic diseases of the spinal cord.

1. *Secondary Degeneration in the Spinal Cord after Cerebral Lesions.*—We already know (compare page 834) that every lesion of the great motor ganglion-cells in the anterior cornua of the spinal cord, and every permanent break in conduction in the motor nerves themselves, is followed by a secondary degeneration of the peripheral portion of the motor fibers. We assume as the reason for this, as we have seen, a "trophic influence" of the said ganglion-cells on the motor fibers arising from them, so that the latter degenerate when the conduction of that trophic influence is interrupted, or when the trophic ganglion-cells them-

selves are destroyed. Precisely analogous conditions exist for the first great portion of the motor conducting tract, the lateral pyramidal tract, from the cortex cerebri to the anterior horns of the spinal cord (*vide* Fig. 137, page 986). The great ganglion-cells of the motor portion of the cortex cerebri also exert a trophic influence on the motor fibers arising from them, which extends to the end of these fibers—that is, up to but exclusive of large motor ganglion-cells of the spinal cord. This atrophic influence may be understood if we regard the ganglion-cell and its corresponding nerve-fiber as forming one complete unit and properly only a single cell, a *neurone*, and that therefore the process of the cell body (the "nerve-fiber") can not possibly lead an independent existence when separated from the "ganglion-cell"—that is, from the nucleus of the cell body. If therefore there be disease situated in the motor portion of the cortex cerebri itself, or in any part of the motor tract in the brain (the motor fibers of the corona radiata, the internal capsule, the crus, or the pons), by which disease the conduction is interrupted—if there be disease there, we repeat, a secondary descending degeneration of the motor fibers ensues in the whole portion below, down to, but exclusive of, the anterior horns of the gray matter. The secondary descending degeneration of the pyramidal tract is found correspondingly in the crus, in the pons, and in the pyramid of the same side on which the focus of disease in the brain is situated. From this point we can trace the main part of the degeneration below the decussation of the pyramids farther down the lateral column of the spinal cord on the opposite side (secondary degeneration of the lateral crossed pyramidal tract) (see Fig. 162), while in many cases besides we find a slighter secondary degeneration in the anterior column of the spinal cord on the same side (secondary degeneration of the anterior uncrossed pyramidal tract). As we know from Flechsig's investigations, the relative amounts of the crossed lateral fibers, and the anterior fibers that remain uncrossed, vary in individual cases within certain limits. In the cases where no anterior pyramidal tract exists—that is, where all the motor fibers pass over to the lateral column of the opposite half of the spinal cord in the decussation of the pyramids—of course a descending degeneration in the anterior column is wholly wanting. We must add, however, that in some cases a small number of fibers seem to proceed uncrossed in the lateral column, so that accordingly we may also have a slight secondary descending degeneration in the lateral pyramidal tract of the same (affected) side (Pitres).

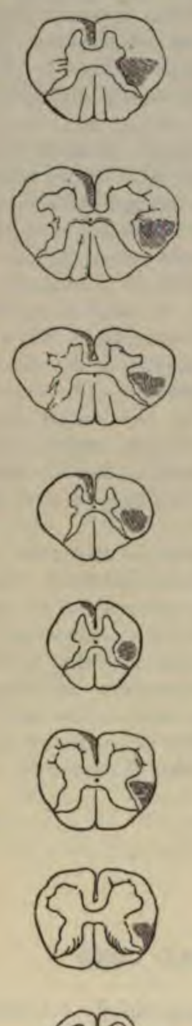

FIG. 162.—Secondary descending degeneration of the pyramidal tracts in a primary lesion of the left half of the cerebrum. The lateral pyramidal tract of the right half of the cord is degenerated down to the lowest part of the lumbar region (1–8); the anterior pyramidal tract of the left half of the cord is degenerated to the beginning of the lumbar enlargement (1–6).

2. *Secondary Degeneration in the Spinal Cord in Transverse Affections of the Spinal Cord itself.*—If a lesion be situated in any part of the spinal cord, by which more or less of its transverse section is affected, the interruption of conduction in these fibers is also followed by the appearance of secondary degenerations which may be made out both in a descending and in an ascending direc-

tion (see Fig. 163). It is most frequently transverse myelitis, compression of the spinal cord, and tumors of the cord, which give rise to secondary degenerations. The latter, however, of course, are never due to the sort of lesion, but only to its seat, and to the interruption of conduction caused by it.

The secondary descending degeneration affects the pyramidal tract in a fashion precisely analogous to that which we have also learned to recognize in secondary degenerations after cerebral lesions; but since the primary affection usually affects the pyramidal tract on both sides, the descending secondary degeneration of course develops in both lateral pyramidal tracts, and also in the anterior pyramidal tracts, if they exist below the point of lesion. In general the area of the descending degeneration of the pyramidal tract is larger in transverse diseases of the spinal cord than it is in lesions of the brain. The principal reason of this is that lesions of the brain hardly ever destroy completely all the motor fibers belonging to one half of the body. Furthermore, in a transverse lesion of the cord certain other small systems of fibers of uncertain significance also degenerate downward: among these are a small zone of fibers lying internally to the lateral cerebellar tract and Gowers's tract (*vide infra*) and extending to the anterior border of the cord near the anterior pyramidal tract (Löwenthal's "*intermedian fasciculus of the antero-lateral column*"), and also, very constantly, a small comma-shaped fasciculus in the middle of each posterior column, formed probably of descending collaterals of the fibers of the posterior columns (the so-called Schultze's comma, Fig. 163), which we can not usually trace very far downward.

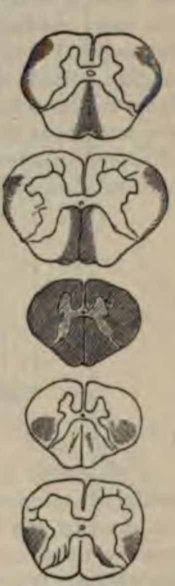

FIG. 163.—Secondary ascending and descending degeneration in a transverse affection of the upper dorsal region. The columns of Goll and the direct cerebellar tracts are degenerated upward. The lateral pyramidal tracts are degenerated downward.

The secondary ascending degeneration, developing upward from the primary point of disease, affects several systems of fibers which conduct centripetally, the so-called columns of Goll (the internal portion of the posterior columns), and also two systems of fibers in the outer border of the lateral columns. The degeneration of the posterior columns is most extensive immediately above the point of interruption and gradually decreases upward (Fig. 163). We can readily understand this degeneration if we remember that the fibers of the posterior columns are the ascending fibers of the posterior roots, which have their origin in the cells of the spinal ganglia. Since the new fibers from the posterior roots which enter higher up are always external to the fibers coming from the lower roots, the degeneration must be limited more and more, as it ascends completely, to the region of the columns of Goll. A secondary ascending degeneration in the posterior columns also follows division of the posterior roots, and therefore, of course, injuries of the cauda equina. The secondary ascending degeneration in the lateral columns is made up of a smaller posterior and a broader anterior portion. The former is the lateral cerebellar tract discovered by Flechsig; its fibers arise from the cells of Clarke's columns in the lower dorsal cord, and therefore they must degenerate when separated from these cells. The fibers may be traced upward into the corpus restiforme, and then to the cerebellum. The bundle of fibers anterior to the lateral cerebellar tract, which also degenerate upward, is distinct from the lateral cerebellar tract in its development and its origin. It probably arises from cells of the posterior horns, and is called Gowers' tract or the antero-lateral ascending tract. Its termination and significance are still unknown.

Although no clinical significance at all can be attributed to secondary ascending degeneration, the theory first advanced by the French observers (Charcot and others) prevails almost universally, that secondary descending degeneration causes definite clinical symptoms. The secondary contractures and the increase of the tendon reflexes in the paralyzed limbs, occurring in hemiplegia, are especially referred to this. We shall see in a later section that this theory is by no means proved, and is even improbable, so that, in our opinion, the secondary descending degeneration also has no material clinical significance.

3. *Secondary Degeneration in the Spinal Cord after Amputations.*—If we examine the spinal cord of persons who have had an arm or a leg amputated some time before their death, we sometimes find that the entire corresponding half of the cord is smaller; but the diminution of the posterior column on that side is the most marked. On careful microscopic examination we can detect a slight but evident loss of fibers in the region of the column of Goll—that is, in the median portion of the posterior columns. We can also discover a slighter loss of fibers in the column of Goll on the other side (opposite to the amputation). We can not enter into a more detailed description here of this condition, which is of interest theoretically, but which need not be considered practically and which, furthermore, is in many respects not fully explained.

CHAPTER XVII

UNILATERAL LESION OF THE SPINAL CORD

(Brown-Séquard's Spinal Paralysis)

UNILATERAL lesion is not a definite disease of the spinal cord, but a peculiar group of symptoms, which occurs whenever an interruption of conduction is produced by any affection in one lateral half of the spinal cord. Since the symptoms in these cases were first carefully studied clinically and experimentally by Brown-Séquard, we often call the type of disease in question "Brown-Séquard's paralysis." We see this paralysis in its purest form in injuries of the spinal cord. Almost perfectly exact sections of one lateral half of the spinal cord have been produced by stabs from a knife, a sword, etc. Tumors and syphilitic affections especially, and more rarely central gliosis, multiple sclerosis, hæmatomyelia, compression of the cord in caries or cancer of the vertebræ, etc., may also, during a certain period of their course, cause the symptoms of a more or less sharply defined unilateral lesion.

The peculiar condition of the symptoms in unilateral lesion is easily explained by a consideration of the course of the fibers in the spinal cord. In the accompanying diagram (see Fig. 164) the motor fibers from the anterior roots are marked *v*, the sensory fibers from the posterior roots, *h*. As is well known, the sensory fibers, *h*, pass at once into the opposite half of the spinal cord, and accordingly decussate with the corresponding sensory fibers of the other side.* The motor fibers, *v*, however, pass upward uncrossed on the side on which they enter the spinal cord, and mainly in the lateral column. If now, for example, there be situated on the right half of the spinal cord at *a* a lesion, e. g., a section of one

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[* More recent investigations by Mott render this theory of immediate decussation somewhat doubtful, and unilateral lesion may not always present the group of symptoms described below.—K.]

ıe cord, the conduction of those motor fibers which come from the right side
errupted, as well as the conduction of those sensory fibers which come from
ft side. From this it follows that there must be motor paralysis on the same
f the body as the lesion in the spinal cord, and some degree of anæsthesia
other side of the body. If the affection be situated in the dorsal or lumbar
he leg on the corresponding side is paralyzed, and the leg on the other side
sthetic. If the lesion be situated in the cervical cord, above the entrance of
rves for the upper extremities, the arm and the leg on the side of the lesion
th paralyzed (spinal hemiplegia), while the arm and the leg on the other
re anæsthetic, but possess normal mo-
but strictly unilateral disease of the
end of the cord (the sacral cord and
terminalis) may not give rise to the
e of a Brown-Séquard's unilateral
, because at this level no full decussa-
f sensory fibers has taken place. At
we find a flaccid atrophic paralysis in
g with a disturbance of sensation on
ame side, and an anæsthesia of the
m, perineum, and penis on the other
because the sensory fibers for this re-
nter the lowest part of the cord, and
ore have their decussation the lowest.
ı more careful examination, further
ions of physiological interest appear.
ensibility on the side of the motor pa-
s is usually not wholly normal. In
rst place, we sometimes find an evi-
liminution of the muscular sense (the
tion of passive movements), and in
econd place the cutaneous sensibility
always normal, but there is some-
a pronounced hyperæsthesia for all,
least for some, of the forms of irrita-
Slight pricks are very painful, and
ng the soles of the feet is felt with
mal strength. Above the hyperæs-
territory in the skin we usually find
ow anæsthetic zone (Fig. 165, *b*), and
this at times again a narrow hyper-
tic strip (see Fig. 165, *c*). The
hetic zone is easily explained. It corresponds precisely to the level
e lesion in the spinal cord—that is, to those sensory fibers coming
the same side, which are immediately involved as soon as they enter
ord; but a satisfactory explanation is entirely lacking for the appear-
of the hyperæsthesia on the paralyzed side, and for the origin of the
most narrow hyperæsthetic zone. The reflexes, especially the tendon reflexes,
sually increased on the paralyzed side. There is often a vigorous ankle
, a symptom which must be explained by the loss of the reflex inhibitory
nces coming from above. Definite statements are lacking as to the condition
cutaneous reflexes. The cremasteric and abdominal reflexes are often
t on the paralyzed side, just as in cerebral hemiplegia (*q. v.*). In some
ataxia has been observed on the side of the lesion, instead of paralysis; this
arently due to an affection of the posterior column on that side. Finally,

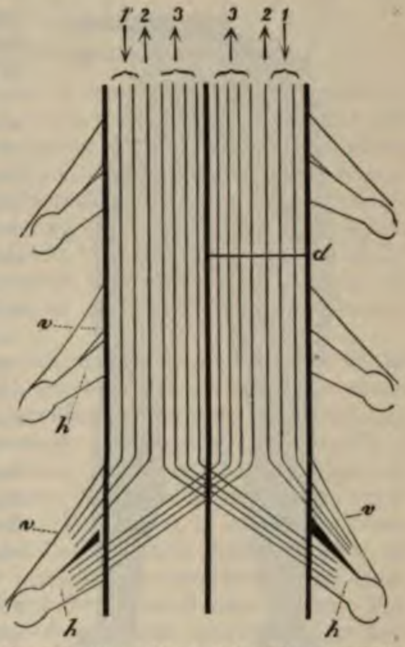

FIG. 164.—(From ERB.) Schematic representation of the course of the main tracts in the cord, represented for a single pair of roots. *v*. Anterior roots. *h*. Posterior roots. 1. Paths for motor and vaso-motor conduction. 2. Paths for muscular sense. 3. Paths for cutaneous sensibility on the right. 1', 2', 3'. The same paths on the left. The arrows indicate the direction of physiological conduction.

we often find on the side of the lesion the signs of a vaso-motor paralysis, especially a marked rise in the cutaneous temperature, even 2° (1° C.), or more.

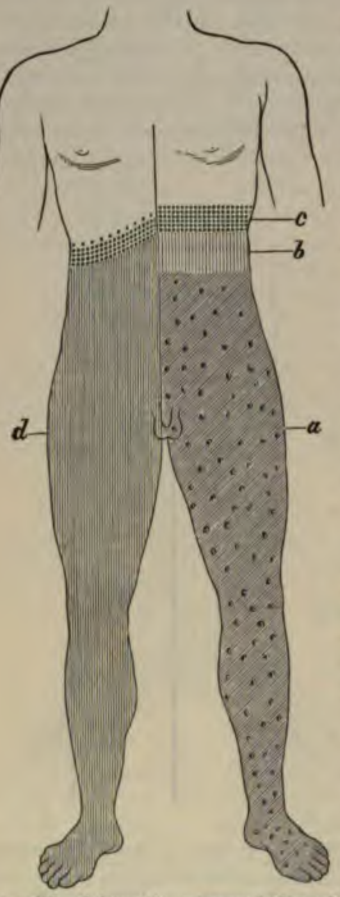

FIG. 165.—Schematic representation of the chief symptoms in unilateral lesion of the left dorsal cord. (After ERB.) The oblique shading signifies motor and vaso-motor paralysis; the vertical shading signifies cutaneous anæsthesia; the dots signify cutaneous hyperæsthesia.

On the other side, opposite the lesion, the motility is normal. The disturbance of sensibility affects the sensibility to pain and temperature chiefly, while the sensibility to touch is usually wholly or almost wholly retained. The muscular sense (the perception of passive movements) is not affected, in contrast to the paralyzed side (*vide supra*). Above the anæsthetic region we also find frequently a narrow hyperæsthetic zone (see Fig. 165, *c*). The reflexes are usually normal, or a little increased.

The statements just made in regard to the sensory disturbances agree well with the rest of our knowledge as to the sensory conducting tracts. We know that only the posterior root-fibers which enter the posterior gray matter decussate in the cord (page 799), and that these fibers serve chiefly to conduct impressions of pain and temperature (compare page 1031). The anæsthesia in unilateral lesions therefore affects chiefly these two qualities of sensibility. The muscular sensations are conducted by fibers which ascend uncrossed in the posterior columns. The disturbance of the muscular sense is therefore manifested, as has been said, upon the paralyzed side. There are apparently so many ways of conduction open for simple tactile sensations that these are only rarely much diminished.

If other spinal symptoms (disturbances of micturition and defecation, neuralgic pains, muscular atrophy, changes in the electrical excitability, etc.) are associated with the symptom-complex just described, these symptoms are dependent upon the special localization of the disease; for we must take special note that the clinical picture of a unilateral lesion may not be perfectly distinct, but we can often recognize only a few prominent features.

We need add nothing as to the prognosis and treatment of unilateral lesion, because, of course, they are governed entirely by the form of the primary disease.

IV.—THE DISEASES OF THE MEDULLA OBLONGATA

CHAPTER I

PROGRESSIVE BULBAR PARALYSIS

(*Glosso-labio-laryngeal Paralysis*)

DUCHENNE in 1860 described for the first time in complete detail the symptoms of a disease to which Wachsmuth subsequently gave the name of progressive bulbar paralysis. Duchenne did not, however, recognize the true seat of the disease, and it was not until 1870 that Charcot in France, and E. Leyden in Germany, were enabled to confirm the suggestion of Wachsmuth that the lesion is a progressive degeneration and atrophy of the nuclei in the medulla oblongata. Since then our knowledge of the disease has rapidly increased, both from the clinical and the anatomical standpoints; and Kussmaul and others have thoroughly investigated its relations to two other closely allied forms of disease—amyotrophic lateral sclerosis and progressive muscular atrophy.

Ætiology.—We have scarcely any positive information about the cause of the disease. Heredity seems in general of slight importance; but some observations indicate that the disease may also occur in families, so that we must probably assume in the last instance that the special cause of the affection is usually a congenital abnormality of the system of neurones which later become diseased. In some cases its origin is ascribed to catching cold, emotional excitement, traumatic influences, or excessive bodily exertion. Perhaps it is sometimes occasioned by excessive use of the muscles to which the disease is chiefly confined, as in playing on wind-instruments; but in many instances no possible external cause can be found. Men seem somewhat more liable to be attacked than women. The disease hardly ever appears until middle or old age—that is, after thirty-five. Only a few cases have been observed in childhood.

Clinical History.—The symptoms are almost always very slow in their development. There may be mild premonitory symptoms—such as painful sensations in the back of the neck. Then there is a very gradual appearance of difficulty in articulation. Many words are pronounced indistinctly. The first trouble is noticed especially with letters in the utterance of which the tongue plays an essential part: E, R, L, S, G (hard), K, D, T, and N.* It is easily seen that the derangement is not aphasic. There is no forgetting or confounding of the words or letters; but the innervation of the tongue has become impaired. Long before the ordinary movements of this member are visibly embarrassed, the patient has lost the ability to make those more delicate manipulations of it which are essential to normal speech. This disturbance of articulation is termed articulatory disturbance of speech (alalia or anarthria).

By the time this has become somewhat marked, it is usually possible to detect, on close examination, that the tongue is beginning to atrophy. It seems flabby, thin, and less rounded. Here and there its surface presents furrows and depressions; and often the individual fasciculi exhibit active fibrillary contractions.

[* Except as otherwise specified, the letters and words used as examples here and later on are to be given the ordinary English pronunciation.—K.]

Just as in progressive muscular atrophy, the impairment of motion usually keeps equal pace * with the atrophy. The greater the atrophy, the less is the mobility. Finally it becomes quite impossible to project the tongue from the mouth or move it from side to side. The tongue lies flat and limp on the floor of the mouth. Its surface is often diversified with furrows and depressions, containing much desquamated epithelium or the like. Evidently any great impairment of motility in the tongue hinders not only speaking, but also chewing and swallowing. The organ can no longer bring out such portions of the food as get between the cheeks and the teeth, nor can it push the bolus backward within the grasp of the pharyngeal constrictors.

Even before the atrophy of the tongue becomes extreme, analogous disturbances usually appear in neighboring groups of muscles. As a rule, the muscles of the lips are affected next after the tongue. The first thing the patient notices is a peculiar feeling of stiffness or tension in the lips. Movement becomes gradually more and more difficult; and the patient becomes unable to pucker his lips so as to whistle. Speech is also noticeably interfered with, for now all those letters the pronunciation of which demands labial movements are very imperfectly articulated, and at last they can not be uttered at all. These are O, A (long), P, F, B, M, and V; and also the sound of double O, as in tool. It also becomes gradually evident that the lips atrophy. They grow thin, with sharp edges and wrinkled skin. Fibrillary contractions are not infrequently visible.

This atrophy of the lips (orbicularis oris) is followed by atrophy and paresis of some of the other muscles of expression supplied by the lower division of the facial nerve. The general facial expression of a patient with bulbar paralysis thus comes to bear a very characteristic stamp: the mouth remains half open, and seems to be broadened, the corners of the mouth are drawn down, and the lower lip hangs down, so that the whole aspect is persistently lachrymose. Even in laughing, the lower half of the face relaxes comparatively little; while the region supplied by the upper division of the facial nerve, and the movements of the eyeball, remain as a rule perfectly normal.

The third group of muscles affected are those of the pharynx and larynx. The soft palate becomes paretic, and produces further trouble in swallowing. Quite often the liquid ingesta are regurgitated through the nose. The voice becomes nasal. The production of many sounds, and in particular of B and P, is now impossible, since, in addition to the labial paresis, a portion of the essential current of air escapes through the nostrils. This explains why the letters mentioned can sometimes be pronounced better if the nose be compressed. The paralysis of the constrictors of the pharynx impedes deglutition more and more, till the impairment of nutrition becomes extreme.

The enfeebled action of the laryngeal muscles is betrayed, in the earlier stages of the disease, by a certain weakness and monotony in speaking. Modulations of the voice, and the production of the higher notes, as in singing, are no longer possible. If the innervation of the larynx becomes still more impaired, it becomes a very serious matter. If the arytænoid cartilages do not press together firmly on swallowing, the entrance to the larynx is inadequately closed, and food is often swallowed the wrong way. Liquid and even solid ingesta get into the larynx, and excite a violent cough; or, being inhaled into the air-passages, they cause bronchitis or lobular pneumonia. The paralysis may reach such a degree

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* At the commencement of the disease the paralysis may possibly seem greater than the atrophy, so far as the latter can be detected. Nor would it be impossible for a primary lesion of the nuclei of nerves to result in a paralysis before the secondary descending degeneration had become completely developed, or for the degeneration to begin in the corresponding central neurones. On the other hand, it must be borne in mind that numerous individual fibers in the lip or tongue might be already atrophied before the eye or the touch could appreciate any change in bulk.

that the voice is at best a hoarse whisper. With the laryngoscope we can see that the vocal cords are paralyzed. The inability to close the glottis tightly is extremely unfavorable, for it renders the patient unable to cough vigorously. Mucous accumulations may therefore come to be the source of extreme dyspnœa.

The catalogue of symptoms is not yet ended. As we have seen, the muscular atrophy of the tongue and lips can invariably be detected. That of the pharyngeal and laryngeal muscles can not be demonstrated during life, although it is to be found post mortem. Inasmuch as the process is one of genuine degeneration with consequent atrophy, the affected fibers ought to give the reaction of degeneration to electricity; but this is difficult of actual proof, just as it is in progressive muscular atrophy, because numerous healthy fibers lie side by side with the degenerated ones. Still, in an advanced case, careful examination will usually bring out an evident degenerative reaction here and there in the tongue and lips.

The disturbance of reflex action is often striking. Usually the reflexes are greatly diminished or even absent, so that one can tickle the root of the tongue and the epiglottis without causing the patient to gag. In a few instances the facial muscles exhibit an increase of tendon reflex, as can be shown by tapping upon the tendons, the periosteum of the jaws, or the bridge of the nose. This behavior reminds one of the condition of the muscles in amyotrophic lateral sclerosis (*q. v.*).

Exceptionally, still other muscular groups are involved. Of such disturbances, the most frequent is in the region supplied by the motor branch of the trigeminus, impairing mastication. The impairment of these muscles now combines with the labial and lingual atrophy to render chewing almost impossible. In very rare cases the ocular muscles are also involved, with resulting ptosis and strabismus.

All the symptoms thus far enumerated are exclusively motor. Sensation is perfect to the end. The sensibility of the skin of the face and of the mucous membrane of the tongue and mouth, as well as the sense of taste, are unimpaired. Disturbances of sensation in the distribution of the trigeminus, and more or less deafness, have been reported in one or two cases; but there is some doubt about the observations. It does, however, seem certain that secretory and vaso-motor derangements are frequent. Salivation deserves especial mention. In many cases of bulbar paralysis it is a constant symptom, so that the patient is obliged to keep a pocket-handkerchief to his mouth, to catch the fluid as it dribbles away. This is due, to a certain extent, to the fact that the secreted saliva can not be swallowed, and, as the lips do not shut tightly, it naturally escapes from the mouth; but volumetric examinations have rendered it pretty certain that the amount of saliva is abnormally large. The explanation of this has not been determined. Nor as yet do we know much about the vaso-motor disturbances. Many patients complain of a feeling of heat and "boiling" in the head. We may also mention in this connection that occasionally, toward the close of the disease, the pulse becomes very rapid (140–160). This is probably due to paralysis of the vagus.

The course of the disease is invariably protracted. The order in which the symptoms appear is, as a rule, that in which they have just been described. The atrophy and paresis appear first in the tongue, then in the lips and the neighboring muscles of the face, and lastly in the muscles of the soft palate, pharynx, and larynx. Still, there may be some deviation from this. Usually the progress of the disease is very gradual. There may be an apparent arrest of the trouble; or less often there are quite sudden exacerbations. When all the different symptoms are well developed, the clinical picture is unusually characteristic. The peculiar immobility of expression; the broad, slightly gaping mouth, with the atrophied lips; the almost unintelligible speech, low, monotonous, and labored; and the inability to swallow—these often betray the disease at once. The last stage of the illness is the more distressing, in that the intelligence remains to the end entirely unclouded.

The entire duration of the disease is usually several years—perhaps two to five. If death is not caused by some intercurrent trouble, it is brought about in one of three ways: either through inanition, due to the increasing difficulty of deglutition; or through pulmonary complications—namely, bronchitis, lobular pneumonia, or gangrene, as a result of food passing down the trachea; or through sudden asphyxia or cardiac failure.

Pathology. Nature of the Disease, and its Appearance as a Symptom of Progressive Muscular Atrophy or of Amyotrophic Lateral Sclerosis.—If we seek the anatomical lesion corresponding to the group of symptoms above depicted, we shall find, on microscopic examination of the nervous system, in all cases of this description, a typical disease of the medulla oblongata. The ganglionic nuclei and the nerves (compare the accompanying anatomical diagram, Fig. 166), corresponding to those muscles which we have found to undergo atrophy in bulbar paralysis, present distinct evidences of degeneration. This is most readily demonstrated in the nucleus of the hypoglossus. The ganglionic cells have some of them entirely disappeared, while others are greatly atrophied. The connective tissue is increased in amount, and the walls of the blood-vessels traversing the nucleus are thickened. In the earlier stages there are often many cells which contain granules of fat. The same changes, though perhaps less pronounced, are exhibited by the common nucleus of the vagus and accessorius, that of the facial, and sometimes also that of the glosso-pharyngeal nerve. The other nuclei are perfectly normal. We never find a diffuse "inflammation," but in every case a primary degeneration of the nuclei, which spreads no farther.

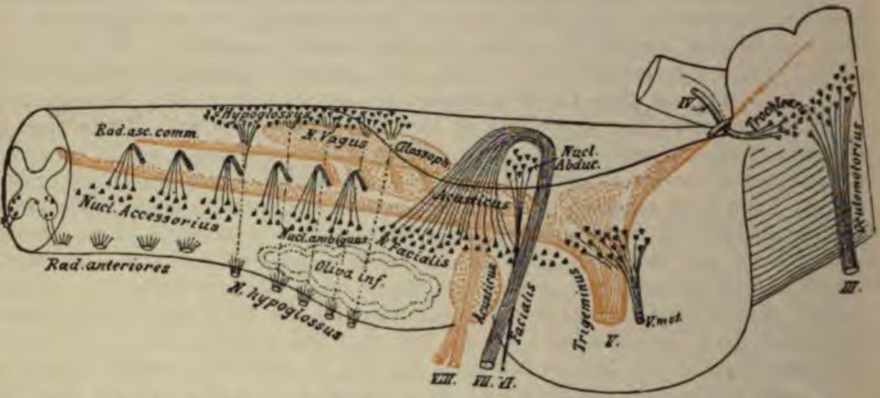

Fig. 166.—The position of the nuclei of the cranial nerves. The medulla oblongata and the pons are supposed to be transparent. The nuclei of origin (motor) are black, the terminal nuclei (sensory) are red. (From Edinger.)

Starting from these nuclei, the degeneration and atrophy may be seen to extend into the nerve-fibers which issue from them. The roots of the hypoglossus, vagus, accessory, and facial nerves can often be seen by the naked eye to be diminished in size and of a gray color. The microscope always shows a partial atrophy of their fibers. Finally, there is a corresponding atrophy of muscles of the tongue, lips, and other parts. We need not enter into detail, for the histological conditions are precisely those seen in the muscles of the trunk and extremities in the spinal form of progressive muscular atrophy.

Thus we find progressive bulbar paralysis perfectly analogous with progressive muscular atrophy. The motor nuclei in the medulla oblongata are the motor and trophic centers of the bulbar nerves and of the muscles which these nerves supply. The relation is precisely the same as exists between the anterior cornua of the spinal cord on the one hand, and the spinal nerves and the muscles which they innervate on the other. In both diseases there is a degenerative atrophy of the individual neuro-muscular elements, the so-called neurones (consisting of nerve-cells and peripheral nerve-fibers), and their corresponding muscular fibers. In both diseases the atrophy and the functional disability of the muscles keep pace with each other, and in both the affection is limited strictly to the motor tract, sensibility suffering no impairment whatever. Certain questions about bulbar paralysis are as unsettled as similar ones about progressive muscular atrophy. It is uncertain whether the primary degenerative process is limited to the bulbar nuclei, and the degeneration of the nerves and muscles is to be regarded as secondary; or whether the entire motor apparatus, from the ganglionic cell to the muscular fiber, is simultaneously attacked; or, finally, whether the atrophy begins in the muscle and involves the corresponding nerve-fiber and nerve-cell only in its further progress. We think it improbable that these points will be cleared up very speedily. Their solution would seem to be only of theoretical interest.

We certainly must recognize, however, the essential identity of progressive bulbar paralysis and progressive muscular atrophy. The resemblance becomes even more striking if we consider that very frequently both diseases are present simultaneously. Often, after a case of progressive muscular atrophy has lasted for some time, the symptoms of bulbar paralysis also appear. On the other hand, an illness may begin with bulbar symptoms, and later on be complicated by atrophy of the muscles of the extremities—almost always seen first in the arms. If cases of this sort come to autopsy, we find a combination of the anatomical lesions of both diseases; in addition to the degeneration of the nuclei in the medulla oblongata, there is marked atrophy of the ganglionic cells in corresponding places in the anterior gray cornua of the spinal cord.

Progressive bulbar paralysis has as close a relation to amyotrophic lateral sclerosis (*vide* page 986) as to progressive spinal muscular atrophy. Amyotrophic lateral sclerosis is at first situated, as a rule, only in the portions of the motor conducting system, including the pyramidal tracts, which supply the extremities; but finally there is very often an involvement of the motor bulbar nuclei—that is, bulbar paralysis sets in. It seems justifiable, therefore, to say that these three diseases—progressive bulbar paralysis, progressive spinal muscular atrophy, and amyotrophic lateral sclerosis—differing as they do in the localization of their lesions, are yet closely allied. They are to be regarded as manifestations of one morbid process which is probably essentially the same in its pathology and ætiology. We do not know the special cause of this process of degeneration. In many cases, although perhaps not in all, we must finally assume a congenital weakness of the affected nerve territory. Such a defective nerve territory succumbs prematurely to the injurious influences which either are associated with the normal functions themselves or act upon them from without in some other fashion. At any rate, there is always a primary chronic degeneration of portions of the main motor conducting tract, in one or another area and varying extent. If we accustom ourselves to regard these three groups of symptoms as really identical, we shall be less puzzled by the slight variations which different cases may present than if we attempt to differentiate the disorders too nicely on account of unessential variations.

Diagnosis.—The diagnosis of a typical case of progressive bulbar paralysis has no difficulties, if we only hold firmly to the definition of the disease and its symptoms as above depicted. Upon careful examination of the other muscles, and consideration of the course of the disease as a whole, we shall be able in each case to determine whether the bulbar trouble is the sole disease, or merely a part of a more extended degeneration of the motor tract. If there are no symptoms but those referable to the medulla oblongata, we must bear in mind that the phenomena of genuine progressive bulbar paralysis may be closely simulated by other bulbar diseases. The acute troubles, such as thrombosis or hæmorrhage, although they produce similar symptoms, can easily be differentiated by the manner of their appearance, contrasting with the invariably slow development of genuine bulbar paralysis. It is, however, much more difficult to eliminate gradually forming tumors situated in the medulla oblongata or its vicinity. Here prolonged observation is frequently needed, until finally such phenomena appear as are foreign to typical bulbar paralysis. Such symptoms are disturbances of sensation and invasion of the upper division of the facial, the nerves of special sense, and the ocular muscles. The same is true of that rare trouble, diffuse sclerosis of the medulla oblongata.

It should also be mentioned that bilateral cerebral trouble may occasion so complete a paralysis of the tongue and lips, according to Lépine and others, as to simulate bulbar paralysis. Such cases have been termed "glosso-labio-pharyngeal paralysis of cerebral origin," or pseudo-bulbar paralysis. Indeed, in rare instances, a similar group of symptoms seems to be referable to unilateral cerebral disturbances. This is explained by assuming that the muscles involved upon both sides receive at least a portion of their motor nervous fibers from the same hemisphere. And yet in most of these cases of pseudo-bulbar paralysis the exclusion of the genuine disease is possible, because certain variations from the typical course of the disease are pronounced enough to set us right. Thus there is an apoplectic onset or repeated apoplectiform attacks. The paralysis is not perfectly symmetrical. There is also paralysis of the extremities on one or both sides. The lips and tongue react normally to electricity without much atrophy, or there are marked signs of mental weakness. In most cases there is an extensive arteriosclerosis at the bottom of the whole morbid process, for the sclerosis of the cerebral arteries often leads to multiple foci of softening or to multiple hæmorrhages.

Prognosis and Treatment.—Despite the unfavorable prognosis of progressive bulbar paralysis, we must at least try to check the progress of the disease. Electricity may perhaps be regarded as the most promising means to employ. To influence the seat of the trouble, galvanization is chiefly used. The poles are applied to the two mastoid processes, every day if possible, for two or three minutes, and the current is repeatedly reversed. We may also galvanize the sympathetic nerve and the affected muscles of the lips and tongue. Upon the muscles the faradic current may also be tried. When deglutition begins to be impaired, it is an excellent thing to excite the action of swallowing, by galvanism. For this the anode is placed upon the nape of the neck, and the cathode upon one side of the larynx. At every cathodic closure (KaS), or every time that the cathode is passed across the side of the larynx, there is a reflex act of deglutition. The current should be of medium strength.

It may be well to prescribe further a resort to treatment by baths, or the "cold-water cure" may be cautiously tried, but we should not expect either of these procedures to have any special influence on the disease. The same internal remedies are recommended as in the chronic diseases of the cord, especially argentic nitrate, ergotine, and potassic iodide. For salivation, atropine may prove beneficial, in pills of $\frac{1}{120}$ of a grain (gramme 0.0005), three or four to be taken daily.

The way of giving nourishment is important if deglutition is impaired. We should try carefully to avoid having the food go down the wrong way, lest pulmonary complications ensue. It is therefore wise not to defer the use of the stomach-tube too long, through which we may introduce milk, eggs, wine, and the various infant foods.

In the distressing close of the disease, narcotics must be exhibited to lessen the patient's suffering, at least as far as we can.

CHAPTER II

THE RARER FORMS OF CHRONIC BULBAR PARALYSIS, PROGRESSIVE OPHTHALMOPLEGIA AND ASTHENIC BULBAR PARALYSIS

1. Progressive Ophthalmoplegia

As we have seen, the form of chronic bulbar paralysis described in the previous chapter as "typical," is practically limited in its effects to the distribution of the hypoglossus, the labial division of the facial, and the pharyngeal muscles. Possibly the reason it extends no farther is merely that death is so speedy. But there are a few rare cases where the chronic degenerative process comes to involve other motor nuclei, together with the corresponding nerve-fibers and muscles. Of course the clinical phenomena of these cases vary from the ordinary; and yet there is no real reason to distinguish them from common bulbar paralysis, particularly as all sorts of transitional forms are to be observed. Thus, we have ourselves noticed that there is sometimes a symmetrical and slowly progressive paresis of the upper division of the facial, and in particular of that portion which supplies the cheek, complicating the glosso-pharyngeal paralysis. In other cases we have seen the degeneration attack from the start the entire distribution of the facial, gradually producing a complete "*diplegia facialis.*" Sometimes, also, the ordinary symptoms of bulbar paralysis are accompanied by disturbances in the area of distribution of the ocular nerves, probably the result of degeneration of the corresponding nerve-nuclei (see Fig. 166, page 1042).

What seems very remarkable is that the process may be confined entirely to the ocular muscles. A. von Graefe named this condition progressive ophthalmoplegia. Another name is "anterior bulbar paralysis." In this form a bilateral paralysis of all the external ocular muscles progresses with extreme slowness and is perfectly symmetrical. The movements of the eye are therefore impaired in all directions. Diplopia is never present. The pupil reacts to light, and almost always the power of accommodation is preserved. Finally both eyeballs become absolutely motionless, and there is a well-marked though incomplete ptosis. There is, beyond a doubt, a progressive degeneration of the nuclei and fibers of the corresponding nerves—that is, the abducens and motor oculi; but with these the process may stop, spreading no farther. We have ourselves lately met with a patient who presented total bilateral ophthalmoplegia, in whom the condition had existed without the slightest change for fifteen years. Besides the oculo-motor paralysis the muscles of the face (Fig. 167) and even those of the body are sometimes involved. On the other hand, in a few cases the process seems to be limited to only a part of the oculo-motor nerves (as the abducens).

The diagnosis of genuine progressive ophthalmoplegia is usually easy, if we hold strictly to the definition of the disease as a degeneration of the motor nuclei and nerves exclusively (that is, completely analogous to progressive spinal muscular atrophy and bulbar paralysis). We shall thus avoid confusing it with those cases in which progressive ophthalmoplegia occurs as one symptom of a complicated morbid process of quite a different sort. Thus we must bear in mind particularly that tabes and general paralysis may lead to complete ophthalmoplegia To these cases may be added those that follow syphilitic infection. Multi sclerosis may also lead to almost complete ophthalmoplegia; and, finally, the same symptom-complex has been observed as a post-diphtheritic nervous disease.

The progressive ophthalmoplegias which develop after birth are also to be distinguished from the congenital defects in the movements of the eyes. Complete external ophthalmoplegia (that is, paralysis of all the external ocular muscles) has been repeatedly observed as a congenital condition, and also congenital bilateral abducens paralysis, congenital ptosis, etc. Autopsies of such cases are still lacking. There are probably defective conditions of the corresponding nerve-nuclei, and perhaps of the nerves or muscles also.

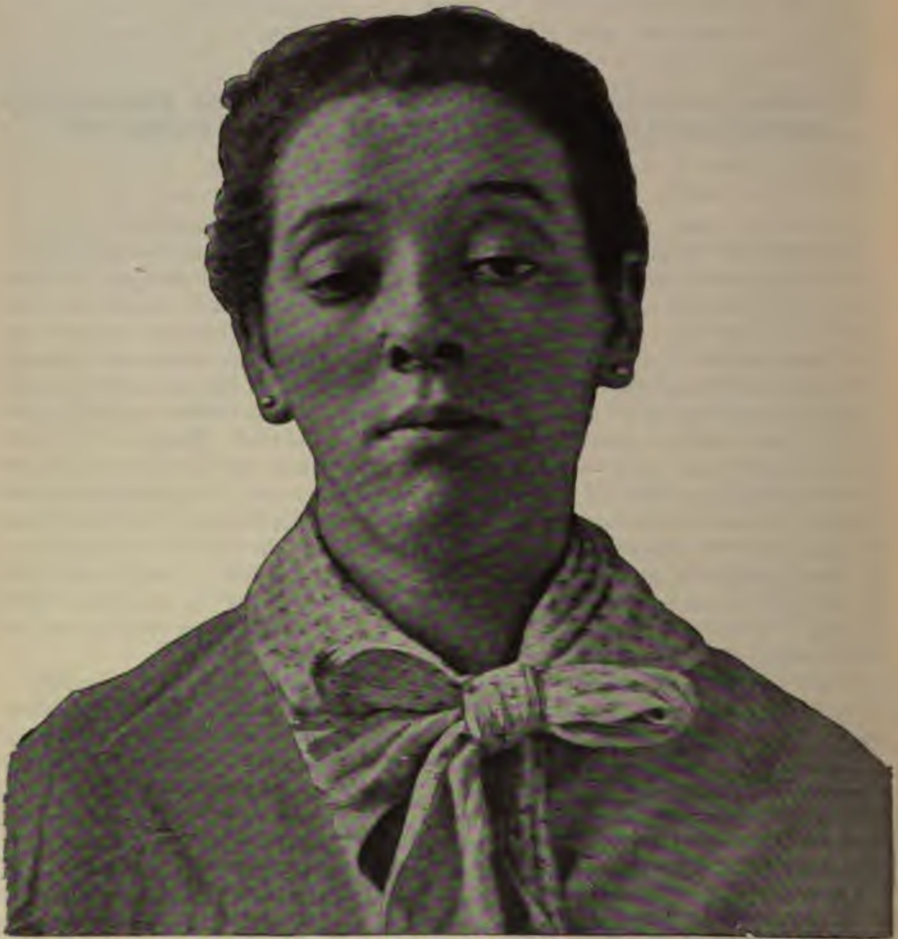

FIG. 167.—Progressive ophthalmoplegia. Paralysis of both eyeballs with bilateral ptosis. Weakness of both facial nerves. (Erlangen Medical Clinique.)

2. ASTHENIC BULBAR PARALYSIS. PSEUDO-PARALYTIC MYASTHENIA

We will speak at this point of a very remarkable and peculiar type of disease, the milder forms of which were first described by Erb, and the severe and fatal forms by Oppenheim. The symptoms of the affection consist of ptosis, paresis of the facial muscles, and disturbances of mastication, swallowing, and talking. On more careful observation it is seen that these symptoms are only in part uniform and persistent, but that they are due chiefly to an extremely rapid fatigue and exhaustion of the affected muscles (Goldflam). Such a patient can, for instance, utter a few sentences quite plainly, but, if he continues to talk, his speech grows more indistinct and inarticulate, and finally it becomes an incomprehensible lalling. The same thing is seen in chewing and swallowing: the first mouthfuls are chewed and swallowed in quite the normal fashion, but in a few minutes it becomes wholly impossible to take any more food. Besides these "bulbar" symptoms, which at first are the most striking, we also see very similar symptoms in the muscles of the extremities; usually the same rapid fatigue progressing to the most complete failure of their function. One of our patients, if she had previously rested, could go up a flight of stairs very well, but on the second attempt she had to take hold of the banisters, and the third or fourth attempt failed completely on account of the complete muscular weakness that ensued.

As Jolly has found, we can detect this same abnormal fatigue by continued faradic stimulation of the muscles.

This clinical condition permits us to draw the conclusion that there is no permanent and complete atrophy or degeneration of the motor apparatus as a basis for the clinical symptoms; and, in fact, in those cases in which death finally ensued (in one or two years) from choking, insufficiency of respiration, etc., the autopsies have thus far been wholly negative (hence the term "bulbar paralysis without anatomical lesion"). This is also the reason why in this affection decided improvement or even complete recovery may occur after the disease has continued for several months, if the functional impairment of the affected nerve territory again disappears. In order to express this distinction between the bulbar paralyses due to coarse anatomical changes and the merely functional disease now in question, we have proposed for the latter the name of "*asthenic bulbar paralysis*." We must, however, note that the disease is not limited exclusively to the territory of the bulbar nerves, although the bulbar disturbances are usually most prominent in the clinical picture. On the other hand, there are apparently some cases where the abnormal fatigue and the consequent symptoms occur only in the extremities, without any implication of the bulbar muscles. For this condition the name proposed by Jolly, "*pseudo-paralytic myasthenia*," is more suitable.

From what has been said above, the prognosis is to be regarded as not absolutely unfavorable, but recurrences are to be dreaded even when recovery has apparently set in. The treatment is first of all to provide the greatest physical rest and care for the patient. If it is difficult to take food, nourishment must be given cautiously by the œsophageal tube. We may try mild massage and galvanization of the muscles, and central galvanization of the spinal cord and medulla oblongata. Internally we have given strychnine, arsenic, and phosphorus, without any perceptible benefit. On the hypothesis that the trouble was perhaps dependent upon some chemical-toxic influence, the cautious use of sweating has also been recommended.

[**Family Periodic Paralysis.**—Goldflam has described a peculiar affection, first carefully studied by Westphal, which merits brief mention. It consists of recurring attacks of paralysis, most marked in the legs, associated with loss of reflex and electrical excitability, without mental or sensory symptoms and with intervals of perfect health. Thirty-five out of fifty-three cases occurred in three families, but the families showed no special signs of nervous degeneracy. The attacks are apt to begin at puberty, and they are also apt to come on during sleep after some unusual muscular exertion. The frequency of the attacks varies greatly, from one or two in a lifetime to several times a week. The average duration of the individual attack is from ten to forty-eight hours. The attacks begin somewhat gradually, usually in the legs, and they may increase to complete paralysis of all the muscles of the limbs, body, and neck. The cranial nerves are rarely involved. The deep reflexes are abolished, and the plantar reflex is often absent. There is a quantitative diminution of electrical reactions, sometimes amounting to complete abolition, but there is no reaction of degeneration. The mechanical irritability of muscles and nerves is also lost. In two cases the heart became enlarged during the attack. The sphincters are not affected. The pathology is wholly unknown. Goldflam believes that the trouble is in some way connected with the muscular dystrophies. The prognosis is good so far as life is concerned, but the attacks usually recur through life. Treatment is unavailing.—K.]

CHAPTER III

ACUTE AND APOPLECTIFORM BULBAR PARALYSIS

1. Hæmorrhage into the Medulla Oblongata and the Pons

Hæmorrhage into the medulla oblongata and the pons is much more frequent than into the spinal cord, but it is much rarer than cerebral hæmorrhage. As to its production, the same views are held as will be considered in detail under cerebral hæmorrhage, in the next section. In the first place, there is probably always some disease of the blood-vessels—that is, atheroma or miliary aneurism—and then some factor productive of increased arterial tension. There may be cardiac hypertrophy, nephritis, excessive bodily exertion, or alcoholism. Now and then injuries of the occiput are followed by an effusion into the medulla. It is not rare to have secondary and usually small ecchymoses in acute inflammation of the spinal cord (*vide infra*), and in purulent meningitis, or in connection with new growths which are richly vascular.

The anatomical conditions produced by bulbar hæmorrhage are so completely analogous with those of cerebral hæmorrhage that the reader may safely be referred to the succeeding section about these also. The size of the lesion varies greatly. Bleeding extensive enough to affect the greater part of a transverse section is more frequent in the pons than in the medulla oblongata. If the blood is poured out close under the floor of the fourth ventricle, as has been repeatedly observed, it may break into the ventricle. If death be not speedy, the blood is mostly absorbed, and in its place develops either an "apoplectic scar" or an apoplectic cyst.

There may be slight prodromata, but the real symptoms of bulbar hæmorrhage are very sudden. There is almost always a pronounced apoplectic seizure. The patient has a shock, falls down, and becomes dizzy or even unconscious. In other cases there may be headache, vomiting, tinnitus aurium, and clonic spasms, or even a typical epileptiform attack.

In most cases death is speedy, if not immediate. This is probably due in every instance to grave lesions of the respiratory and circulatory centers, rendering continued existence impossible. Sometimes the initial symptoms abate, whereupon the local results of the lesion become appreciable.

The first characteristic feature of bulbar paralyses now manifests itself, that disturbances are particularly great in the distribution of the bulbar nerves, while in cerebral apoplexy they never appear in the same way. Another point is, that these paralytic symptoms are combined with paralysis of the extremities in a peculiar way, as a result of the anatomical relations. For the same reason, the order of the paralysis in the extremities may also be peculiar. Of the bulbar paralyses we may mention more or less complete paralysis of the tongue, and a consequent difficulty in articulation (anarthria); frequent inability to swallow; and paralysis in the distribution of the accessorius, facial, and trigeminus. If there is a lesion of the pyramidal tracts in the pons or medulla, we have paralysis of the extremities in addition to the specific bulbar symptoms. If the hæmorrhage be extensive, all four extremities may be more or less completely paralyzed; but in most instances the paralysis is unilateral. In the larger number of hæmorrhages into the pons there is crossed paralysis. The paralysis of the extremities is upon one side, and that of the facial is on the other side. This is a great aid to diagnosis. It is easy to see how this happens if we bear in mind that the cerebral fibers of the facial cross at a point certainly much higher than the decussation of the pyramids, in which latter place the motor fibers of the extremities cross. Now a hæmorrhage may be situated in one side of the pons, above the decussation of the pyramids but below that of the facial. This would occasion (*vide* Fig. 168, *y*) a paralysis of the facial on the same side with the lesion, and of the extremities upon the opposite side; but if the lesion be higher, above the place where the facial crosses over, all the paralytic symptoms would be on the opposite side of the body (*vide* Fig. 168, *x*).

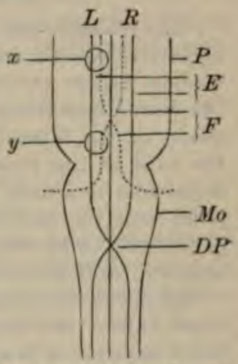

FIG. 168.—Diagram of focal diseases in the pons. *L.* Left. *R.* Right. *P.* Pons. *Mo.* Medulla oblongata. *DP.* Decussation of the pyramids. *E.* Fibers to the extremities. *F.* Facial fibers. *x.* Lesion in the upper half of the pons. *y.* Lesion in the lower half of the pons.

In other, rarer instances we observe similar combinations; only some other bulbar nerve replaces the facial, such as the hypoglossus or abducens. In a few cases the lesion is at the very decussation of the pyramids. This is extremely rare in hæmorrhage, though somewhat more frequent in troubles of a different nature. The result may be that the motor fibers for one extremity are cut off before they cross, and those of the other extremity after they have crossed. Thus is produced the rare phenomenon of a crossed hemiplegia—i. e., paralysis of the arm on one side and of the leg on the other.

Disturbances of sensation in the skin of the paralyzed extremities sometimes result from trouble in the pons, but they are very seldom extreme, and they can not be made available for making out the exact locality of the hæmorrhage, since the course of the sensory fibers through the upper extremity of the spinal cord is still almost unknown. The anæsthesia sometimes observed in the distribution of the trigeminus is of more value, as this may be due to a lesion of the nucleus or root of the nerve.

There are other symptoms, which are indeed rare, but which bear an important relation to certain nervous centers of the medulla. Thus, there may be marked respiratory disturbance; the pulse may become rapid or irregular; there may be vaso-motor derangement, as shown by a rise of the cutaneous temperature and by a subjective sensation of warmth; and occasionally there are temporary albuminuria and glycosuria. The temperature of the body is generally normal at first, or nearly so; but in case of a fatal termination it often rises greatly, even to 107.5° (42° C.) or higher.

As to the prognosis of bulbar hæmorrhage, speedy death has been repeatedly observed, as we have said. If the immediate effects be successfully withstood, the prospect becomes more favorable. The effusion is gradually absorbed, the symptoms of compression abate, and there is a steady progress toward comparatively good or even perfect health. More often, however, some of the paralytic symptoms remain stationary, either in the distribution of the bulbar nerves (the lingual or pharyngeal), or in the extremities, as shown by persistent hemiplegia. If this latter be the case, the subsequent contractures and other symptoms are the same as in ordinary cerebral hemiplegia.

The diagnosis of bulbar hæmorrhage is based upon the apoplectic onset, and upon the presence of specific bulbar symptoms, such as disturbance of speech and of deglutition, and, most characteristic of all, if it occur, a crossed hemiplegia. The differential diagnosis between embolism and hæmorrhage can hardly ever be made with certainty (*vide infra*).

The treatment, not only of the seizure but of the persistent paralysis, should conform to the principles which will hereafter be set forth in describing the treatment of cerebral hæmorrhage. If the bulbar nerves present obstinate symptoms, we must employ the same means as in chronic bulbar paralysis, the most effective being electricity.

2. Embolism and Thrombosis of the Basilar Artery

The medulla and pons receive their blood chiefly from branches of the anterior spinal, vertebral, and basilar arteries. These branches penetrate the anterior median fissure and then proceed to the nerve-nuclei. A far smaller portion of the circulation flows through the "arteries of the roots." These are minute offshoots of the lateral branches of the basilar and vertebral arteries, which enter the cord at the roots of the nerves and penetrate to the corresponding nuclei. According to Duret, the nuclei of the hypoglossal and accessory nerves are supplied from the anterior spinal and vertebral arteries; those of the vagus, glossopharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries; and the nuclei of the facial, trigeminus, and the three nerves to the ocular muscles by branches of the basilar. There may be individual exceptions to these rules. Occlusion by embolism or thrombosis of the arteries just named must occasion a secondary softening in corresponding portions of the medulla, and it is, therefore, a not very infrequent cause of apoplectic, or at least very rapidly developing, bulbar paralysis.

The causes of thrombosis or embolism in the arteries just mentioned are the same as we shall consider minutely when treating of cerebral softening. Emboli are most frequent in cardiac disease. They occur only in the vertebral arteries, oftenest in the left one, and are never primary in the basilar artery; but an embolus may be enlarged by thrombosis after lodging in one of the vertebral arteries, and then block up the basilar. Thrombosis is of more frequent occurrence, and results from chronic changes in the arteries, mainly atheroma or syphilitic endarteritis. The latter disease, one favorite locality for which is the basilar artery, is the commonest cause of acute softening of the pons.

The anatomical condition is likewise similar to that in cerebral softening (*q. v.*). In the region which is deprived of arterial blood by the occlusion of the affluent vessel, the acute anæmia entails necrosis and disintegration of tissue. A spot of "softening" results, made up mainly of vestiges of nervous tissue and numerous cells filled with granules of fat.

When the basilar artery is blocked up, the symptoms appear very suddenly. There is either an apoplectic seizure, or at least a very rapid development of paralysis (occupying only a few days). The symptoms of the first onset are, in all essential points, those of bulbar or even of cerebral apoplexy. Although there is usually no marked loss of consciousness in apoplectic bulbar paralysis, yet no great diagnostic significance can be assigned to its absence. The sudden obstruction of the basilar artery produces such a disturbance of the circulation even in anterior portions of the brain as may suspend consciousness. In some few instances this circulatory derangement may even give rise to choked disk, as seen by the ophthalmoscope. Often there are noticeable respiratory and cardiac symptoms, such as Cheyne-Stokes respiration, and rapid pulse.

If death be not immediate, and we are therefore enabled to make out the symptoms due to the local disturbance, we usually observe the same phenomena as have just been described under bulbar hæmorrhage. There is sometimes paralysis of all the extremities, but usually there is trouble only upon one side. Then we have the characteristic crossed hemiplegia. The facial nerve or the nerves of the ocular muscles may be paralyzed. It has repeatedly happened that the paralysis seemed at first much greater upon one side, but after a few days changed over to the opposite one. This must be due to changes in the circulation; the thrombus grows larger, or a collateral circulation is developed. The specific bulbar symptoms are the familiar ones of all bulbar derangements—namely, lingual paralysis with resulting difficulty in articulation, pharyngeal paralysis, and rarely deafness, as a result of lesion of the acoustic center. Of course, the severity and extent of all these symptoms must vary according to the location and size of the spot of softening.

The prognosis of cases of this sort is almost always unfavorable. Death results in a few days at latest. It is often ushered in by a high temperature. Exceptionally, there is a transition into a chronic form.

We need say nothing about treatment, except that the same remedies are employed as in other acute bulbar diseases.

3. Acute Inflammatory Bulbar Paralysis

(*Acute Bulbar Myelitis. Multiple Bulbar Neuritis*)

"Acute bulbar paralysis," in the stricter sense of the term, means a form of disease where marked symptoms of bulbar derangement appear acutely—that is, within a few days or weeks. The anatomical lesion is probably an acute inflammation of the medulla oblongata. It is a rare disorder, and its ætiology is doubtful. There are usually mild prodromata: vertigo, headache, and, in one case of our own, painful sensations in the back of the neck. Evident bulbar symptoms very quickly follow. Usually the first of these is dysphagia. Not only is deglutition impaired, but the paresis of the soft palate and of the laryngeal muscles allows liquids to enter the nostrils or the larynx. The tongue also becomes gradually paralyzed, speech becomes indistinct, and, if the soft palate be involved, nasal.

Sometimes the extremities also become paretic, as a result of the extension of the disease to the region of the pyramids; but in many instances the extremities remain unaffected to the end. Paralyses of the facial nerve and of the ocular muscles are somewhat more frequent. The temperature is sometimes a little elevated (100°–102°, 38°–39° C.), but not always. The pulse is almost invariably rapid; in our patient it was 148.

The prognosis is apparently always bad. Often death takes place in four to eight days, or it may be not till the end of two or three weeks. It is invariably preceded by all the tokens of paralysis of respiration. In our case there was well-marked paralysis of the diaphragm at the end.

As yet, few autopsies have been reported. Generally the medulla presents no macroscopic changes. Exceptionally, it can be seen to be softened and mottled with minute hæmorrhages. The microscope detects abundant evidence of inflammation: granule-cells, infiltration with nuclei around the blood-vessels, thickening of the walls of some of the blood-vessels, small extravasations, swollen axis-cylinders, etc. It should also be borne in mind that precisely similar clinical phenomena seem often to be referable to peripheral changes, such as multiple neuritis affecting the bulbar nerves.

The treatment of acute bulbar paralysis is, of course, almost hopeless. In early stages we should apply counter-irritation to the nape of the neck; and we might prescribe mercurial inunction. It might be well to employ the constant current, applied at the back of the neck, and also in such a manner as to excite the movements of deglutition. We found injections of strychnine useless. Toward the end, narcotics are indispensable.

CHAPTER IV

COMPRESSION OF THE MEDULLA

ACUTE compression and other injuries of the medulla are due most frequently to fracture or dislocation of the atlas and axis. As is well known, dislocation of the axis, or backward dislocation of the atlas, usually causes instant death.

Gradual compression is seen in chronic disease of the bones around the medulla, in caries and tumors of the occiput and of the first two vertebræ. Enchondroma of the base of the skull; new growths of the sphenoid, at its junction with the occipital; tumors of the dura; and sometimes even tumors of the cerebellum—may all excite by their pressure the gravest bulbar symptoms. We should also mention aneurism of the vertebral artery at its upper end, and of the basilar, as capable of doing similar harm. In all these cases the main cause of disturbance is undoubtedly the mechanical pressure, either directly destroying the nervous tracts or interrupting the transmission of nervous influences; but the circumstances may be further complicated by hæmorrhages, and sometimes perhaps by inflammation of the medulla itself.

The clinical phenomena of gradual bulbar compression resemble those of spinal compression, in that they usually begin with symptoms of irritation in the distribution of those nerves the roots of which are first affected. There are neuralgia of the trigeminus, twitching of the facial muscles, tinnitus aurium, etc. If the compression becomes greater, there are more serious bulbar symptoms: disturbances of speech and deglutition, paralysis of the tongue, soft palate, face, and very likely motor (paretic or ataxic) and sensory symptoms in the extremities. Usually we also see general cerebral symptoms, such as vertigo, headache, vomiting, and sometimes epileptiform convulsions.

We can not, of course, draw up a definite and rigid list of symptoms, since both the individual symptoms and the general course of the disease exhibit great variations according to the way in which the compression is brought about. The diagnosis can be made in those cases only where some ætiological factor such as trauma or caries of the vertebræ is known to exist. Aneurism of the vertebral artery is said by Möser sometimes to give rise to a loud systolic murmur heard between the mastoid process and the spine. In all other cases we can seldom do more than surmise the truth. Slow compression is distinguished from genuine progressive bulbar paralysis chiefly by the course it pursues—that is, there are initial symptoms of irritation—by the greater complexity of the clinical phenomena, such as sensory lesions and hemiplegia, and sometimes by the asymmetry of certain symptoms. If the anterior part of the medulla is compressed in the region of the pyramids, there may for a time be no bulbar symptoms, but merely motor symptoms in the extremities. These are chiefly paretic or spastic.

The prognosis is almost always bad, as can be inferred from the nature of the causative disease. Death is brought on either by inhalation-pneumonia, or by paralysis of respiration. Treatment must be purely symptomatic, and should follow the same rules as in progressive bulbar paralysis.

V.—THE DISEASES OF THE BRAIN

SECTION I

Diseases of the Cerebral Meninges

CHAPTER I

HÆMATOMA OF THE DURA MATER

(Internal Hæmorrhagic Pachymeningitis)

Ætiology and Pathology.—Hæmatoma of the dura mater is the name given to effusions of blood found on the inner surface of the dura mater. These effusions are of considerable area, but of moderate thickness, and are usually encapsulated. There has been much discussion as to their mode of origin, and views still differ. One is that the hæmorrhage is the primary lesion, and that the connective-tissue membranes are developed only by the organization of the clot. This conception was originally the prevailing one, but was opposed by Virchow, who was led by the results of his own investigations to maintain that the hæmorrhage was always secondary. The primary process he believed to be a peculiar sort of inflammation—"hæmorrhagic pachymeningitis." This gave rise to a new growth of richly vascular connective tissue, into which the hæmorrhage took place. Of late, however, the tendency is again to regard the hæmorrhage as the initial change, at least in certain cases, and to refer it to an affection of the walls of the blood-vessels which diminishes their power of resistance.

The mildest forms of internal pachymeningitis present a delicate membrane upon the inner surface of the dura mater, quite easily separable, of a reddish color, and dotted with numerous red and brownish spots. These spots are due to minute hæmorrhages and collections of hæmatoidine. The membrane itself is a delicate interstitial tissue, traversed by numerous wide capillaries.

In more advanced cases the thickening is much greater. There are usually several layers, the newest and most superficial being nearest the brain. The oldest, which is in apposition with the dura mater, is composed of connective tissue that has already become rather firm and fibrous. It is evident from this lamellar structure that the whole process goes on by fits and starts. The clinical course of the disease will be seen from what follows to agree well with such a view. The effusions are sometimes very extensive, even larger than a hen's egg, and exercise no slight pressure upon the underlying cerebral parenchyma. The hæmorrhage always takes place inside the mass, or between its layers. The effusion may, however, break through the innermost layer, so that the blood flows into the arachnoideal spaces; this is known as "intermeningeal apoplexy."

The favorite location of the hæmatoma is the parietal region. It is sometimes found at the base of the brain, in the posterior or middle fossa. Occasionally the hæmatoma is bilateral.

Hæmorrhagic pachymeningitis is not a rare disease. It is sometimes found to exist in a moderate degree in chronic cardiac, renal, or pulmonary cases, which come to autopsy. Usually there have been no special symptoms, the lesion being discovered incidentally. It has been found in like manner in connection with a great many acute infectious diseases, such as typhoid fever and small-pox. It is a more important and more frequent complication in other chronic cerebral diseases, in particular such as induce marked atrophy of the brain as a whole. It is especially common in general paralysis of the insane and in other forms of dementia. In the apparently primary hæmorrhagic pachymeningitis chronic alcoholism is the commonest and most important causal factor. In hard drinkers it is not very unusual for the hæmatoma to be so extensive, if it occurs at all, as to cause grave cerebral derangement. Very likely changes in the vascular walls, such as atheroma and fatty degeneration, contribute an important part to the result in such patients. Injury is a second important cause. We have seen a case of hæmatoma of the dura with a fatal termination in a patient who some months before had received very severe blows on the head in a fight. Hæmatoma may also occur in all diseases where there is a general hæmorrhagic diathesis. Thus it is seen in pernicious anæmia, leukæmia, and scurvy. Here certainly the hæmorrhage is the primary event.

As might be inferred from the ætiological factors enumerated, the disease is found chiefly in advanced life, and much oftener in men than in women.

Symptoms.—Not infrequently a hæmatoma of the dura is found post mortem, which had during life been entirely unsuspected. Either the hæmorrhage was not extensive enough to cause any symptoms, or the brain exercised that remarkable tolerance which it sometimes shows even when there are wide-reaching lesions; or such symptoms as may have existed escaped particular notice, in the severity of the more general symptoms (of typhoid fever or some similar disease). But in other cases hæmorrhagic pachymeningitis excites grave symptoms, although they are seldom so characteristic as to reveal the diagnosis; for individual cases vary greatly, according to the size of the hæmorrhages, their location, and the frequency of their recurrence.

Almost always the beginning of the disease is rather sudden. It may even be like an apoplectic seizure. The symptoms are referable partly to the general effect of the hæmorrhage upon the brain, and partly to the exact locality of the hæmorrhage. The more general symptoms comprise headache, impairment of intelligence (that is, stupor or even complete coma), slow or irregular pulse, vomiting, and contracted pupils—all being symptoms of cerebral compression. Now and then we even find choked disk. Other phenomena are added to the above when the hæmatoma occupies its usual position, upon one side and in the neighborhood of the motor cortical region, or central convolutions. The hemiplegic symptoms are not infrequent, such as hemiparesis, and, from the irritation which the effusion produces in the motor centers, twitchings and convulsions in one half of the body. Sometimes these symptoms are limited to a single extremity or to the distribution of the facial nerve. Aphasia has been repeatedly observed when the hæmorrhage was near the island of Reil. If the effusion increases, the motor disturbance becomes correspondingly aggravated, and it may become bilateral. Sensation is usually little impaired.

The further course of the disease varies greatly in different cases. In the worst cases there is speedy death, usually ushered in by deep coma. In others, the first symptoms are followed by improvement, although mild indications of cerebral pressure persist, such as headache or vertigo, or else local symptoms, such as hemiparesis. It is possible for the effused blood to be absorbed, and complete recovery to ensue; but usually new hæmorrhages and corresponding symptoms arise. It is precisely this appearance of the symptoms in separate attacks, this frequent recurrence of severe cerebral disturbances, that is characteristic of hæmatoma of the dura mater. As already intimated, the way in which the anatomical lesions develop explains this perfectly. Thus the disease may drag on for months and years, sometimes improving and sometimes getting worse again. Then some attack at last proves fatal. Arrest and actual improvement are still possible even in the later stages, although often the features of the case have meanwhile undergone essential alteration because of the progress of some causative disease. In general, the clinical phenomena of hæmatoma of the dura are often complicated and obscured by the co-existence of the primary disease.

The **diagnosis** is therefore difficult. The main points may be recapitulated as follows: First, the existence of ætiological factors, such as alcoholism, previous injury, or chronic cerebral disease; second, the sudden onset, and also the abrupt appearance of further symptoms, the alternation of rapid aggravation and improvement; and third, the existence of symptoms which experience has taught us to refer mainly to the cortex of the brain, namely, unilateral convulsions, monoplegic paresis and contractures, and contracted pupils. Nevertheless, frequent errors in diagnosis can not be avoided.

Treatment.—The possibility of therapeutic interference being successful is very small. In apoplectic shocks, ice to the head is useful; and if the patient be robust, it may also be advisable to use local depletion, by leeches on the temples or behind the ears. It is also customary to prescribe some such drug as senna or calomel for "intestinal depletion."

If the first onset is successfully withstood, the main points in the further treatment are general hygienic and dietetic directions, so as to guard as far as possible against fresh hæmorrhages. Alcohol and excessive bodily or mental exertion should be forbidden. Of course, paralysis or other persistent disturbances may call for special attention.

CHAPTER II

PURULENT MENINGITIS

(Purulent Cerebral Leptomeningitis. Meningitis of the Convexity)

Ætiology.—Purulent inflammation of the dura mater has no clinical importance, for it is very rare, and occurs only as the result of the extension of disease from neighboring parts. We shall accordingly consider below purulent inflammation of the pia mater only. One important variety of this disease has already been discussed (see page 111 *et seq.*) as one of the infectious diseases, under the name of epidemic cerebro-spinal meningitis. There we saw also that the occasional sporadic cases of primary or "idiopathic" meningitis are probably identical ætiologically with those of epidemic meningitis. In all other instances, purulent meningitis is a secondary disease—that is, the specific pus coccus (usually the streptococcus pyogenes), which excites the purulent inflammation, originates in some other organ primarily, and affects the meninges only secondarily. We should, therefore, seek most carefully in every case of purulent meningitis, at the bedside and more particularly at the autopsy, to discover the way by which the pathogenic virus reached the meninges. We have no right to say that the case is one of primary meningitis, strictly so called, until we have made a most careful examination with a negative result. From a clinical standpoint, it is true that many cases of secondary meningitis do seem as if they were primary, because not infrequently the real primary disease excites insignificant symptoms, or perhaps no symptoms at all.

Secondary purulent meningitis is due most frequently to disease of the cranial bones, and in particular to disease of the petrous portion of the temporal bone, with the auditory apparatus therein contained. If we consider the anatomical relations of the middle and internal parts of the ear, we can easily understand why inflammation in them is not infrequently followed by meningitis. Usually it is a caries of the petrous portion of the temporal bone, itself due to an otitis media, which leads to an irruption into the cranial cavity. This is especially apt to take place through the thin vault of the tympanic cavity. It may also extend from the mastoid cells, or by direct propagation along the sheaths of the acoustic or facial nerves, or along the vessels which lie in the petroso-squamous suture. The dura is first attacked, and then the pia. In many instances, the neighboring venous sinuses (transverse, cavernous, and superior petrosal) transmit the inflammation, being first attacked by a suppurative thrombo-phlebitis. Again, exceptionally, a purulent inflammation in the upper part of the nasal cavity or in the orbital cavity (panophthalmitis) may lead to meningitis.

Another and frequent source of meningitis is found in the various injuries of the cranium. In the great majority of these cases there is an open wound, admitting infectious agents which are suspended in the air. The suppuration often commences in the spongy texture of the diploë, thence extending to the dura and pia, either directly or by way of a purulent thrombosis of some sinus into which the veins of the diploë enter. It was, indeed, often affirmed in the past that we could have a traumatic purulent meningitis without any open wound; but this is not easily explained, according to our present views as to the origin of purulent inflammations. There are probably in every case small wounds of the skin, which are perhaps overlooked. The statements are also to be rejected that the heat of the sun's rays, striking upon an uncovered head, may excite purulent meningitis. In most cases of fatal sunstroke we find marked hyperæmia of the meninges, but no inflammation.

Meningitis may have an intra-cranial origin; it is sometimes the sequel of cerebral abscess. No matter what starts the abscess, if it extends to the surface of the brain, it causes a more or less extensive purulent meningitis at that point. An abscess may burst into one of the lateral ventricles, and the infection be carried from that point to the pia at the base of the brain.

All the cases thus far contemplated allow of the explanation that the inflammation reaches the meninges by direct extension; but there is another group of cases where the agent that infects the pia mater originates at some distant part of the body, and is probably conveyed by the blood or lymph currents. These cases are often termed metastatic meningitis.

Of this sort is the secondary meningitis seen in connection with genuine lobar pneumonia, a combination already discussed (see page 215). The meningitis is also sometimes a complication of empyema, rarely of pyæmia and septicæmia, or ulcerative endocarditis, and very rarely of typhoid fever and the acute exanthemata (small-pox, scarlet fever), and of acute articular rheumatism. In each case we must, of course, determine whether the meningitis may not have a connecting link between itself and the primary disease, such as otitis in scarlatina, or secondary empyema in typhoid fever.

Pathology.—For the pathological anatomy of purulent meningitis, we may refer mainly to the statements made on page 112, under epidemic meningitis, for the lesions are similar. The only way to determine whether a meningitis is secondary or primary is by finding or failing to find disease in neighboring or remote parts, for example, pneumonia. The seat of the meningitis will vary according to that of the primary inflammation, if there be any. If the meningitis is due to caries of the petrous bone or to an injury of the skull, the purulent exudation is usually most abundant in the immediate neighborhood of the primary lesion, between the pia and arachnoid. Thence it gradually extends along the surface of the brain, sometimes chiefly on the convexity and sometimes at the base. But in general it may be said that both the secondary and the metastatic varieties of meningitis as a rule affect the convexity, although this is by no means invariably the case. This rule explains why these cases are sometimes termed meningitis of the convexity, in contrast to tubercular meningitis, which latter, as we shall find, has a preference for the base of the brain, and hence is called basilar meningitis. The spinal pia mater is sometimes simultaneously attacked, but not so constantly as in primary, or epidemic meningitis. The brain is almost always involved—the inflammation extends along the vessels which dip from the pia mater into the cerebral parenchyma. It is not a rare thing to find minute abscesses or ecchymoses in the interior of the brain. The whole parenchyma is usually moist, œdematous, and of a doughy consistency. The meningeal exudation exerts upon the brain a pressure which gives rise to important symptoms; by it the superficial cerebral convolutions are often considerably flattened. The lateral ventricles almost always contain more or less sero-pus.

Clinical History.—So varied are the primary diseases which may entail a meningitis, that it is hardly possible to make a sketch of the disease which would suit all cases. If the meningitis comes on during the course of pyæmia, pneumonia, or some other severe illness, its proper symptoms are often inextricably confused with those of the primary trouble; and when the skull or the brain has been mechanically injured, it is very hard to determine whether a meningitis has been excited, because the trauma may of itself produce such serious effects. The following description, therefore, applies chiefly to cases of apparently primary meningitis, or to cases where the meningitis, although secondary, is well marked.

The beginning in such cases may be sudden, or it may be somewhat insidious. Sometimes the grave symptoms appear almost at once, accompanied by a chill and high fever. Sometimes there are for a time indefinite and more or less ambiguous prodromata, but almost always it is the headache which first attracts attention. This grows worse with more or less rapidity, and almost always becomes very violent. Exceptionally it may be insignificant. Not infrequently it varies considerably, being much worse at some hours or on some days than on others. The location of the pain is sometimes frontal, sometimes occipital, and sometimes over the whole head. Next in prominence to the headache, particularly in the later stages of the disease, is the mental disturbance. The patient complains of vertigo, becomes dull and stupid, or begins to wander. The delirium may be extremely violent, but usually there is depression rather than exaltation, and the stupor merges into coma. That the headache still continues may be inferred from the frequent raising of the hand to the head and the grimace of pain whenever the head is moved, until finally the coma becomes so profound that even these reflex actions cease.

Usually these general cerebral symptoms are attended by others referable to the particular locality affected. The neck is rigid. This is most marked when the posterior fossa and the medulla are affected. Then there are all sorts of paralytic or irritative symptoms in the distribution of the cranial nerves, mainly due to lesions of the nerves where they emerge from the base of the brain; there is derangement of the oculo-motor nerves, as shown by paralysis or nystagmus; the pupils are unequal, or are contracted or dilated, and do not react to light; there is paresis of the facial, or trismus, or grinding of the teeth. All these symptoms may be equally pronounced in other forms of meningitis. Sometimes we can detect optic neuritis with the ophthalmoscope. Other symptoms are due to cerebral disturbance, often apparently located chiefly in the cortex. Thus there may be twitchings of individual muscles, or even genuine convulsions in one or more limbs, or paralysis of one extremity or of half the body. Sometimes the autopsy explains these phenomena, but often we fail to find any marked anatomical lesion to correspond to them, and are obliged to ascribe them to circulatory or functional derangement.

Of the remaining symptoms, the fever is most important. Almost always the temperature is decidedly elevated, not infrequently reaching 104° or 105° (40°-40.5° C.). The fever is, however, very irregular. There may be repeated chills with great elevations of temperature. The pulse is generally rapid, and often somewhat irregular. Exceptionally it is less frequent than normal, because of cerebral compression. Vomiting is not a rare symptom, particularly at first. There is almost invariably constipation, and the abdomen is often tense and concave. The urine is scanty, and often contains a trace of albumen. Secondary diseases are sometimes found post mortem, such as lobular pneumonia, due to inhalation of food during the comatose state.

The entire course of the disease occupies only a few days in very acute cases, and scarcely ever exceeds a week or ten days. The termination is almost sure to be fatal. In the few cases of recovery which have been reported the diagnosis is doubtful. In most instances deep coma precedes death, though sometimes it is ushered in by convulsions. There is often a great rise of temperature (107.5°, 42° C., or higher) before the close of life.

Diagnosis.—The diagnosis of purulent meningitis is sometimes pretty evident; but it may be very obscure, so that we can not always avoid confounding it with other severe acute diseases, such as typhoid fever, pyæmia, and general tuberculosis. In general the most characteristic symptoms of any variety of meningitis are intense headache, rapid onset of grave cerebral disturbances, delirium and insensibility, stiffness of the neck, and disturbances in the distribution of the cranial nerves (especially impaired motion of the eyeball and optic neuritis). These last, although often slight, are generally present; and in connection with these separate symptoms we must also always consider the whole course of the disease and any ætiological factors which may exist. Typhoid fever is excluded by its usually slower onset, the greater delay in the appearance of grave cerebral symptoms, the rose-spots, the greater size of the spleen, the characteristic stools, and the peculiar fever-curve. It may also be of importance in diagnosis that in typhoid fever there is usually no leucocytosis of the blood, since in purulent meningitis the number of leucocytes in the blood is increased. Severe septic and pyæmic diseases, including ulcerative endocarditis, likewise excite cerebral disturbances which might be misleading, but these diseases are to be recognized by their ætiology (external wounds, abortion, etc.), cutaneous ecchymoses, septic retinitis, swelling of the joints, and repeated rigors. Uræmia may also simulate meningitis. Sometimes the character of the urine, and the predominance of convulsions, will set us right, but not always. We may state in conclusion that every one who sees many cases must (like ourselves) repeatedly have met with patients presenting the symptoms of a severe and acute cerebral affection apparently primary, without demonstrable cause, and seeming to justify a diagnosis of meningitis, but yet yielding post mortem no signs of disease beyond "hyperæmia," "œdematous swelling," and similar changes of only secondary importance. We are as yet wholly unable to explain such cases.

Granting that meningitis exists, what variety is present? The ætiology is a great help in answering this question. We should endeavor to learn whether there has been traumatism or some old ear trouble. It is well to employ the aural speculum. We can not say that a patient has epidemic meningitis unless several cases occur simultaneously, although herpes is very characteristic, as it appears only exceptionally in other varieties. [Lumbar puncture may sometimes be of assistance in the diagnosis.—K.] Usually tubercular meningitis also can be diagnosticated only by means of the ætiology. Its symptoms, of course, are in almost all particulars identical with those of purulent meningitis. Sometimes, however, tubercles can be detected in the choroid by means of the ophthalmoscope. For further particulars see the next chapter.

Prognosis.—The prognosis of purulent meningitis is almost invariably wholly unfavorable, as follows from what has been said above. In the reported cases of recovery from meningitis the diagnosis must be determined with the greatest caution, but it is not impossible that sporadic cases of epidemic diplococcus meningitis may terminate favorably. We may also state that sometimes in severe infectious diseases "meningitic" symptoms may appear which lead us to fear the worst, but which finally disappear. In acute purulent inflammation of the middle ear, especially in the young, meningitic symptoms (headache, stupor) sometimes set in, which also seem threatening, but which disappear. It is, of course, usually impossible to determine positively the anatomical processes in such cases.

Treatment.—The treatment of the different forms of meningitis varies but little. Locally, the favorite remedies are ice applied to the head, which should be shaved if practicable, and local depletion by means of leeches behind the ears or on the temples. Many physicians recommend cutting off the hair and rubbing in antimonial ointment, or applying ethereal tincture of iodine. We have never tried this. Cool baths with douching can not be employed unless the patient can be moved without too much pain. For violent pain or great restlessness we must use narcotics. The best is morphine subcutaneously. We can not hope for much benefit from other internal remedies, such as iodide of potassium or calomel.

Prophylaxis demands, above all, prompt recourse to the otologist for aural trouble of any kind, and strictly antiseptic treatment of all injuries of the skull. Even in cases of meningitis that have already begun, surgical interference (trephining the mastoid, etc.) may perhaps often check the advance of the inflammation and lead to recovery.

CHAPTER III

TUBERCULAR MENINGITIS

(Basilar Meningitis)

Ætiology.—Tuberculosis of the leptomeninges is always a secondary affection—a sequel to previously existing tubercular disease of some other organ. Why the pia mater should be so often singled out for secondary infection with the tubercular virus, or what path that virus traverses to reach the pia, we can hardly say. We can merely state what the other tubercular diseases are, which, as experience shows, entail tubercular meningitis most frequently. These primary affections may be of themselves productive of grave clinical phenomena, the meningitis merely adding to the complexity of the picture. Again, the primary trouble may not have betrayed itself at all, or its symptoms may have been long ago arrested, so that the meningitis seems to be a primary disease. In some cases even the most careful examination will fail to detect the origin of the trouble.

Tubercular meningitis is oftenest a sequel to pulmonary tuberculosis. It may appear as a terminal complication in cases of advanced phthisis, or it may come on while the signs of pulmonary disease are as yet very slight. Next in order as a causative affection comes tubercular pleurisy. This origin is not infrequent. As we have already seen, most cases of apparently primary pleurisy are due to tubercle. This statement is supported by the fact that it is not very exceptional for the symptoms of tubercular meningitis to supervene suddenly upon what had seemed to be genuine convalescence from pleurisy. In children, and sometimes in adults, the virus may be carried to the meninges from cheesy, tubercular, bronchial or mesenteric glands, or from tubercular or "fungous" disease of the bones or joints. Another danger to adults is tubercular disease of the genito-urinary apparatus. It should also be noticed that a single large tubercle in the brain may lead to miliary tuberculosis of the meninges. In short, we see that it is not impossible for any tubercular infiltration, wherever situated, to communicate infection either to the meninges alone (in some remarkable way), or simultaneously to them and many other organs. In this latter case, where in all probability the blood carries the virus through the system, the meningitis is merely a part of a general miliary tuberculosis (see page 261), while in the former case the reason why the meninges are exclusively or primarily affected depends upon the special form of the infection; but we are almost wholly ignorant of the way by which the tubercle bacilli reach the meninges. We are ourselves disposed to suspect that, in many cases at least, the infectious material first enters the arachnoid sac of the spinal cord through the lymph sheaths of the nerves (e.g., the intercostal nerves), and thence extends upward to the base of the brain.

We sometimes hear the attack ascribed to such causes as over-exertion, mental excitement, traumatism, etc.; but we need hardly say that these can not be properly regarded as ætiological factors, and that usually they are merely coincidences. Age, however, does have an influence; children are much oftener attacked than adults, although the latter also are liable to the disease.

Pathology.—As in tuberculosis of serous membranes, so in tuberculosis of the pia, there are two effects of infection to be distinguished from each other: (1) the development of the specific new growth—that is, of miliary tubercles; and (2) the inflammation. The relative degree of these two varies. Sometimes the tubercles are very abundant and the inflammatory exudation comparatively scanty; and in other cases the inflammation is considerable, although relatively few tubercles are discoverable. The tubercles are usually found in greatest number along the course of the larger blood-vessels, and therefore chiefly in the furrows and clefts of the surface of the brain, in the fissure of Sylvius, at the chiasma, the pons, the medulla, and the cerebellum. And, in general, the base of the brain is usually more affected than the convexity—hence, as we have said, the name of "basilar meningitis." There are, however, exceptions to this rule. We very often find that the region supplied by one or more arteries suffers above other parts; this must be due to the manner of infection. The inflammatory lesions consist of hyperæmia, usually well marked, and a sero-gelatinous exudation of variable amount. That this exudation is partly cellular can always be proved by the microscope, and often even macroscopically from the great cloudiness of the pia; but still we seldom find enough to justify us in calling the process one of genuine purulent inflammation. Small hæmorrhages into the pia are quite often found. The brain itself is usually flattened from the pressure of the meningeal exudation. Often the inflammation involves the brain-substance itself, as shown microscopically by tubercles, inflammatory changes, and capillary hæmorrhages. The ventricles usually, although not invariably, contain a hydrocephalic effusion. This led earlier observers to term the disease "acute hydrocephalus." The effusion is serous, but generally turbid from cellular constituents, and not infrequently tinged with blood. The choroid plexus is engorged, and it may present tubercles. The spinal cord, in the majority of cases, shares in the tubercular disease. Here, too, we find inflammation of the pia and miliary tubercles.

Clinical History.—Tubercular meningitis almost always begins with a prodromal stage, which is often brief, but which may last one or two weeks, or even longer. The patient may be apparently well (*vide supra*) until this comes on, or he may have already shown signs of some other tubercular affection. He now feels somewhat ill at any rate, and begins to complain of headache, worse at some times than at others. There is anorexia and very often constipation. Another frequent prodrome is an attack of vomiting, which may or may not recur. Sleep is disturbed, either by the headache or by a certain general restlessness. We have occasionally met with cases where the illness began with pronounced mental disturbance. The patient became irrational and said and did queer things, and then a few days later there appeared distinctive meningeal symptoms. In two patients, who were hard drinkers, the disease began just like delirium tremens.

After an initial period of variable duration, the general health becomes more and more impaired. The headache increases. The patient takes to his bed, begins to be delirious, and soon presents well-marked symptoms of grave brain trouble. Intelligence becomes more and more impaired. The patient is sleepy, and he can be roused imperfectly by the voice, if at all. At the same time he is usually quite restless at first, grasping at invisible objects in the air, picking the bed-clothes, and continually moving his legs. The delirium may be low or noisy; the patient may keep up a constant singing, screaming, or whistling. The persistence of the headache even in this stage is shown by the facial contortions and complaints of the sufferer, whenever there is a temporary approach to consciousness. There is also, as a rule, decided tenderness in the nape of the neck on pressure, frequently accompanied by great stiffness of the neck. Sometimes there is stiffness of the entire spinal column, and pain in the same. This is certainly due to the coincident spinal meningitis.

Another group of symptoms in the distribution of the cranial nerves are identical with those seen in other forms of meningitis. Ptosis is not infrequent, on one or both sides, due to paresis of the motor oculi. There is strabismus, either internal or external. Symptoms of irritation of the nerves governing the movements of the eye are very frequent, especially in the early stages of the disease. Thus we see slow involuntary lateral movements of the eyeballs, and sometimes nystagmus. The pupils are often unequal; they may be enlarged or contracted, and often they undergo marked and repeated variations in size. The reaction of the pupils to light is usually sluggish, and it may be absent. With the ophthalmoscope we find not infrequently neuritis, or choked disk. In some instances, but not in all, we find also tubercles in the choroid, which, of course, greatly assists diagnosis. Sometimes there is occasional twitching in the distribution of the facial nerve, or a slight tonic contraction, or again paresis on one side. The natural explanation of all these phenomena is that the nerve-trunks are interfered with at the base of the brain, either by the pressure of the exudation or by participation in the inflammatory process, or by the minute hæmorrhages which sometimes take place into the sheath of the nerves.

Disturbances in the extremities may be caused by various lesions. Motor symptoms of irritation are apparently referable for the most part to changes in the cortex of the brain. We see occasional twitching of larger or smaller groups of muscles, or rarely convulsions. These latter may be unilateral, or limited to a single extremity. Sometimes there is well-marked paresis of one half the body or paralysis of one limb, or there may be aphasia, although it is only in a part of the cases that we find post mortem any lesion which explains these symptoms. In most cases, however, there is a particularly large collection of tubercles in certain places upon the cortex cerebri, occasioning a local compression or an inflammatory œdema, which in its turn excites the phenomena mentioned. Sometimes the brain-substance itself is found in a state of red softening underneath these spots. Another not very rare symptom is a peculiar stiffness of the limbs, due either to direct irritation or to reflex action. The reflexes in the lower extremities are generally exaggerated at first, but later on become diminished, and finally abolished. The reflexes upon one side may be more vigorous than upon the other. As to sensation, it is hard to reach definite conclusions, because of the patient's stupor. Sometimes there is well-marked cutaneous hyperæsthesia, probably referable to an implication of the spinal cord in the process.

The behavior of the pulse and temperature is interesting. The temperature is usually elevated, but often only to a slight extent—that is, varying between 100.5° and 102° (38° and 39° C.). Often the temperature falls quite low, only to rise again, the alternations being at irregular intervals. Exceptionally the temperature may remain high (104° F., 40° C.) most of the time. Toward the end there is usually a decided change in temperature, either upward or downward. In many instances there is a very low temperature before death; in two cases we have seen a temperature of 87.8° (31° C.). Or the temperature may rise to 106° (41° C.) or higher just before death. The pulse is often abnormally slow in the early stages of the disease, even numbering only 40 to 50 beats per minute. This is referable without doubt to the increased intra-cranial pressure. Later on the pulse becomes small and rapid. The transition may be very sudden. The vagus is at first irritated, and then paralyzed. The pulse is often irregular.

Respiration is generally moderately accelerated. If the breathing is very deep and rapid, we should always think of simultaneous miliary tuberculosis of the lungs. Toward the close of the disease the respiration often assumes the Cheyne-Stokes type: there is a long pause, followed by very superficial and gentle respiration, which gradually grows deeper and deeper, then diminishes again, and is succeeded by another complete pause. This symptom is always most ominous, for it indicates that the excitability of the respiratory center is already greatly impaired.

Symptoms referable to still other organs are few. Vomiting is rare in the later stages of the disease. The abdomen often presents a "boat-shaped" concavity, as the result of tonic muscular contraction, and is hard and tense. There is almost always constipation. The spleen may be somewhat enlarged. The urine sometimes contains a trace of albumen. On account of the drowsiness, it is usually voided in the bed or retained in the bladder. Almost invariably there is rapid marasmus.

The entire duration of tubercular meningitis varies somewhat, chiefly because of the varied length of the first stage. When the disease is once fully developed, the illness seldom lasts more than three to ten days longer. Frequently the illness is divided into three stages: 1. The stage of cerebral irritation, with headache, stiff neck, vomiting, and delirium; 2. The stage of cerebral compression, due chiefly to the hydrocephalus, and causing drowsiness, slowness of the pulse, paralysis of the oculo-motor nerves, hemiplegia, etc.; and 3. The paralytic stage, presenting deep coma, relaxation of the previously contracted muscles, accelerated pulse, and marked variations of temperature. Such a division is too diagrammatic to correspond accurately to the real phenomena, but it will, nevertheless, often aid us in getting a general idea of the course of the disease.

The termination of tubercular meningitis seems to be inevitably fatal. Sooner or later the patient loses consciousness completely, his pulse grows very small and rapid, his respirations irregular and intermittent (Cheyne-Stokes), his temperature, as we have said, either rises high or falls far below normal, and, finally, death is ushered in by a paralysis of all the vital functions. A few physicians have reported cases of recovery; but was the diagnosis correct? While we would by no means absolutely deny that recovery from tubercular meningitis might occur, it would certainly be very difficult to prove, in any particular instance, that such a thing had happened.

Tubercular Meningitis in Children.—The disease is so prone to attack children that it seems desirable to subjoin a few remarks about the peculiarities of the affection as observed in them.

Often the little patient is pale and weakly, with tubercular antecedents; but sometimes apparently healthy and vigorous children are attacked. Tubercular meningitis may be the sequel of measles, whooping-cough, or some other disease, which has occasioned the development of the tubercular process. Usually the severer symptoms are preceded by a rather long prodromal stage (a week or ten days), which often does not forebode the severity of the disease that is beginning. The child looks pale, has a poor appetite, grows thin, is restless in his sleep, is out of humor and cries during the day, is often tired and sleepy, does not play naturally, the tongue is coated and the bowels constipated. The second stage is generally ushered in by headache and vomiting. The headache is not very often violent; but children complain with remarkable frequency of pain in the abdomen and chest. The cause of this symptom is unknown. The pulse is almost invariably slow, often somewhat irregular and whirring (that is, as if it trembled beneath the finger), and it frequently undergoes surprisingly rapid changes in rate—for example, varying twenty or more beats inside of a few hours. Very early the child becomes dull and drowsy. Frequently it emits a peculiar deep sigh, or that sudden loud scream or "cephalic cry" which physicians long ago learned to recognize and fear. The symptoms referable to the cranial nerves and the nervous disturbances in the limbs are similar to those seen in adults. Strabismus is almost constant. Very often there are trismus, and a distinctly audible grinding of the teeth, most distressing to the by-standers. Trousseau laid weight upon the appearance of red spots (*taches cérébrales*) upon the skin after it has been mechanically irritated; but these have no diagnostic value. They are due to increased reflex action, and are seen in all sorts of acute diseases. The fever is irregular throughout and generally, as in adults, not very high, 100° to 102° (38°–39° C.). Respiration is usually rapid, and often is irregular.

The change from bad to worse is almost always announced by a rapid increase in the pulse-rate, to 160 or 200. The child becomes completely comatose. Very often there are repeated epileptiform convulsions, either universal or affecting single extremities. Death is usually preceded by a decided rise in temperature.

Diagnosis.—When the symptoms are pronounced, the diagnosis of meningitis is easy, and we have merely to determine just what variety is before us. That the disease is due to tuberculosis is never to be ascertained by means of the meningeal symptoms themselves, but is rendered possible only by the ætiology, if that be discoverable. Here, as in all tubercular diseases, we have chiefly to regard (1) heredity and (2) the evidence of previous or existing tubercular affections in other parts of the body. Under this second head are to be considered scrofula, diseases of the bones and joints, pulmonary tuberculosis, pleurisy, and tuberculosis of the genitals or of the choroid. If our search be unsuccessful, we may sometimes get a hint of the truth from the general appearance of the patient: for example, he may be pale, or narrow-chested. And, again, the absence of trauma, aural disease, or epidemic meningitis will make tuberculosis more probable.

In its early stages, or when it varies from the usual course, tubercular meningitis may be very difficult to diagnosticate. This is particularly true when the patient is a child. The early malaise and vomiting are treated as "ordinary gastric catarrh" until the grave cerebral symptoms disclose the mistake in diagnosis. In such cases we should be careful not to disregard the initial slowness and irregularity of the pulse. This alone should make our prognosis guarded. The fever may be prominent at the commencement, and tempt us to call the case one of incipient typhoid fever; and, indeed, the correct diagnosis is often impossible until the disease develops further. In regard to this, see the preceding chapter on purulent meningitis, where the exclusion of severe septic diseases and of uræmia is also considered.

Before the autopsy we must remain in great uncertainty as to the number and distribution of the tubercles, the existence of a large effusion into the lateral ventricles, etc. We are often amazed at the post-mortem examination by the apparent insignificance of the lesions. Paralysis of the cranial nerves (eyes, face) implies that the base of the brain is gravely affected. If such symptoms are absent, and there are mental disturbances, and symptoms of motor irritation displayed in the extremities, we are led to infer meningitis of the convexity. If the nervous disorder be mainly unilateral, we may assume that one hemisphere is more affected than the other.

Treatment.—However hopeless the prospect, we are nevertheless bound to employ all the remedies at our command, as in other forms of meningitis. Above all, we should be thorough in applying ice to the head, and we may also try inunctions of iodoform ointment to the shaven scalp, and lukewarm baths, with douching. The inunction of mercurial ointment (especially on the head, neck, and back) has also been recommended. The most common internal remedies are calomel—half a grain to a grain (gramme 0.03–0.05) for a child every two hours—and infusion of senna. Iodide of potassium may also be freely given, fifteen grains daily to a child, and two or three times as much to an adult. Whether it does any good is extremely doubtful. If the patient is very restless, narcotics are indispensable. Stimulants are often given in the last stage of the disease, but generally without effect.

About prophylaxis, we need merely refer to the general statements on page 254, in regard to prophylaxis from the various tubercular diseases.

CHAPTER IV

THROMBOSIS OF THE CEREBRAL SINUSES

Ætiology and Pathology.—The sinuses of the dura mater sometimes present a thrombosis, under circumstances similar to those which induce the same process in other veins. The most frequent occasion for such thrombosis is marasmus, however brought about, with the accompanying feebleness of circulation. This is the explanation of those not very rare cases found among wretched and ill-nourished children in the first year of life, and also in patients with phthisis, cancer, etc. We must note in particular that in severe anæmic conditions (especially in chlorosis) sinus thrombosis is comparatively common.

Half way between marantic thrombosis and the inflammatory variety immediately to be described, come those cases which are seen in typhoid fever and other severe acute infectious diseases. Here the specific virus apparently plays an important part (just as in thrombosis of the femoral vein), although very likely the cardiac weakness also promotes the thrombosis.

Genuine inflammatory thrombosis (thrombophlebitis)—that is, thrombosis in connection with real phlebitis of the sinus—is almost always due to the extension of inflammation from some neighboring part. The most fruitful cause is suppuration in the petrous portion of the temporal bone, the result of otitis or caries. This spreads to the walls of the transverse or petrosal sinuses, which are close by. Also wounds, necrosis, or other affections of other cranial bones may excite thrombosis; likewise, although seldom, deep-seated inflammation of the soft parts of the head and face, large furuncles or erysipelatous abscesses, may produce the same result. The reason why sinus thrombosis may arise from the causes last mentioned lies in the fact that the external veins of the skull are also connected with the sinuses of the dura mater by means of the emissary veins of Santorini. It is easy to understand that purulent thrombophlebitis of the cerebral sinuses is often found associated with purulent meningitis or brain abscess.

Thrombosis due to marasmus is most frequently found in the superior longitudinal and transverse sinuses, while the inflammatory variety usually occupies either the transverse, petrosal, or cavernous sinuses. Of course the thrombus may grow out from its original sinus into neighboring ones. Important clinical symptoms are caused by secondary venous stasis in the veins which empty into the occluded sinus. These symptoms are most pronounced when the longitudinal sinus is affected; objectively, we find the meningeal veins which lie on the surface of the brain distended and tortuous; and often there are extensive meningeal ecchymoses. Even the cerebral parenchyma beneath shows distinct evidence of passive congestion, and minute capillary hæmorrhages have been repeatedly found in it.

Symptoms.—In some instances moderate thrombosis of the cerebral sinuses has been found post mortem, although there had been no symptom suggesting it before death. In other cases the thrombosis does excite undeniable cerebral disorder, but the symptoms are so general and ambiguous that the most we can do is to suspect the existence of the clot without being at all certain about it. Sinus thrombosis in marantic children usually causes coma, stiffness of the neck and back, strabismus, nystagmus, and sometimes clonic spasms in the face and limbs. The symptoms in adults are similar, comprising headache, drowsiness, occasionally delirium, sometimes coma, and varying symptoms of irritation or of paralysis in the distribution of the cranial nerves (nystagmus, strabismus, trismus) and in the extremities. But even all these symptoms are insufficient to make the diagnosis certain. They must be re-enforced by certain other phenomena more distinctly referable to the peculiar circulatory disturbances occasioned by the thrombosis. Occlusion of the cavernous sinus sometimes excites well-marked symptoms of stasis in the ophthalmic veins. Thus the retina may be seen through the ophthalmoscope to be passively congested, there is œdema of eyelids and the conjunctiva, the eyeball is unusually prominent, and the frontal vein is distended. In case of an inflammatory thrombosis, the periphlebitic swelling may cause symptoms in the distribution of the neighboring nerves, especially paresis of the oculo-motor or abducens, or trigeminal neuralgia. In thrombosis of the transverse sinus an œdematous swelling is occasionally seen behind the ear, near the mastoid process. If the clot projects into the petrosal sinus or even actually into the internal jugular, the lower part of this vein collapses. And inasmuch as the external jugular can empty itself with unusual ease into the unfilled internal jugular vein, the external jugular is also affected and becomes less prominent upon the affected than upon the normal side. Sometimes it is even possible to feel the thrombus in the internal jugular. Such thrombosis causes pain and swelling in the neck on the affected side. When the superior longitudinal sinus is blocked up, there are symptoms of nasal engorgement (epistaxis), and distention of the veins about the temples, which veins are connected with the longitudinal sinus by emissary veins. We must confess, however, that all these special symptoms are comparatively rare, and often difficult of demonstration even when present.

Primary thrombosis of the transverse sinus with extension of the clot into the venæ magnæ Galeni, thus causing internal hydrocephalus, has been repeatedly observed in the chlorotic. The clinical picture consists of the rapid onset of severe cerebral symptoms (headache, vomiting, somnolence), which rapidly progress to deep sopor and lead to death in a few days. When unilateral paralysis and symptoms of motor irritation occur (choreic twitchings, epileptiform attacks), they are due in part to hæmorrhages from stasis in the brain substance.

The symptoms become more complex where there is a suppurative phlebitis, because there are usually pyæmic symptoms as the disease progresses. Thus, there may be rigors and high fever, pulmonary abscess, suppurative arthritis, etc. We have already mentioned the combination of thrombosis of a sinus with purulent meningitis or brain abscess.

The prognosis is almost always bad, both because of the nature of the causative disease and because of the grave cerebral derangement or the secondary pyæmia. Spontaneous recovery may occur, but it is rare. The nervous symptoms may last at times only a week or two, but they may extend over several months.

Treatment in most cases must be purely symptomatic. Only when a purulent sinus thrombosis follows ear disease can surgical interference give very satisfactory results. The details of such treatment are to be found in the text-books of operative aural surgery.

SECTION II

DISEASES OF THE BRAIN-SUBSTANCE

CHAPTER I

DISTURBANCES OF CIRCULATION IN THE BRAIN

(Cerebral Hyperæmia. Cerebral Anæmia)

It is presumable that so sensitive an organ as the brain is much influenced even by slight disturbances of circulation; but as yet we have comparatively little knowledge of the production and character of such disturbances, their very existence being very difficult to demonstrate. There are many instances where marked cerebral symptoms justify the assumption that the brain is in some abnormal condition, where yet there are many arguments against any marked anatomical lesion. Here we surmise that there is some circulatory derangement, although we have no actual and direct arguments to rely upon. For example, we refer to this cause certain cases of headache, of sensations of pressure in the head, of vertigo, of general hyperæsthesia, and of that protean and nevertheless easily distinguishable disease known as cerebral neurasthenia (*q. v.*). At present, however, we can not determine how far circulatory disturbances are actually concerned in these cases, or of what kind they are, or whether there may not be purely functional disease of the brain entirely independent of changes in the blood-vessels.

Certain groups of cerebral symptoms, which come on in paroxysms, seem the most clearly referable of all to circulatory disturbance. There can hardly be a doubt that the phenomenon known as fainting or syncope is due to sudden cerebral anæmia. As is well known, fainting is usually the result of a clearly demonstrable cause. Frequent and familiar causes are emotional excitement, terror, unusual psychical impressions (like the sight of blood), the influence of great heat, or great physical strain, as by long standing. The condition of the stomach has certainly a great influence in many cases. There are many persons who, if they go long beyond the usual time without eating (particularly if they go without breakfast), are very liable to syncope. Some individuals are especially subject to fainting fits. Such persons are often slight and anæmic (for example, convalescents), but some are in appearance robust and vigorous. Many children are subject to fainting.

The immediate cause of the cerebral anæmia in all these cases is doubtful. Mental excitement is usually supposed to lead, in fainting, to a contraction of the minute cerebral arteries. It is not, however, impossible that in these cases also, as in others, sudden cardiac weakness is one factor, although, if so, it is strange that we never see a trace of cyanosis. Where the attack is apparently connected with unusual conditions of the abdominal organs we are reminded of the relations of the splanchnic nerve to the heart (Goltz's experiment of beating the belly of a frog), and of the possibility that the brain might be left anæmic if the abdominal vessels suddenly dilated and absorbed a large proportion of the whole blood-supply.

The symptoms of an ordinary fainting fit are known to every one. There are usually certain prodromata. The person begins to "feel badly." Dizziness comes on, the senses are confused, the patient gapes, the ears ring, there are spots before the eyes or total darkness, the floor seems to move, and surrounding objects begin to spin around. All this is almost always accompanied by nausea, and sometimes there is actual vomiting. If the person can lie down promptly the attack is sometimes averted without complete loss of consciousness. Otherwise there is unconsciousness for a time varying from some minutes to even a half-hour or longer. What is most noticeable, even in the beginning of the attack, is the pallor which overspreads the face and often becomes extreme, and is the visible expression of the coincident cerebral anæmia. Very often the face and body are bathed in cold perspiration. The pulse is usually small and rapid.

There is no real danger in an ordinary attack. The most important therapeutic measure is to lay the patient horizontal as soon as possible, to favor the return of blood to the brain. Mild stimulants should also be employed; the face should be sprinkled with cold water, the temples rubbed with vinegar or cologne-water; brandy or wine should be administered. We can overcome a tendency to fainting fits only by strengthening the constitution.

The results of chronic cerebral anæmia are observable when the cerebral condition is part of excessive general anæmia. Almost all cases of chlorosis, pernicious anæmia, and acute anæmia from loss of blood (as in ulcer of the stomach) display most plainly the symptoms of cerebral anæmia. The phenomena are essentially the same as in syncope, only less in degree. Consciousness is maintained, except in the worst cases. A sort of persistent drowsiness, however, often attended by frequent gaping, is one of the most constant symptoms. The patient is most distressed, as a rule, by loud tinnitus aurium, persistent nausea, and sometimes by obstinate headache. All these symptoms are aggravated, and often increase to an actual attack of fainting, if the patient sits up in bed, and are least marked when he lies as quietly as possible in a horizontal position. The treatment of this condition is of course identical with that of the causative disease and the general anæmia.

Cerebral hyperæmia, like cerebral anæmia, may be either chronic or paroxysmal. Of the chronic variety we know almost nothing. It seems doubtful, to say the least, whether there is really a "general plethora," or whether the headaches and vertigo of which "full-blooded" persons complain are due to hyperæmia of the brain. Nor have we any direct proof that the cerebral symptoms resulting from chronic poisoning (alcohol, tobacco, etc.), or from persistent mental over-exertion, are brought about by hyperæmia, as some assume, rather than by functional disorder of the nervous elements themselves.

These conditions, which are called attacks of "cerebral congestion" or "rush of blood to the head," are often referred to the sudden onset of cerebral hyperæmia. There is a more or less sudden appearance of general excitement, with a sensation of warmth in the head and neck, strong pulsation of the carotids, a red face, general hyperæsthesia and irritability, headache, vertigo, tinnitus, spots before the eyes, and nausea. An attack lasts half an hour to an hour. In severe cases there may be maniacal excitement, or there may be stupor and drowsiness and other symptoms of lowered intellectual activity, resembling a slight apoplectic attack (see a later chapter). The special cause of such attacks, however, is usually obscure. It is, indeed, very probable that in these attacks vaso-motor disturbances play a part (dilatation of the cerebral vessels from vascular paralysis or from irritation of the vaso-dilator nerves); but it is very questionable whether these vascular changes are really the primary diseased state, rather than the result of some other different morbid condition. Possibly we have to do sometimes with epileptic states, or more frequently with one manifestation of general nervousness, neurasthenia or hysteria (*q. v.*) with primary mental disturbances (anxious ideas, etc.).

In treating "congestion" we should keep the patient as quiet as possible, with head and shoulders raised; and, secondly, we should endeavor to draw the blood away from the brain. This purpose will be served by hot foot-baths, sinapisms applied to the chest and the calves, and such purgatives as senna or colocynth. It is also beneficial to apply cold to the head. In a severe case it is proper to put leeches to the temples or the mastoid processes.

To prevent, as far as possible, the recurrence of the attacks, we must have regard to the whole mental and physical constitution of the patient. We may mention, as of chief importance, diet (no alcohol) and a course at some watering-place, or cold-water treatment.

CHAPTER II

GENERAL PRELIMINARY REMARKS UPON THE LOCALIZATION OF CEREBRAL DISEASES

(*Topical Diagnosis of Cerebral Lesions*)

THE physiological relations of the brain are such that the symptoms of cerebral disease are determined to a greater extent by the locality than by the nature of the lesion. If, for example, there arises at any place a break in the continuity of the cerebral motor tract, the result, as we already know (see page 831), is hemiplegia upon the opposite side of the body. The result is precisely the same, whether the interruption be due to a hæmorrhage, an abscess, a new growth, or an embolic softening. If in any way the function of the motor fibers is suspended, then the necessary sequence in every case is a paralysis of definite extent and definite characteristics. Much the same may be said of many other symptoms which appear when there is a lesion of one or more definite places, but which are never referable to a special abnormal process, regardless of the portion of brain thereby affected.

However self-evident these simple statements may appear, it required a long time for them to gain universal acceptance among physicians. The chief cause of this was the conception entertained by the older physiologists in regard to the functions of the brain. Flourens, in 1842, taught that functionally all parts of the cerebrum were alike, and therefore any one part could act vicariously for any other; and this view had numerous adherents among physicians, as well as physiologists. It was nevertheless experience at the bedside and the autopsy-table which first led to observations and discoveries irreconcilable with this view. Above all, it was the lesions found in aphasia which forced men to localize one particular cerebral symptom as due to an affection of one particular spot in the brain. In 1861 Broca announced that the appearance of aphasia is always due to a lesion of the left third frontal convolution; and this was the starting-point of the doctrine of localization in general. Nine years later (1870) appeared the famous treatise of Fritsch and Hitzig detailing successful attempts at excitation of the surface of the brain in animals, and thus overthrowing the old idea that the gray cortical substance could not be irritated. It was shown that excitation of certain places in the cortex is followed by muscular contractions in well-defined portions of the opposite side of the body, so that we are justified in assuming the existence of a number of cortical centers, which are quite limited in extent. These results were soon confirmed by numerous observations in cerebral pathology in man; and to-day our information about the motor functions of the cerebral cortex forms the best-known portion of the doctrine of cerebral localization. Of late years successful work has been done in this exceedingly difficult field by Meynert and Flechsig among anatomists; Ferrier, Munk, Goltz, and other physiologists; and such pathologists as Charcot and his pupils, Nothnagel, Wernicke, Hughlings Jackson, Horsley, and others. It is true that we are only just beginning to know something about the subject. There are numerous contradictory views asserted, and numerous questions unanswered. The following summary, therefore, is to be regarded merely as expressing the prevailing opinions now existing. Much in it will surely be altered in the course of time; but still this doctrine of special localization of the various cerebral functions marks out in general outlines the only foundation upon which we can hope to erect a system of cerebral pathology and diagnosis. In the following sketch we shall, for practical reasons, put the results of clinical observations upon man in the foreground, and merely speak incidentally of the corresponding experimental achievements. This will be the quickest way to gain acquaintance with the practical points in the diagnosis of what Griesinger called the "focal diseases"; and then, when we take up the separate varieties of cerebral disease, we shall have these general remarks to refer to.

1. The Motor Region of the Cortex Cerebri

Clinical observation and the results of experiment both teach that a part of the cerebral cortex is distinct from the rest, inasmuch as it is the exclusive seat of motor functions. This "motor region" (*vide* Figs. 169 and 170)

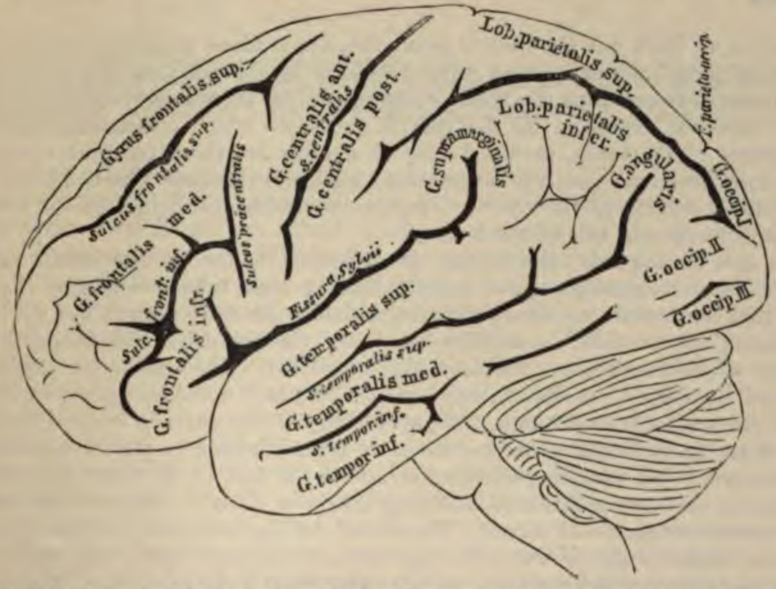

FIG. 169.—Lateral aspect of the brain (from ECKER). The gyri and lobules are in Roman type, the sulci and fissures in italics.

is made up chiefly of the two central convolutions (*gyri centrales anterior et posterior*, in Fig. 169), and the paracentral lobule (*vide* Fig. 171), which lies on the median surface of the cerebrum. To a slight degree the motor region also embraces the neighboring portions of the frontal convolutions and the upper parietal convolutions. It is also anatomically different from the other regions of the cortex, as Betz was the first to point out, for it alone possesses certain large pyramidal ganglion-cells, which must certainly be regarded as the same motor ganglion-cells from which the fibers of the pyramidal tract arise (*vide supra*, page 829). However extensive the destructive processes which attack other parts of the surface of the brain, provided they do not involve these particular convolutions, they cause no paralytic symptoms; while all diseases which destroy any considerable portion of the "motor" region inevitably result in a motor disturbance on the opposite side of the body.

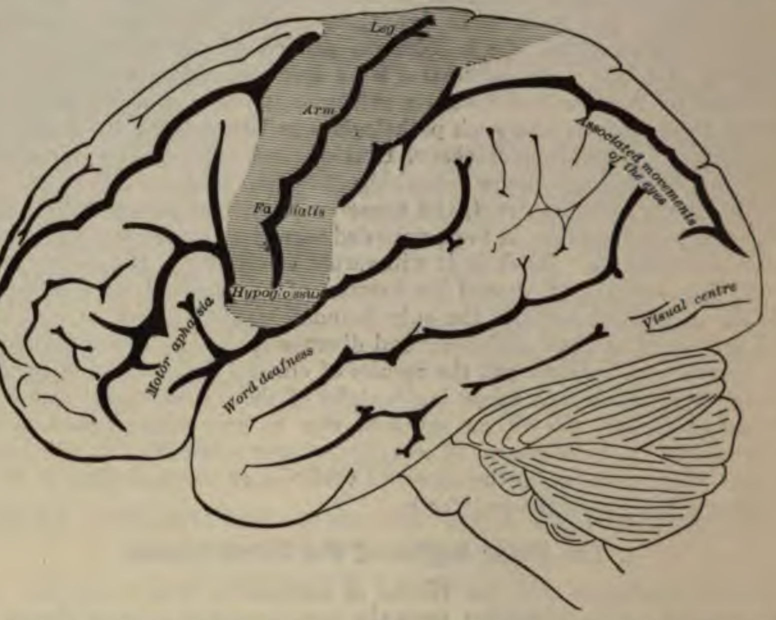

FIG. 170.—Lateral aspect of the brain (after ECKER). The motor region of the cortex, consisting of the anterior and posterior central convolutions, as well as of the paracentral lobule shown in Fig. 171, is shaded.

We can distinguish still further in different regions of the cortex special centers for the various groups of muscles, but we must take special note that the individual centers are not sharply distinguished from one another, but that they overlap. Thus the different centers which are associated have an intimate connection, which also seems necessary physiologically. The center for the movements of the facial muscles (facial region), lies, as it would seem, at the lower end of the central convolutions, and particularly of the anterior central convolution. Near by, probably somewhat lower, is found the center for the movements of the tongue. This center probably extends to the foot of the third frontal convolution, so that on the left side it is accordingly in close proximity to the special motor-speech center (*vide infra*). The center for the movements of the arm lies somewhat higher than the center for the facial, occupying roughly the middle portion of the anterior and probably the posterior central convolutions. The center for the lower extremity is found partly in the uppermost portions of the central convolutions, but apparently lies for the most part in the paracentral lobule. The center for the movements of the trunk probably lies in front of the leg center in the most posterior portion of the upper frontal convolution and also on the median surface of that convolution (the so-called marginal gyrus, the median portion of the upper frontal convolution).

Within these great cortical centers for the chief portions of the body (leg, arm, face, tongue) there is a much more accurate localization for individual groups of muscles, and perhaps even for individual muscles. This fact has been established especially by the interesting experiments of excitation of the cortex of monkeys by Ferrier, Horsley, and others. Pathological observations and occasional attempts at excitation of the cortex which have been practiced on man after trephining or in extensive cranial defects arising from injury or disease, have shown that the conditions in man are in general quite the same as in the higher apes. Within the leg center lie special centers for the toes and foot, chiefly above and especially in the posterior central convolution. Then come the centers for the movements of the knee, and then those for the movements of the hip. In the paracentral lobule are found chiefly the centers for the thigh and the pelvic and glutæal muscles. Within the arm center and for the most part above—that is, toward the leg center—are the centers for movements of the shoulder and elbow. Farther downward come the centers for the movements of the hand and fingers, chiefly in the anterior central convolution. The separate position of the center for the thumb is especially noticeable; this is in the posterior central convolution, adjoining the centers for the facial muscles. Farther downward lie the centers for the upper facial territory above the centers for the muscles of the mouth and lips. In the lowest part of the central convolutions and in the adjacent portions of the two lower frontal convolutions lie the motor centers for movements of mastication (the centers for closing the mouth are said to lie above those for opening the mouth), and for the movements of the tongue and vocal cords. The exact position of these centers in man is not yet definitely known. The centers for the associated movements of the eyes to one side seem to be quite scattered. We elicit these movements (usually coincident with a corresponding

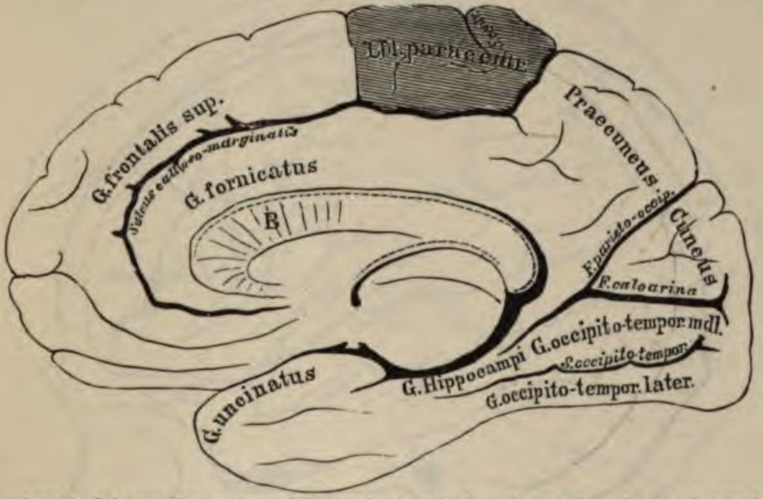

FIG. 171.—Aspect of the median surface of the cerebrum, which is shown when the two hemispheres are separated from each other by a sagittal section. B. Corpus callosum. The differences in the type have the same meaning as in Fig. 169. The paracentral lobule, as a part of the motor region of the cortex, is shaded. (Copied from ECKER, only the paracentral lobule is made more sharply prominent than in the original.)

turning of the head) on irritation of the posterior portion of the frontal lobe adjoining the region of the centers for the arm and face; but, on the other hand, pathological observations favor the existence of a center for associated movements of the eyes in the region of the angular gyrus (*vide infra*). This multiple localization is perhaps explained by the fact that the movements of the head and eyes are closely associated both with all complicated activities of the hand and with the functions of sense (listening, looking at objects). The diagram on page 1073 (Fig. 173), from a drawing by C. K. Mills, gives a review of all these details.

The topical diagnosis of diseases in the motor region of the cerebral cortex is based upon the following considerations:

In the first place, we have already remarked (page 831) that the relative positions of the motor centers for the face, arm, and leg are such as to allow readily of isolated paralysis of any one of these portions of the body—that is, "monoplegia." In fact, we already possess a long series of observations where circumscribed lesions in the motor area of the cortex produced paralysis of one side of the face, or of one arm or leg, and of no other part. Such paralysis is termed monoplegia of the face, the arm, or the lower extremity. It follows, from what has been said, that even during life we can state, with considerable accuracy, the spot on the surface of the brain where the disease must be situated. Still more frequently, a combined paralysis of two portions of the body is to be observed as a result of cortical lesion; the commonest is a simultaneous paralysis of the arm and face; more rarely we see the arm and leg paralyzed together. On the other hand, we may feel certain, from the position of the motor centers, that no single center of disease could paralyze simultaneously the leg and the face, while the arm escaped injury. As a matter of fact, no such combination has ever been observed. It is also worthy of note that the muscles that always act bilaterally (frontal muscles, muscles of mastication, eye muscles, vocal-cord

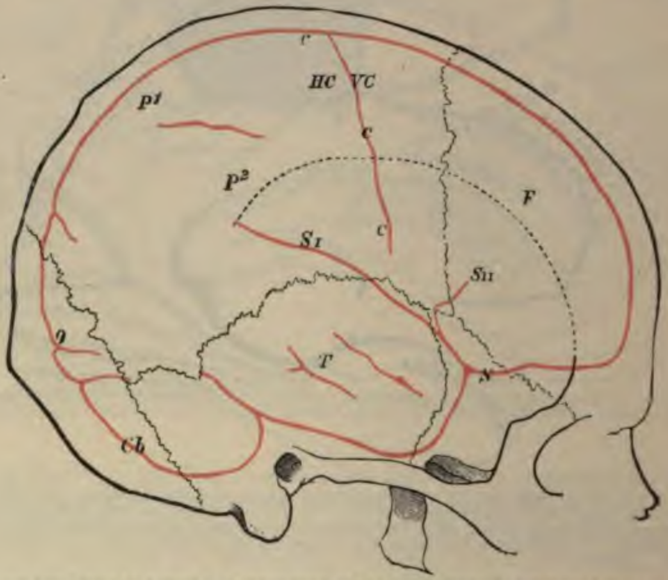

FIG. 172.—(Drawn according to ECKER.) Explanation of the topographical relations between the surface of the brain and the skull. *c.* Fissure of Rolando. *HC.* and *VC.* Posterior and anterior central convolutions. *S. SI* and *SII.* Fissure of Sylvius. *P^1 P^2.* Upper and lower parietal lobes. *O.* Occipital lobe. *Cb.* Cerebellum. *T.* Temporal lobe. *F.* Frontal lobe.

muscles, etc.) have also a bilateral innervation. A unilateral focus of disease in any center, therefore, does not cause paralysis of these muscles.

Besides this limitation of the paralysis to one portion of the body just discussed, localized disease of the cortex has another very characteristic feature. In it the symptoms of irritation of the motor centers are noticeably frequent. There are tonic and clonic spasms, which, like the paralysis, not infrequently affect one arm, or the arm and half the face. Sometimes, however, they involve the entire half of the body. These paroxysmal spasms are termed "cortical epilepsy," partial epilepsy, or Jacksonian epilepsy; for the movements are just the same as in genuine epilepsy. Numerous cases of disease have taught us that such circumscribed epileptiform attacks occur only in affections of the motor cortex. They furnish still more definite information as to the precise locality of the lesion; for spasms in the distribution of the facial nerve imply that mainly the lower third of the central convolutions is affected; of the arm, the middle third; and of the leg, the upper portions of the same. If we have the opportunity to observe such attacks of partial cortical epilepsy accurately, we can almost always determine that the twitching begins in quite a limited muscular territory—for example, in the face, the thumb, etc. The attack often begins with a slight sensory aura (paræsthesia in the part of the body affected); then follows a tonic muscular contraction which soon passes into a decided clonus. From this point the twitchings extend, and always in the order corresponding to the local arrangement of the different centers which lie next to one another. It is therefore always of importance in diagnosis to determine in what group of muscles the twitchings begin, because we can thus decide that the corresponding cortical center is the chief seat of the morbid affection. In the muscles innervated bilaterally (the frontal muscles, the eye muscles, the muscles of mastication, etc.) the convulsions also occur bilaterally. In very severe attacks the spasm finally involves the other half of the body, the irritation being conveyed through the fibers of the corpus callosum to the corresponding cortical motor centers of the

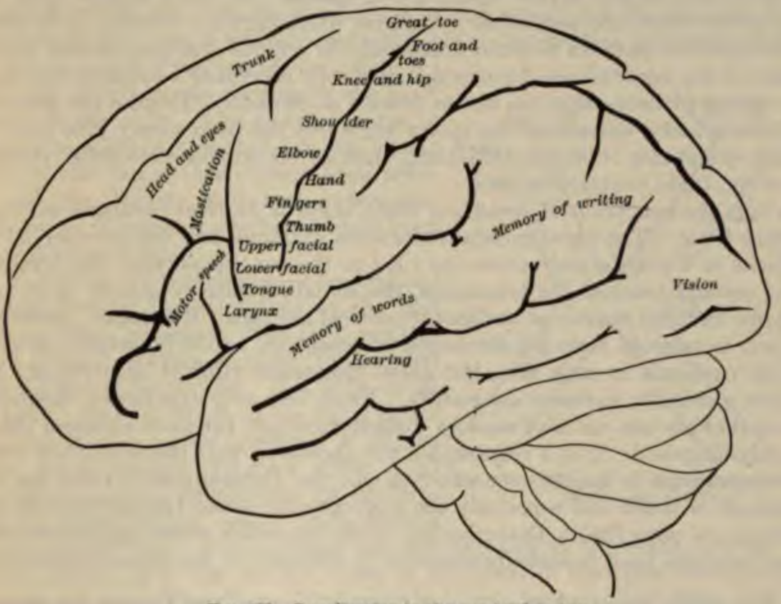

FIG. 173.—Localization in the cerebral cortex.

other hemisphere. The twitchings usually persist the longest in the muscles first affected. In the attacks of circumscribed cortical epilepsy there is no disturbance of consciousness, but if the convulsion extends to both halves of the body, there is usually loss of consciousness, or at least a decided clouding of consciousness. The individual attacks last only a short time—about one or two minutes. After they cease there usually remains a very transitory motor weakness or even complete paralysis in the muscles most affected, which is in a degree the indication of exhaustion of the affected center. Temporary disturbances of speech are often associated with right-sided Jacksonian epilepsy. The relation of convulsions to permanent paralysis varies greatly. Often, for example, when there is hæmorrhage into the central convolutions, violent unilateral convulsions come on simultaneously with the paralysis. In the case of tumors and other lesions which develop slowly, partial epileptiform spasms will often appear quite a long time before there are persistent symptoms of paralysis. Finally, it is not rare for epileptiform attacks to occur repeatedly in regions that are already paralytic. Either of the occurrences described in the two preceding sentences are particularly strong evidence that the cortex cerebri is diseased. Besides the pronounced epileptic attacks, disease of the motor region of the cortex may give rise to less violent symptoms of motor irritation, such as occasional twitching, rhythmical twitching, tonic contraction, etc.

About the condition of sensation when there is cortical paralysis we know as yet very little. The experimental researches of Munk and the anatomical investigations of Flechsig and others have led to the conclusion that the termination of the sensory tract of the lemniscus, the so-called sensory sphere, is in part in the same cortical region as the motor cortical centers. We might, therefore, be inclined to suppose that a disturbance of sensation would invariably accompany cortical paralysis in man also, but about this point clinical observations do not yet give perfectly harmonious results. From our own experience, however, we believe that we can say that sensory disturbances are far more common than was formerly supposed. It is a remarkable fact, however, that the sensibility for pain and temperature is hardly ever affected, but the disturbances involve the simple sensations of touch and especially the stereognostic sense (recognition of objects by touch, see page 795). Disturbances of the muscular sense and ataxic disturbances have also been repeatedly observed in diseases of the motor cortical region.

2. The other Parts of the Cortex Cerebri, except the Center for Speech

1. *Frontal Convolutions.*—Unilateral disease of the anterior portion of the brain may be quite extensive without causing notable disturbance of any kind. Certainly the greatest part of the upper two frontal convolutions have no motor functions. It is, however, maintained that the posterior portion contiguous to the anterior central convolution, called the foot of the frontal convolutions, does contain motor centers. We have stated above (page 1071) that the motor centers for the muscles of the trunk are situated in the upper frontal convolution, especially on its median aspect. This is the reason why a decided swaying of the trunk in walking and in standing (just as in cerebellar ataxia, *vide infra*) has been repeatedly observed in diseases of the frontal lobe, especially in tumors. The third (lowest) frontal convolution on the left side has, as we shall soon see, an undoubted connection with the motor processes of speech.

There is a quite generally accepted opinion that the cortex of the frontal portion of the brain is to be regarded as the "seat of the higher psychical functions." Some few cases are on record where extensive bilateral lesions of these parts had for their only manifestations mental symptoms (loss of mental functions, changes in character, the so-called morbid wittiness, *Witzelsucht*, etc.) without any other disturbances. In general paralysis also, and in other forms of dementia, the predominance of atrophy in the frontal lobe is very striking, so that it is actually very probable that the frontal lobe has a close relation to the higher mental processes. Nevertheless, we can not emphasize too much the fact that, at present, we have no certain knowledge about the precise form of this relation or of the nature of the physical functions.

2. *Parietal Convolutions.*—We know but little about the functions of the cortex of the parietal lobe, and the symptoms which might indicate disease of that portion of the cerebrum. According to Flechsig, a large part of the sensory tract of the tegmentum ends in the parietal cortex. We might therefore expect decided disturbances of sensibility in diseases of the parietal lobe, but unequivocal clinical and anatomical evidence in support of this hypothesis is still almost wholly lacking. Observations on the occurrence of motor disturbances in diseases of the parietal cortex are somewhat more numerous. Centers for associated movements of the eyes seem to be situated in this region (in the angular gyrus). In diseases of this region we may often observe an interesting symptom of irritation, the so-called "conjugate deviation"—that is, the simultaneous turning of the head and eyes to one side. The angular gyrus is also said to contain a center for the ocular portion of the facial (orbicularis oculi muscle) and the levator palpebræ superioris; but of course these statements have not been universally accepted.

3. *Occipital Convolutions.*—The clinical and experimental investigations of the last few years have all shown that the occipital portion of the cerebrum contains the cortical center for visual sensations. It is here, in all probability, that the fibers of the optic nerve terminate in the cortex cerebri. A glance at the accompanying diagram (Fig. 174) will make it easy to understand the disturbances of vision which result from lesions of the occipital lobe. *L* represents the left eye and *R* the right, *Ch* the optic chiasma, where, as is now certain, some of the fibers of the optic nerves cross to the opposite side. The fibers (distinguished by a broken line) from the outer or temporal half of each retina extend, without crossing, into the corresponding optic tract (*Tract. opt.*), while those from the inner or nasal half of the retina cross over in the chiasma. The right occipital lobe, for example, comes in this way to receive the fibers from the outer half of the right retina and from the inner half of the left. If the right occipital lobe becomes disorganized, then the images formed upon the parts of the retinæ just named, belonging to the left half of the field of vision, are unperceived. With each eye the patient sees only such objects as lie in the right half of his field of vision, and is blind to all that lies upon his left. This sort of visual disturbance, where each eye becomes blind to the same "homonymous" portions of the field of vision, is termed hemianopsia, or hemiopia. A lesion of the right occipital lobe causes, therefore, left-sided hemianopsia, and, *vice versa*, destruction of the left occipital lobe entails right-sided hemianopsia.*

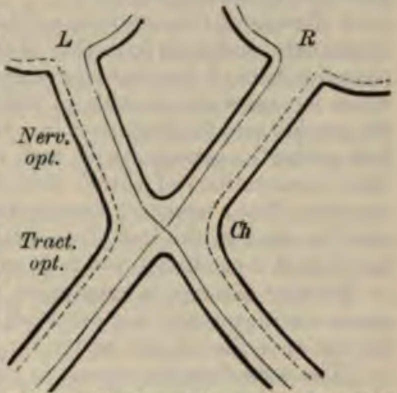

FIG. 174.—Diagram of the course of the optic fibers in the chiasma.

[* When hemianopsia is due to a lesion between the chiasma and the pulvinar and geniculate body (second optic neurone, *vide infra*, page 1089), the pupil does not react when light is thrown on the blind half of the retina, although it does react when light is thrown on the sound half (Wernicke's "hemiopic pupillary reaction"). When hemianopsia is due to a lesion between these ganglia and the cuneus (third optic neurone) this hemiopic reaction does not occur.—K.]

The anatomical evidence is still very contradictory as to the precise localization of the visual center. Disturbances of vision are found most constantly in diseases of the cuneus and the first occipital convolution. In this region the sensations of light as such seem therefore to take their rise. In diseases of the medullated layers of the occipital lobes and their vicinity (angular gyrus)—that is, of the connecting tracts between the occipital cortex and other cortical regions—we sometimes observe the interesting symptom of so-called "soul blindness" (Munk). This disturbance is when the patient can still see objects, but can not recognize what they signify. He no longer knows what to do with them, or he uses them in the most utterly wrong way. He no longer recognizes his acquaintances by their appearance, but only by the sound of their voice. In diseases of the left occipital lobe the connections between the optic impressions and ideas of language may sometimes be solely affected; then there develop optic aphasia and alexia; the patient recognizes objects, but he can not name them. He can not read or copy, but he can write very well spontaneously and from dictation (compare the following section).

4. *Temporal Convolutions.*—The relation of the temporal lobe to hearing is apparently analogous with that of the occipital lobe to vision. Destruction of the posterior half of the superior temporal convolution, according to some observations, is apparently capable of causing actual deafness in the opposite ear; but the trouble usually disappears in a very short time. Each auditory nerve is therefore probably connected with both temporal lobes. It is much more certain than these observations of actual disturbance of hearing, that diseases of the [left] superior (first) temporal convolution cause so-called word deafness ("soul deafness")—that is, the loss of comprehension of spoken language. We will return again to this condition, which is usually associated with motor aphasia.

We may observe, in conclusion, that the olfactory nerve probably ends in the gyrus uncinatus and the parts adjacent (Fig. 171, page 1071), so that we may assume that that is the center for smell.

[The sensori-motor centers in the Rolandic region, and the special centers for vision, hearing, and smell which have just been described, occupy only about one third of the area of the cerebral cortex. Flechsig has found that the fibers connected with these areas assume their myeline sheaths at an early period of fœtal life, while the fibers connected with other regions become myelinized much later. These other regions he regards as association centers, and believes that they are especially concerned in the higher intellectual functions. The anterior association centers, embracing the anterior portions of the frontal convolutions and the gyrus rectus, are supposed by him to be the chief centers for ideas of personality. The posterior association centers include the præcuneus, the parietal convolutions (except the posterior central convolution), all portions of the occipital cortex except the visual area, and all portions of the temporal cortex except the auditory and olfactory areas. These centers are supposed to be the centers for ideas referring to the external world. The island of Reil and the parts adjacent form the middle association center. Flechsig's hypotheses have been strongly opposed, especially on the anatomical side. It is doubtful whether the projection and the association centers in the cortex can be so definitely distinguished, but the hypotheses thus advanced have also some clinical and pathological support, and they present a rich field for further investigation.—K.]

3. The Centers of Speech and the Disturbances of Speech

(Aphasia and Allied Conditions)

The Various Forms of Aphasia, and their Anatomical Localization.—As we remarked at the very beginning of this chapter, the peculiar derangements of speech observed in many cerebral diseases were the first symptoms which were found to be caused by a distinctly localized cerebral lesion. For the better understanding of this extremely interesting subject, it is necessary that we should enter somewhat minutely into the processes connected with normal speech.

Incitement to speech—that is, to the oral expression of our thoughts to others—comes either from an internal impulse or from external causes which excite this impulse. Speech always requires internal mental activities—the presence of ideas and their transformation into that which we wish to communicate by speech. Where there are no conceptions there can be no words. The demented person is silent, because, like the newborn infant or the brute, he has nothing to say; but, on the other hand, the impulse to speak must also be present. In melancholic insanity we sometimes observe persistent loss of speech, not from any lack of something to say, but because there is no incitement to the act, or because inhibitory influences immediately repress any tendency to utter words. If we take for granted that the mental material for speech exists, then the transformation of this material into actual speech is a result of the following complicated processes, the disturbance of which, individually, produces the various forms of aphasia.

In the first place, the speaker must be acquainted with the word which expresses the mental conception. If, for instance, he wishes to tell another the name of some animal, he must know the appropriate word—"dog," "sparrow," "frog." This knowledge, which, as far as our mother tongue is concerned, we all acquired in childhood, may, as experience shows, be lost again in case of cerebral disease. Just as we may ourselves forget a word momentarily, or as any healthy person may, at the sight of an animal that is perhaps rather rare, be "unable at the minute to think of its name," so in disease one may forget all or a greater or less number of words. Such a patient sees a dog, and knows well enough that it is an animal possessed of such and such qualities, but he has forgotten its name. The association between the conception "dog," and likewise between the perception of a dog by the eye, and the appropriate vocal representation "dog," is lost. This condition is termed amnesic or sensory aphasia, because it is due to complete or partial loss of the memory for words. The patient knows perfectly well what he wishes to say, but the words escape him. At the same time, in cases of pure amnesic aphasia, the power of repetition is unimpaired. As soon as we say "dog" to the patient, he repeats the word perfectly well; and sometimes he also perceives that this is really the correct word; but in other instances, although the word is correctly repeated, the patient does not become conscious of its meaning (*vide infra* "word deafness").

If we examine cases of amnesic aphasia more carefully we may notice by what special sensory perceptions the memory of the lost word can be eventually called back. Although the mere idea alone does not call the proper word back to consciousness, the sensory perception (so far as we have to do with external objects) may often by association recall the corresponding word. It is, of course, most frequently the visual perception which recalls the memory of the word. If this association also fails we speak of an optic-amnesic aphasia. In other cases auditory impressions (e. g., the ticking of a watch, the note of a musical instrument) may restore the recollection of the proper word, or, in other cases still, sensations of touch, taste, or smell. All these associations between given perceptions and the corresponding verbal representations may be destroyed at the same time or one by one, or they may be partially retained. Sometimes it is only by the simultaneous action of different sensory impressions—e. g., perceptions of sight and touch—that the idea of the word can again be recalled to consciousness.

Of great interest are certain cases where there is only partial amnesia. These have been repeatedly observed. Thus a patient forgot nothing but his own name, remembering all other words perfectly; or the loss of words may be confined to but one language, the patient being still able to express himself tolerably well in another tongue. In a case observed by Graves, the patient still knew the initial letter of all words; if, for example, he saw a cow, he would know that the corresponding word began with C, and would look under C in a dictionary till he found the word.

Among the amnesic disturbances, we may also class the dissolution of closely associated sequences of words, as are found, for example, in the order of numbers, in the names of the days of the week, the names of the months, familiar prayers, etc. Although such sequences of words which have been practiced from childhood may on the one hand (*vide infra*) greatly facilitate the pronunciation of the individual words, on the other hand the connection often becomes so loose that the patient may repeat the whole list very well after any person, but he can not recite it spontaneously. Such tests often show most interesting peculiarities. Thus we have repeatedly noticed, for example, that aphasic patients can repeat the names of the days of the week very well in their ordinary sequence, but that it is very hard to say them backward. Many patients find great difficulty in counting backward.

If the memory for words be retained, the next requisite for speaking is the transfer of the word image into such action of the muscles of our organs of speech as is calculated to produce the word in question as an actual sound. This motor process is so complicated that an extremely accurate co-ordination of movements is demanded for the correct pronunciation of the word. Man therefore possesses a separate center, in which this transfer of the word image into the motor processes of speech takes place. If this center be diseased, there again results a loss, or at least a greater or less impairment, of speech. The patient is in this case well aware of the word he wishes to say, but he can not pronounce it. He has, if we may use the expression, forgotten the movements that are essential to speaking. His tongue and lips are not really paralyzed, but the patient no longer knows how to make use of them for talking. He has reverted to the condition of childhood, before he had learned to talk. The patient often makes the greatest effort to speak. The word he wishes to utter "keeps hovering before him"; he moves his mouth in the most striking manner, but brings out only an occasional sound, and that incorrect. This commonest form of speech disturbance is known as ataxic aphasia. Of course it is equally impossible for the patient to repeat a word after some one else. He keeps his eyes fixed on the mouth of the speaker, and endeavors to imitate the motions of his mouth, but he is either partially or totally unable to reproduce the sound.

Ataxic aphasia exhibits many degrees of intensity. On the one hand there are cases of complete aphasia, where the patient can utter only such separate sounds as "a," "e," etc. And, on the other hand, there are also mild cases where there are merely errors in pronunciation. Such cases may best be termed "ataxic" aphasia. The patient pronounces many words correctly, but with others there are such mistakes as the transposition of individual letters, the misplacement or omission of letters, or, finally, the adding on of letters. For example, he will say thens instead of then, widow instead of window, dipter instead of dipper, hefd instead of held, wrelster instead of wrestler, and belnow instead of below. This mildest form of ataxic disturbance is termed "stumbling over syllables" (*Sil-*

bernstolpern), or "literal ataxia." Patients sometimes affix the same inappropriate termination to many words; for example, a woman used to count: "onen, twoen, threen, fouren, fiven," etc. In most instances the patient can pronounce some words tolerably well, others only imperfectly and with difficulty, and still others not at all. Usually the patient gradually learns a few common words and expressions (e. g., "good morning") by means of persistence in repeating them as they are uttered by another, so that he pronounces them better and better. What is very remarkable, and not so very rare, is that a patient will be able when in a passion to pronounce a word, such as an expression of anger, an oath, or an exclamation, perfectly well, because it is done to a certain extent involuntarily, while he can not utter the same words if he wishes to say them. One patient under our observation always said "well," in answer to the question, "How are you?" but she was wholly unable to repeat the word "well" by itself when asked to do so. Association also often exerts an appreciable influence; for example, a patient who finds it absolutely impossible to pronounce "six," utters it with perfect distinctness if he begins to count from one, in the ordinary way, up to six. When patients have at last managed to say the right word, they sometimes stick to it—that is, they continue to repeat this word when they should say something else. In many severe cases of motor aphasia the whole capacity for speech is limited to a few syllables or a few brief phrases, which always appear as soon as the patient makes any attempt to speak (so-called *monophasia*).

A patient of our own could for a long time utter nothing but the meaningless words, "*selber sag ich nämlich selber*" (self say I namely self). The entire verbal thesaurus of another (female) patient whom we saw consisted of the meaningless sounds "bibi" and "eibibi." Still a third could say only "tinne, tinne." The patient is quite well aware that what he says is wrong, but, despite all his efforts, every attempt to speak excites these same sounds. It produces a comical impression to see the patient use the same invariable word, with all sorts of facial expression. Thus the woman mentioned above begged for things, with "bibi," in a coaxing tone, while sometimes she would give vent to violent anger with a loud "bibibibi."

Amnesic (sensory) aphasia may also vary greatly in degree. It is only rarely observed in its pure form, but it is usually associated with motor aphasia. The amnesic (sensory) aphasia does not always consist of the complete loss of memory of words, but very often merely of a disturbance of the firm bond of association between ideas and their corresponding words. Patients with sensory aphasia therefore very often confuse their words or form entirely false and senseless words (so-called *paraphasia*). Such a patient may talk a long while without conveying any idea to the listener, inasmuch as he says "brush" instead of "bed," or "gove" instead of "give," etc. It is a very interesting fact that in paraphasia the influence of certain associations are often manifest. The patient, for instance, utters a wrong word which has a certain resemblance in sound to the right one, begins with the same syllable, etc. Purely ideal associations sometimes also play a part; thus one of our patients called a white handkerchief "snow," etc.

The aphasic disturbances thus far described all refer to the power of speech—that is, to the uttering of ideas aloud, but we now come to the description of the disturbance in the understanding of speech, which is often associated with this or which in rare cases may occur by itself. This disturbance consists of the patient's inability to understand the words spoken to him, although he can hear them well. The word when heard no longer excites in his consciousness the appropriate idea. The patient is not really deaf, for he hears everything, but he no longer understands what he hears, and he has forgotten what the words signify. The vernacular sounds to him as a foreign language, of which he knows nothing or of which he has learned but little, would to a well man. This condition is best termed, according to Kussmaul, "word deafness" or "soul deafness." Wernicke also calls word deafness "sensory aphasia," which does not seem to us to be quite correct, since word deafness—that is, the defective understanding of speech—is not identical with the inability to speak in consequence of the loss of ideas of speech (amnesic or sensory aphasia in the stricter sense). Amnesic aphasia and word deafness are of course very often associated from allied reasons, but it may very well be the case that a patient has forgotten the word for an idea, but that he at once recognizes its significance as soon as he hears it. Word deafness is in a certain sense a higher degree of verbal amnesia. A healthy person often can not find the word for an idea in a foreign language, but the idea comes to him at once if he hears the word. It is much harder to speak a foreign language than to understand it.

Slighter degrees of word deafness are very often (we might say almost always) associated with motor aphasia. We must, however, search for them carefully, preferably by giving the patient certain commands (to point to certain objects or parts of the body, to perform certain acts), avoiding any suggestive help by signs or expression, and seeing whether he understands what is said and acts accordingly. We may also test him by holding an object in front of him and calling it by various wrong names and then finally giving it the correct name. We thus see whether the patient gives any sign of recognizing the correct name or not. The detection of word deafness is usually limited, however, to concrete nouns, certain verbs or adjectives, but it is hardly practicable to examine for word deafness for any other words (many abstract words, adverbs, etc.), especially when the patient is also aphasic. We very often notice, therefore, that aphasic patients understand very well the usual brief questions directed to them, but that they are quite unable to follow with complete understanding any long speech, anything that is read to them, etc. They often fail to understand because the rapidity of perception is much impaired. We have seen patients who understood any question or sentence only after the words had been repeated several times. In such patients we also see at times a decided failure of memory of the actual words. If we repeat to them any long sentence, they can repeat only the first four or five words correctly, while they have wholly forgotten the end of the sentence, or at best they have retained the sense but not the sound of the words. We can readily understand how this actual weakness of memory, or abnormally easy fatigue of memory, may have an inhibitory influence on the understanding of speech.

The list of disturbances occurring in aphasia is by no means exhausted by the examination of the motor power of speech as such and of the understanding of words when heard. Word and idea are yet closely associated with two other modes of expression, which are brought about not by the ear but by the eye. We mean the movements of mimetic expression (or the language of signs in the ordinary sense of the word) and then the far more important written or printed optical signs—letters, script, numbers, etc. Here also we find the close associations between signs and ideas on the one hand and signs and sounds on the other. These associations may be loosened or wholly lost, and then there follow a series of further morbid symptoms which are often to be observed as existing with aphasia.

The disturbances of movements of mimetic expression ("*amimia*") have not a very great practical significance, but they often show many interesting features. We are often surprised to see how incapable aphasic patients, who can not speak, are in giving expression to their thoughts, desires, etc., by signs. They sometimes employ signs in a way that is quite contrary to their usual meaning. Thus we have often seen patients nod their heads when they clearly wished to deny something. The understanding of signs made by others may also be much diminished.

Agraphia, the disturbance of the power of expression in writing, is far more important than amimia. We can also distinguish here a pure motor (ataxic) and an amnesic agraphia.

By motor (ataxic) agraphia we understand, of course, not the inability to write on account of any paralysis of the arm, but the inability to unite the different letters correctly to make up the written word. The patient is also unable to copy a word put before him, but he can often do this well when he can not write spontaneously or from dictation. Then there is writing amnesia (amnesic agraphia). In such cases also we often see interesting relations of association, since the patient may write a wrong word which in sound or idea is somewhat like the word desired. The entire manner of writing in such cases is often noticeable. The patient holds his pencil or his pen with peculiar awkwardness; he writes a very small hand, or the letters gradually grow smaller, etc.

Alexia has the same relation to agraphia that word deafness has to aphasia. Alexia is word blindness—that is, the defective understanding of written speech, the inability to read. This disturbance is plainly allied to soul blindness above described (page 1076), but alexia may very well exist without special soul blindness, since only the association between the written signs and the corresponding idea has been lost. Patients with pure alexia can neither read nor copy what is written, but they are able to write very well spontaneously or from dictation. Speaking accurately, we must distinguish between reading aloud and reading to one's self. The printed word when seen may excite the idea, although it can not be pronounced aloud. On the other hand, many patients can read aloud without understanding what is read or pronounced.

In conclusion we may mention briefly an interesting disturbance which is also closely related to aphasia—the disturbance in the power of musical expression [*amusia*]. In these cases the most varied morbid symptoms may occur, which refer both to the perception and to the reproduction of musical tones. The associations between ideas of tone and the printed notes may be loosened or wholly lost. The examination of the relations existing between words and tones as they exist in singing is also very interesting. We have often seen aphasic patients who could not repeat the words of a familiar song, but who could at once utter the correct words plainly if they sang it.

From all that has been said we can see how extremely manifold and in how many varying combinations the special symptoms of aphasia may occur. Only a careful and thorough study of each individual case of aphasia will insure an exhaustive review of all the disturbances present.

The Anatomical Changes occurring in Aphasia.—As has already been mentioned, aphasia was the first cerebral symptom which could be referred to disease of any definite part of the brain. The whole "topical diagnosis" of diseases of the brain has therefore started from aphasia. As early as 1825 Bouillaud affirmed that disease of the anterior lobes of the brain is alone capable of producing disturbances of speech. In 1836 another French physician, Marc Dax, pointed out for the first time that only lesions of the left half of the brain cause aphasia; and in 1861 Broca was able at last to declare that the "center for speech" lies in the left third frontal convolution ("Broca's convolution"). This law has since been confirmed by hundreds of autopsies, but it applies only to true motor aphasia. If there is motor aphasia, we may with certainty assume that there is disease of the posterior portion of the left third frontal convolution. It is in this region, therefore, that those complicated processes of motor co-ordination which are essential to the utterance of a word take place. A second important and positive fact we owe to Wernicke: if there is, either with motor aphasia or alone, word deafness—

that is, a defective understanding of spoken language—we may from this fact almost always conclude that there is disease of the left upper (first) temporal convolution (and especially its posterior portion). In this part of the brain, then, is the connection between the heard word and the corresponding idea. Among other localizations we may mention that pronounced motor agraphia probably points to a lesion of the foot—that is, of the posterior portion—of the second left frontal convolution. Pure alexia (word blindness) has been observed especially in focal diseases of the left lower parietal lobe (region of the angular gyrus) and the neighboring parts of the occipital lobe.

The anatomical localizations of the different forms of amnesic aphasia can be determined less accurately because in such cases there may be various possible interruptions between the various centers of perception and the speech centers ("conduction aphasia," "subcortical aphasia"). Since, however, the tracts leading to the motor-speech center probably pass through the neighborhood of the island of Reil, we may expect that amnesic aphasia would occur most frequently in destruction of this region; but, from reasons that are easily understood, foci of disease in other situations may also give rise to such "conduction aphasias." As soon as amnesic aphasia is associated with defective understanding of speech (soul deafness), we may almost always assume a lesion in the vicinity of the upper temporal convolution.

No general rules can be laid down as to the prognosis and course of aphasia, inasmuch as everything must of course depend upon the nature of the disease which excites the aphasia. In the aphasias due to cerebral hæmorrhage or cerebral embolism (*vide infra*), which are the most common, we very often observe that the disturbances are very marked at first, but that they gradually diminish or wholly disappear. On the other hand, there are also cases of almost complete motor aphasia, which may persist for years until the death of the patient. [When aphasia occurs in childhood it usually disappears completely, perhaps because the other hemisphere of the brain becomes educated.—K.]

In regard to treatment we can here mention only one thing, that methodical training in the form of actual lessons in speaking or writing may sometimes be of some service. In motor aphasia we may give lessons, just as to deaf-mutes, by assisting the new practice of the necessary muscular movements by visual impressions, while in verbal amnesia methodical training of the memory is necessary for a new "impression" of the forgotten word. Of course all such training demands much tact and patience on both sides.

[**Dejerine's Views on Aphasia.**—The doctrines of Dejerine and his pupils have acquired so much importance in the study of aphasic disturbances, and these doctrines differ so much from the views expressed above, that it seems advisable to give a brief synopsis of them.

According to Dejerine there are only three centers for images of language: Broca's center, for motor images of articulation, at the foot of the left third frontal convolution; Wernicke's center, for auditory images of words, in the posterior part of the first and second left temporal convolutions; and the center for visual images of words in the angular gyrus. These three centers form the "zone of language," and surround the extremity of the fissure of Sylvius like a horseshoe (see Fig. 173, page 1073). They are connected with one another and also with the adjacent sensori-motor (especially that part which contains centers for the lips, tongue, and larynx), visual, and auditory centers.

The zone of language may be affected by disease in two ways: (1) The lesion may destroy a part of the zone or its intrinsic fibers; or (2) it may isolate a part of the zone from the adjacent cortex. In the first case—aphasia from lesion of the zone of language, *true* aphasia, or cortical aphasia—internal language as a whole and all the forms of language will be affected to a greater or less extent, although the dominant clinical phenomena—motor aphasia, word deafness, word blindness—will vary with the seat of the lesion. The idea of the word is affected, and this gives rise to a considerable degree of mental impairment.

In *true* motor aphasia from destruction of Broca's center the patient may utter an occasional word (*vide supra*, page 1079), but he is unable to do more than that. He has, moreover, lost the idea of the word so completely that he can not even indicate the number of syllables of which it is composed, and he is wholly unable to write spontaneously or from dictation, except that he can often write his name or some very familiar word. The co-existence of agraphia is characteristic of this form of motor aphasia. There is no marked word deafness, although the spontaneous evocation of auditory images is affected and the patient has difficulty in comprehending rapid speech. Word blindness is less marked, and in copying the patient will copy print as script. In *true* word deafness from destruction of Wernicke's center the patient may seem deaf, confused, or even demented, although the mental failure is much less than at first it appears. There is inability to understand what is said, and the loss of all auditory images deprives the motor center of any regulator, so that the patient uses wrong words, or even mere jargon—paraphasia or jargonaphasia. There is usually some word blindness and the patient becomes unable to write, or else, on account of the loss of the idea of the word, he may write nonsense or jargon—jargonagraphia. When he copies he usually makes a servile copy, copying print as print and script as script. In *true* word blindness there is also complete agraphia, and some degree of word deafness and paraphasia. In these sensory forms of *true* aphasia there is less likely to be any paralysis, owing to the situation of the affected centers.

When the zone of language is still intact, but when the lesion has simply cut off the connections of its centers with other centers, we find the condition which Dejerine calls *pure* aphasia, or sub-cortical aphasia. In these cases internal language is unaffected, the idea of the word is retained, and there is never agraphia. The symptoms are referable solely to the affected center. With *pure* motor aphasia the patient can write normally and can tell the number of syllables in a word. With *pure* word deafness the only symptoms are inability to understand what is said and to write from dictation. With *pure* word blindness there is also right homonymous hemianopsia, and the patient copies in a slavish manner, but there is no agraphia; he can write normally, both spontaneously and from dictation. In no form of pure aphasia is there any mental impairment. Dejerine furthermore, does not believe that there is any special writing center at the foot of the second left frontal convolution or anywhere else; for there is agraphia in every case of lesion of the zone of language, and the agraphia is manifested not only by inability to write with either hand, but by inability to produce words intelligently by arranging lettered blocks or by the use of the typewriter.—K.]

4. The Centrum Ovale, Internal Capsule, Central Ganglia, and Region of the Corpora Quadrigemina

Centrum Ovale.—The white substance of the hemispheres is made up, so far as we know at present, both of commissural fibers, which connect the various cortical centers together, and of fibers which proceed downward from the centers of the cortex and connect these centers with peripheral parts of the body (corona radiata). As to the symptoms caused by diseases which destroy the commissural fibers, there is hardly anything known. We can only surmise that in case of disturbances of association, such as we have studied under aphasia and kindred disorders, we should consider lesions of commissural fibers, as of those connecting together the temporal and frontal lobes, the occipital and frontal lobes, etc.

A break in the continuity of the fibers of the corona radiata must of course result in the same symptoms as if the corresponding center were itself destroyed. This explains why circumscribed lesions of the centrum ovale, if they involve the motor fibers of the corona radiata, which proceed from the central convolutions (and only if they do this), cause hemiplegia, or, if very limited, monoplegia. In an analogous manner, disease of the white substance of the occipital lobe may entail hemianopsia; of the temporal lobe, auditory disturbances, such as word deafness. More than once, quite extensive disease of the white substance of the frontal lobe on one side has been discovered post mortem, although no symptoms whatever had been caused by it. Only when the coronal fibers which proceed from the left third frontal convolution are involved in disease is motor or ataxic aphasia inevitable.

Internal Capsule.—The most important facts relating to the functions of the internal capsule, as far as at present known, have been already stated. In particular, it was pointed out that through the posterior limb of the internal capsule, in a comparatively narrow space, passes the pyramidal tract on its way from the central convolutions to the crura cerebri (see Fig. 95, page 829). Here, then, even a very limited focal disease must lead to complete hemiplegia on the opposite side of the body. Clinical experience also shows that the largest number of cases of persistent hemiplegia are occasioned by disease in this spot. The arrangement of the fibers in this region is such that the fibers for the face are for the most part anterior, the fibers for the arm next, and the fibers for the leg posterior. In a brilliant experimental study Horsley and Beevor have shown that in monkeys the different motor portions of the internal capsule can be separately irritated in a much more exact fashion. They found in these experiments that the motor fibers of the internal capsule showed the following arrangement from before backward: opening the eyes, associated movements of the head and eyes to one side, tongue, angle of the mouth, shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankle, great toe, other toes.

The sensory tract (compare page 799, and Fig. 95, page 829) lies at the posterior extremity of the internal capsule, and apparently includes not only the fibers for cutaneous sensation, but also for the organs of special sense. Complete disorganization of this spot ought therefore to cause, in the opposite half of the body, not only anæsthesia of the skin, but also simultaneously a corresponding impairment of smell, taste, and hearing, and hemianopsia—in short, a so-called complete cerebral hemianæsthesia. Still, in regard to this very point we are much in need of further and definite observations.

Certain practical conclusions in regard to diagnosis can be deduced from the preceding facts. A purely motor hemiplegia, unattended by impairment of sensation, implies a lesion that does not involve the posterior portion of the internal capsule; but probably this portion is also affected, when there is not only paralysis, but considerable sensory disturbance. The sensory disturbance does not invariably extend to all the senses; quite often there is nothing but cutaneous anæsthesia.

Lesions in the neighborhood of the internal capsule may also cause symptoms of irritation of the tracts which pass through the internal capsule. Thus unilateral symptoms of motor irritation in particular have frequently been observed in the form of choreic twitchings (so-called post-hemiplegic chorea, *vide infra*), and unilateral symptoms of sensory irritation (pain, paræsthesia) may also be caused sometimes by lesions in the vicinity of the posterior portion of the internal capsule (Edinger).

Central Ganglia; Caudate Nucleus (Corpus Striatum proper), Lenticular Nucleus, and Optic Thalamus.—Before the course of the pyramidal tract had been accurately determined, ordinary cases of cerebral hemiplegia were almost universally ascribed to lesions of the central ganglia, and in particular of the caudate and lenticular nuclei. At present, however, observations seem to force one to the conclusion that a complete hemiplegia can be produced only by a cutting off of the pyramidal tract. There are, indeed, numerous cases of hemiplegia presenting circumscribed disease of the central ganglia; but they can probably all be explained by supposing either that the pyramidal tract, as it lies in the contiguous internal capsule, is directly involved in the disease, or that its functions are suspended by the indirect effects of the neighboring lesion—for instance, by the pressure it exercises. Accordingly, we find that circumscribed lesions of the central ganglia in the neighborhood of the internal capsule generally produce a temporary hemiplegia—that is, the paralysis gradually improves as the indirect influence of the focal disease upon the internal capsule ceases. Chronic and incurable hemiplegia, however, if due to a lesion anywhere in this region, always implies an actual lesion of the pyramidal tract in the internal capsule. In regard to hemianæsthesia, also, the facts seem to be quite analogous. It was formerly held that this phenomenon was especially connected with a lesion of the optic thalamus, the reason being that the sensitive fibers lie so close to the thalamus in the posterior extremity of the internal capsule that they become themselves involved.

About the symptoms which lesions of the central ganglia directly produce little is definitely known. The results of both clinical observation and experiment are quite contradictory, and in repeated instances quite extensive disorganization of these parts has existed without producing any symptoms to speak of during life. In particular, it should be borne in mind that softening may occur in the lenticular and caudate nuclei, and yet not a trace of hemiplegia be observable. It seems probable also that the optic thalamus has nothing to do with voluntary motion; but apparently it has some importance for movements of mimetic expression (Bechterew, Nothnagel). In hemiplegics we sometimes see that the lower half of the face on one side can not be moved voluntarily, but it is moved very actively in emotional expression, such as laughing or crying. In such cases we can decide that the thalamus is intact, while in lesion of the thalamus the contrary is observed—the affected half of the face can be moved voluntarily, but it remains completely rigid in emotional expression. As to sensory functions, there is only one which the optic thalamus is certainly known to possess: the central termination of some fibers of the optic nerve lies in its posterior portion (the so-called pulvinar), while other fibers go to the corpus geniculatum externum. Destruction of the posterior part of the thalamus accordingly produces complete hemianopsia (*vide* page 1075) of the opposite side. The thalamus is also very probably connected with other centripetal fibers from the lemniscus, but we do not know positively whether these are sensory or co-ordinating fibers, and pathology has not yet taught us as to the functions of the thalamus in this regard. Focal disease of the thalamus has repeatedly occasioned post-hemiplegic symptoms of irritation ("post-hemiplegic chorea," *vide supra*), as is easily to be explained from the propinquity of the pyramidal tract. According to recent experiments, the lenticular nucleus contains centers for regulating heat. The striking changes in temperature sometimes seen in cerebral affections may often, therefore, be brought into possible relation to diseases of the lenticular nucleus, but accurate observations are still almost wholly lacking.

Corpora Quadrigemina and Crura Cerebri.—Diseases of the corpora quadrigemina are infrequent, and usually, when they do occur, they are merely a part of more extensive lesions of the brain. They are therefore very rarely considered from a diagnostic point of view.

The anterior tubercles are certainly connected with the fibers of the optic nerve. If both of the anterior tubercles be destroyed, total blindness is inevitable, while if only one be disorganized, hemianopsia is to be anticipated. Still, these symptoms are, of course, too ambiguous ever to be regarded as pathognomonic of localized disease of the anterior corpora quadrigemina. Another point to be considered, whenever the corpora quadrigemina are diseased, is the position of the nuclei of the nerves which preside over the motions of the eyeball, and in particular of the oculo-motor and trochlear nerves. This explains why unilateral, or even bilateral, paralysis of the oculomotorius has been repeatedly observed in connection with lesions of the corpora quadrigemina, as have also more rarely nystagmus and immobility of the pupil. It is also worthy of note that in diseases in the region of the corpora quadrigemina, especially of the posterior corpora, ataxia of the body, similar to cerebellar ataxia, has been repeatedly observed (Nothnagel). This ataxia is in all probability due to a disturbance of the superior cerebellar peduncle coming from the corpus dentatum of the cerebellum, which after decussating goes to the red nucleus (nucleus ruber, Fig. 86, page 798), beneath the corpora quadrigemina, and is further connected with the cerebrum. If we have to do, therefore, with an ophthalmoplegia affecting both eyes, but not completely symmetrical, and manifesting itself especially by symptoms of paralysis of the oculo-motor and trochlear nerves (more rarely the abducens), and if this ophthalmoplegia is associated with an uncertain, staggering gait, we must consider disease of the corpora quadrigemina. Of course a similar association of symptoms may also occur in tumors of the cerebellum. The posterior corpora quadrigemina and the median geniculate bodies are connected with the acoustic nerve, so that in diseases of this region we may expect disturbances of hearing.

Should the crura cerebri become involved in the disease, the resulting symptoms are often very characteristic of the locality affected; there is paralysis of one side of the body (arm, leg, facial nerve), and at the same time a crossed paralysis (that is, one situated upon the opposite side) of the motor oculi. A glance at Fig. 96 (page 830) will explain this phenomenon. Thus, a circumscribed lesion on the right side would destroy the fibers of the third nerve (III) on that side, and therefore produce a right-sided paralysis of the oculo-motor nerves, and at the same time, if extensive enough, the lesion would involve the pyramidal fibers of the right crus, and thus occasion left hemiplegia. That disease of the tegmentum would lead to sensory disturbances or sometimes ataxia may be taken for granted. Diseases of the tegmental region and its vicinity therefore cause a clinical picture which is composed of hemiplegia, tactile hemianæsthesia (or hemiataxia), and crossed oculo-motor paralysis of the opposite side.

5. The Cerebellum

Quite extensive destruction of the cerebellum may take place without any symptoms to indicate it. In such cases, however, the disease is almost invariably confined to the hemispheres; but, if the central portion or vermiform process be attacked to any great extent, peculiar symptoms almost always result, pointing in many instances with considerable certainty to disease of the cerebellum. In some cases, however, disease limited to the cerebellar hemispheres may cause pronounced cerebellar symptoms.

There are two especially characteristic symptoms—a peculiar uncertainty of gait (cerebellar ataxia), and troublesome vertigo.

Cerebellar ataxia affects the trunk and lower extremities. Both standing and locomotion are chiefly interfered with. When the patient is lying in bed he can move his legs almost as well as ever, and with normal vigor; but as soon as he gets up, the characteristic motor disturbances become very evident. Even while standing still, the whole body can usually be plainly seen to sway back and forth. This becomes more marked if the patient brings his heels together. If he stands with his legs widely apart, the trouble is less noticeable. Closing the eyes, as a rule, does not aggravate the swaying, inasmuch as the cutaneous and muscular sensibility of the lower limbs remains normal in uncomplicated cerebellar disease. When the patient tries to walk, he sways and totters, precisely as if he were deeply intoxicated, but usually in a very different way from that seen in tabes dorsalis. Instead of the uniform stamping and pitching gait of the latter, cerebellar ataxia causes a real staggering of the whole body, so that in severe cases the patient loses entirely the ability to walk straight, but seems to fall forward, as it were, in a zigzag line, now to the right and now to the left. Sometimes, but by no means invariably, it is noticed that, in walking, the body sways principally in one particular direction, either forward, backward, or to one side. Such peculiarities, however, do not enable us, with our present knowledge, to determine with certainty just what position in the cerebellum the lesion occupies. The most we can do is to surmise, in such a case, that the middle peduncles of the cerebellum (*vide infra*) are involved. It is worthy of note that, with few exceptions, the ataxia does not involve the upper extremities. Many a patient who can scarcely walk unaided is still able to perform the most delicate manipulations with his hands. This shows that it is only in maintaining the bodily equilibrium—essential to standing and locomotion—that the function of the cerebellum is important.

As already stated, this cerebellar ataxia is in most cases attended with pronounced vertigo. There is not, however, a complete correspondence between the locomotor disturbance and the dizziness. Exceptionally one symptom may be present without the other. The vertigo is usually felt only when the patient is erect, standing or moving about, and is very seldom felt when he lies quietly in bed. Cerebellar vertigo is usually a true vertigo or turning sensation: the patient feels as if he himself or the surrounding objects were revolving. The feeling of vertigo may be constantly present or it may come on in severe individual paroxysms. The cause of the vertigo is probably to be looked for in a disturbance of those tracts which go to the cerebellum through the corpus restiforme as a continuation of the vestibular nerve.

Little definite is known about other symptoms of disease in the cerebellum. True ataxia of the legs (as in tabes) is also seen at times; but in such cases we usually have to do with the distant action of the disease on the tract of the lemniscus. The patellar reflex has sometimes been found absent in cerebellar disease. This symptom also is probably always dependent upon co-existing disturbances of the spinal cord. In some cases intention tremor has been found in the arms; in others hemiparesis of the extremities on the side of the disease (corresponding to the results of Luciani's experiments on animals). The significance of all these symptoms is still doubtful. We will return later to certain special symptoms, especially those occurring in tumors of the cerebellum.

We must add in conclusion a few words about diseases of the middle cerebellar peduncles (crura cerebelli ad pontem). Those peculiar symptoms known as forced movements, and forced positions are usually referred to an irritation of these peduncles on the ground of physiological experiments. Forced positions are when the patient always lies upon one particular side in bed, whether he is perfectly conscious, or in a state of complete unconsciousness. If he is put in any other posture, he at once involuntarily reassumes his former position. Not infrequently this forced position of the trunk is accompanied by a corresponding forced position of the head and eyeballs, while the extremities are seldom affected. Genuine forced movements are seen far less often. They produce either often-repeated rotations of the body on its longitudinal axis, or, if the patient be able to walk at all, involuntary circular movements ("circus movements"), etc. According to the statements of Russell, the forced movements in disease of the *right* middle cerebellar peduncle are in the direction "of a corkscrew screwed *into* a cork," and, in disease of the *left* peduncle, the direction "of a corkscrew screwed *out* of a cork." It must be said, however, that the symptoms of forced positions and forced movements are on the whole rare and of doubtful value in diagnosis; for in some cases of cerebral disease the same symptoms have been observed without any discoverable affection of the middle cerebellar peduncle. On the other hand, in two cases of tumor of the middle cerebellar peduncle with autopsy we failed to find any forced movements.

For convenient reference we subjoin a summary of the most important facts bearing upon the localization of cerebral diseases.

1. The most frequent cause of ordinary hemiplegia is a lesion of the pyramidal tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract has been functionally deranged for a time by focal disease in neighboring parts of the brain.

2. Monoplegic cerebral paralysis is usually due to affections of the cortex of the brain—that is, the central convolutions and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolutions. Monoplegia of the arm is referable principally to some lesion of the middle third of the anterior central convolutions. Monoplegia of the lower extremity implies some affection of the upper portion of the anterior central convolutions and the paracentral lobule.

3. Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one half or one particular portion of the body, are almost always caused by cortical lesions. These same symptoms of motor irritation without accompanying paralysis are likewise to be ascribed to some irritation of the above-mentioned regions of the cortex.

4. Hemiplegia with crossed paralysis of the oculo-motor nerve indicates a lesion of the crus cerebri. Co-existing tactile hemianæsthesia implies that the tegmentum is involved.

5. Hemiplegia with crossed facial paralysis implies, with great certainty, that the lesion is situated in the pons.

6. Post-hemiplegic chorea (*vide infra*) seems to occur especially when there is focal disease in the neighborhood of the posterior part of the internal capsule.

7. Hemianæsthesia of the skin and of the organs of special sense, associated with hemianopsia, is due chiefly to lesions of the most posterior portion of the internal capsule.

8. Hemianopsia may be due to a lesion of the occipital lobe (cuneus). Probably, also, a lesion of the posterior extremity of the internal capsule may cause it, in which case it is usually associated with hemianæsthesia. Finally, it may be produced by affections of the pulvinar of the optic thalamus, of one lateral geniculate body, of one of the anterior corpora quadrigemina, or of one of the optic tracts.

9. Genuine motor aphasia indicates disease of the foot of the third left frontal convolution.

10. Word deafness (loss of understanding of speech) is due to disease of the first left temporal convolution; word blindness (loss of understanding of writing) is due to disease of the left lower parietal lobe (angular gyrus).

11. Difficulty in articulation implies disease of the medulla, as does also dysphagia.

12. Staggering gait and vertigo are the most constant symptoms of cerebellar disease, but they may also occur in diseases of the corpora quadrigemina and of the frontal lobe (*vide supra*). Forced positions and forced movements perhaps indicate lesions of the crura cerebelli ad pontem.

As an appendix, we may add a very brief review of the course of the most important conducting tracts:

1. *Chief Motor Tract.*—(*a*) *First central neurone:* Ganglion-cells in the motor cortex of the brain, axis-cylinder processes through the corona radiata, posterior limb of the internal capsule, crusta of the crus cerebri, decussation in the pyramids, postero-lateral column of the spinal cord, terminal arborization in the anterior horns (for the motor cranial nerves previously in their nuclei). (*b*) *Second peripheral neurone:* Ganglion-cells in the anterior horns (or in the nuclei of the motor cranial nerves), axis-cylinder processes in the anterior root and the peripheral nerve, terminal arborization in the muscular fibers.

2. *Chief Centripetal* (*Sensory and Co-ordinating*) *Tract.*—(*a*) *First* (*peripheral*) *neurone:* Ganglion-cells in the spinal ganglion, axis-cylinder processes partly in the peripheral sensory nerve [modified protoplasmic processes], partly in the posterior root; terminal arborization, partly in the posterior horns (sensory fibers), partly, after passing through the posterior columns of the cord, in the nuclei of the columns of Goll and Burdach (co-ordinating fibers). (*b*) *Second neurone:* Ganglion-cells in the posterior horns or in the nuclei of the columns of Goll and Burdach, axis-cylinder processes through the antero-lateral columns, inter-olivary layer, lemniscus tract of the medulla and pons, tegmentum of the crus cerebri, posterior portion of the internal capsule. Terminal arborization in part in the optic thalamus. (*c*) *Third neurone:* Ganglion-cells in the optic thalamus, axis-cylinder processes to the cerebral cortex (central convolutions, parietal lobe).

3. *Chief Optic Tract.*—First peripheral neurones lie wholly in the retina, and the ganglion-cells of the second neurones are also in the retina, axis-cylinder processes through the nerve and optic tract (partial decussation!) to the lateral geniculate body, the anterior corpora quadrigemina, and the pulvinar of the optic thalamus, where the terminal arborization takes place. Third neurones from the cells of the parts mentioned through the posterior portion of the internal capsule [and the optic radiations of Gratiolet] to the cortex of the occipital lobe.

4. *Chief Acoustic Tract.*—The ganglion-cells of the peripheral neurone lie in the cochlear ganglion of the cochlea, the axis-cylinder processes in the cochlear nerve, termination in the ventral acoustic nucleus. Here the second neurone begins, whose axis-cylinder process goes in the trapezoid body and the striæ acousticæ (?), and then in the lateral lemniscus to the posterior corpora quadrigemina and the median geniculate body. Here probably a third neurone begins, which ends mainly in the cortex of the temporal lobe. The acoustic fibers also run partly crossed and partly uncrossed, so that each auditory nerve is connected with both hemispheres.

CHAPTER III

CEREBRAL HÆMORRHAGE

Ætiology.—The cause of cerebral hæmorrhage should always be sought in some disease of the coats of the minute cerebral arteries. Cerebral hæmorrhage occurs most frequently, therefore, in persons who suffer from general arterio-sclerosis, and especially from arterio-sclerosis of the cerebral arteries; but other forms of disease of the walls of the blood-vessels, fatty and hyaline degeneration, and, in particular, syphilitic endarteritis, may give rise to cerebral hæmorrhage. In 1868 it was first shown by Charcot and Bouchard that in many cases of large cerebral hæmorrhage there are miliary aneurisms of the small atheromatous arteries of the brain substance, some one of which has burst and allowed the blood to escape. All later investigators have confirmed their statements about the occurrence and importance of these miliary aneurisms. The aneurisms may attain a diameter of a millimetre or more. They usually appear like spindle-shaped dilatations of the entire circumference of the vessel, although sometimes the bulging is confined to one side of it. Inasmuch as the intra-cerebral arteries possess almost no true adventitia, it is easy to see that these vessels are especially predisposed to aneurismal dilatation (especially in the form of a so-called dissecting aneurism).

Most of the circumstances which come under our consideration as favoring the development of cerebral hæmorrhage have also a close relation to arterio-sclerosis. Age plays a specially important part. Although exceptionally a younger individual may be attacked, the majority of sufferers are over fifty years old—that is, at the time of life when arterio-sclerosis usually becomes most fully developed. Again, cerebral hæmorrhage is decidedly more frequent in men than in women, which is also true of atheroma. Alcoholism, syphilis, and gout are also reckoned among the ætiological factors of both disorders, and in both a hereditary predisposition is not very rarely demonstrable. What is called the "apoplectic habit" also deserves brief mention. Although there is no variety of constitution which exempts its possessor from the possibility of cerebral hæmorrhage, yet it can not be denied that often the victims of apoplexy do exhibit a certain "habit." Such persons are not very tall, but are corpulent, broad-chested, with a short, thick neck and round face; and they have not been disinclined to the pleasures of the table and the bottle. Such persons often suffer at the same time from emphysema, moderate hypertrophy of the heart, and general arterio-sclerosis, as the condition of the radial and temporal arteries may disclose even during life.

Granting, therefore, that disease of the arteries, and sometimes more particularly miliary aneurisms resulting from chronic endarteritis of the smaller cerebral arteries, must be regarded as the chief cause of cerebral hæmorrhage, then, on the other hand, the question suggests itself whether an abnormal elevation of the blood-pressure may not have some part in determining the hæmorrhage. If the coats of the arteries be normal the greatest increase of pressure would not be able to cause rupture of the vessels; but if the wall of the artery is diseased, or if aneurisms have already been developed, then there can be no doubt that a persistent or even a temporary elevation of the blood-pressure must favor the bursting of the vessels. In this sense a cerebral hæmorrhage, occurring in patients with certain forms of cardiac hypertrophy (contracted kidney, idiopathic hypertrophy, etc.), combined with disease of the vessels, may be referred in part to the increased arterial tension; but it is most of all with regard to many exciting causes, which are immediately followed by a cerebral hæmorrhage, that increased blood-pressure assumes great importance. Here it is temporary. Cerebral hæmorrhage may, for example, follow excessive muscular exertion, the ingestion of a large amount of food, indulgence in alcohol, taking a cold bath, violent mental excitement, etc. An apparently slight injury is especially to be considered. In all such cases, however, the change in the arteries is a necessary prerequisite.

It should be mentioned, in conclusion, that cerebral hæmorrhage is sometimes merely the expression of a general hæmorrhagic diathesis, as we find it in leukæmia, pernicious anæmia, and those affections which are called, in a stricter sense, hæmorrhagic diseases, such as scurvy and purpura hæmorrhagica. The grave infectious diseases, including septicæmia, typhus or typhoid fever, and small-pox, may occasion hæmorrhage into the brain as well as into other organs. The hæmorrhages are generally, however, from capillary vessels, and are very rarely extensive.

Pathology.—Arterio-sclerosis and miliary aneurisms do not develop in all the cerebral arteries with equal frequency, and accordingly we find certain regions particularly liable to cerebral hæmorrhage, being very much oftener affected by it than others. These regions seem also to be the ones where the arterial blood-pressure is relatively the highest.* Hæmorrhage takes place by far the most frequently from the branches of the artery of the fissure of Sylvius. The large central ganglia in the neighborhood of the lateral ventricles, the optic thalamus, caudate and lenticular nuclei, and also the adjacent white matter of the internal capsule and centrum ovale, are therefore usually affected by the hæmorrhage. Hæmorrhages in other portions of the brain are much less frequent—such as hæmorrhages into the convolutions, the pons, the cerebellum, the crura cerebri, or the medulla. If the blood escapes into the neighborhood of a ventricle, it may burst into the latter. Likewise, in rare instances, an effusion of blood in the cortex may make its way out upon the surface of the brain.

An extensive collection of blood in one of the hemispheres may exercise so decided a pressure upon surrounding parts that the results of increased tension upon the affected side are at once recognized when the skull is opened. The dura on that side is more tightly stretched, the falx is crowded over to the opposite side, the convolutions on the convexity seem flattened, and the furrows are shallow. Exceptionally, when there is a very large effusion reaching nearly to the surface, we may even detect fluctuation.

On cutting through the brain-substance we find the seat of hæmorrhage, and are enabled to determine its position and extent. Its size, of course, varies considerably in different cases; it may be small, or it may occupy a large part of an entire hemisphere. The wall of the effused mass is made up of ragged and torn cerebral tissue, and the mass itself contains *débris* of the nervous elements entangled in the coagulated blood. The blood-clots are almost always very dark-colored when fresh. Later on, the mass changes to a chocolate-colored or more brownish-yellow pulp, composed of the disintegrated remnants of the nervous substance and the clotted blood. The microscope reveals, particularly in the immediately surrounding tissues, numerous fatty granular cells. These are white blood-corpuscles which have absorbed the fat resulting from the decomposition of the myeline substance. There is also always an abundance of hæmatoidine crystals, due to the disintegration of the red blood-globules. At a greater distance from the effusion the tissues present a yellowish tinge, from the imbibition of such blood-pigment as has reached them in a state of solution; and there is also usually an œdematous softening of the parts not too far removed from the hæmorrhagic focus.

If the patient survive, the mass is gradually absorbed. It slowly diminishes in size, and the surrounding parts tend to reassume their normal relations. The final result in many cases is a cavity filled with serous fluid and bounded by smooth walls. This "apoplectic cyst" remains stationary. In some instances, however, and particularly if the effusion be rather small, the walls approach each other as more and more of the fluid is absorbed; there is a great hyperplasia of connective tissue; and so, finally, there is nothing left but a so-called apoplectic scar, usually of a yellow color, due to vestiges of the blood-pigment. The position and dimensions of the permanent lesion determine, of course, the question of secondary descending degeneration (*vide* page 1033), as well as the nature and extent of the persistent clinical symptoms.

Clinical History.—The symptoms of cerebral hæmorrhage agree closely with the anatomical lesions just described. The vascular disease responsible for the

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[* Mendel has found that the normal blood-pressure in the lenticulo-striate arteries, which are most prone to rupture, is much higher than it is in the cortical and many other small arteries of the brain.—K.]

hæmorrhage (arterio-sclerosis, miliary aneurisms) in many cases excites no morbid symptoms; but, on the other hand, we often find that persons attacked by a cerebral hæmorrhage have suffered much from vertigo, headache, etc., for more or less time before the shock. Such symptoms are undoubtedly due to the disease of the cerebral blood-vessels and to the slight disturbances of the circulation thus produced.

As soon as the vessel wall or an aneurism bursts, however, and blood escapes into any part of the brain-substance, there is immediately seen a group of grave cerebral symptoms, collectively termed an apoplectic attack, or "shock." As the blood escapes under a pressure nearly equal to the general arterial pressure, and doubtless much greater than that to which the soft substance of the brain is normally exposed, the affected portion of the brain is at once subjected to a considerable increase of tension, which is transmitted for various distances in all directions. It need not be said that the destructive influence of the hæmorrhage, which is probably manifested by a compression of the nervous tissue itself and also more especially by a compression of the blood and lymph channels and a consequent disturbance of the circulation, may vary exceedingly, and that therefore the symptoms are by no means equally severe in all cases. The larger the rent in the blood-vessel, and the more rapid and abundant the consequent hæmorrhage, the worse is the apoplectic attack. Bleeding from larger vessels is therefore usually attended by graver symptoms than from the minute arterial twigs. An extensive cerebral hæmorrhage sometimes causes the patient to fall down suddenly in complete unconsciousness, while smaller hæmorrhages may occasion only a temporary attack of vertigo and slight cloudiness of intellect. If the tear in the wall of the artery be very small and narrow, permitting the blood to escape but slowly, then there may be no sudden attack at all, the phenomena requiring a certain length of time for their development.

There is also an important relation between the location of the hæmorrhage and the severity of the apoplectic attack. The chief symptom of these cases is loss of consciousness (about which we shall soon speak at length); and as this is certainly due to an interruption of the functional activity of the cerebral cortex, it is plain that the nearer the cortex is to the hæmorrhagic focus, the more apt are the symptoms to be serious. It is confirmatory of this that hæmorrhage into the more deeply situated portions of the brain, the crura cerebri or the pons, quite often occasions comparatively slight symptoms. But there is a fact about the circulation in the brain that often causes the shock from hæmorrhage into the brain-stem to be greater than the shock following hæmorrhage into the cortex or the white substance of the hemispheres. This fact is that the brain-stem has comparatively much larger arteries than the other parts just mentioned, which contain only minute blood-vessels. Furthermore, as Duret and Heubner have shown, the blood-vessels are so distributed that the arterial tension in the brain-stem is not a little higher than in the other portions. This renders intelligible the clinical phenomenon that hæmorrhages in the territory of the main arteries, besides being, as we have said, the most frequent of any, produce apoplectic symptoms even when the effusion is comparatively small; while sometimes hæmorrhages of about the same size in the cortex or white substance may not be noticed.

The clinical phenomena of the apoplectic attack will now be considered in detail. The onset is sometimes absolutely without warning, but in other cases it is preceded for a greater or less length of time by certain prodromata. These are either the result of the disturbance of circulation caused by the disease of the blood-vessels in the brain, and then, as already stated, they comprise occasional headache, vertigo, tinnitus aurium, spots before the eyes, languor, and muscular weakness; or they are caused by minute hæmorrhages, which seem not infrequently to precede a greater one. In such a case, the friends state that of late the patient has had one or more slight and brief attacks, characterized by faintness, temporary trouble in speaking, sudden but temporary weakness of an arm or leg, and similar symptoms. The prodromata may extend over several days or even weeks and months preceding the severe attack.

In other cases there are no such premonitory symptoms. The apoplexy occurs unexpectedly and suddenly. In the midst of apparently vigorous health the patient sinks down "as if he had been struck." In still other cases there are indeed no prodromata, but the symptoms do not at first appear in all their severity, and occupy some hours or even a whole day in their gradual development. This is due to a slow and gradually increasing hæmorrhage, and is termed a slow or delayed apoplectic attack. The patient grows confused, anxious, and delirious (a case of our own had pronounced hallucinations of sight); the arm and leg on one side become paretic, and gradually more and more completely paralyzed; and after a few hours complete unconsciousness comes on.

The attack may be rapidly fatal. In such cases the abnormal pressure probably involves the medulla oblongata and paralyzes the cardiac and respiratory centers there situated. Usually, however, there is merely a complete loss of consciousness, more or less rapidly developed. Sometimes the patient has time to lie down. He usually sinks back in his chair or falls to the floor, and becomes deeply comatose. The face is often noticeably flushed, and the pulse full and tense, but not infrequently somewhat slow, because of the increased cerebral pressure. The respirations are deep, noisy, stertorous, and likewise often slow. The relaxed cheeks and lips are often drawn deeply in at every inspiration, and puffed out at every expiration. The temperature is usually subnormal at first, later regaining the normal, or even a higher point. In a rapidly fatal case, however, the temperature remains depressed till death. It is not very rare in severe cases to observe a peculiar position of the head and eyes, both being turned in the same direction. This phenomenon is termed by Prévost conjugate deviation (*déviation conjuguée*) of the eyes and head. It is generally temporary, and is said by Landouzy to be connected principally with a lesion of the lower parietal lobule (see also page 1075). There is no perfectly constant relation between the lateral deviation and the half of the brain affected. The most common condition is that the eyes are directed toward the affected hemisphere, and so to a certain extent "look toward the lesion" and away from the paralyzed side of the body. The pupils present no constant peculiarities. Often they are of normal size. In other cases they are contracted, dilated, or unequal. No definite diagnostic conclusions can be drawn from them. In the worst cases the pupils will not react to light; in other cases they react, but often sluggishly.

During the deep apoplectic coma the extremities generally lie completely motionless and limp. In the worst cases reflex action is wholly suspended; but sometimes the vigorous thrust of a pin or the pinching of the skin will excite an occasional slow reflex twitch, or a motion as if to ward off the tormentor. Whether the apoplexy has caused hemiplegia at all, and if so in what place, can not always be easily determined during the initial coma. Still, it is often to be observed, even now, that one angle of the mouth hangs down lower than the other, and that the corresponding cheek is more puffed out during expiration than is the other; that the extremities of one side are much heavier and fall much more limply than those on the opposite side of the body, and that the reflex action and defensive movements are almost absent upon one side (the paralyzed side), while they can be clearly demonstrated on the other.

In contrast to the usual laxness of the arms and legs during the apoplectic coma is the tonic rigidity sometimes seen in the extremities, particularly on the side opposite to the hæmorrhage. This symptom seems to be especially, although not exclusively, connected with a bursting of the escaping blood into a lateral ventricle. It is rather exceptional for cerebral hæmorrhage to be attended with general or unilateral epileptiform convulsions—a symptom which, as we have seen, is referable to irritation of the motor regions of the cortex.

It should be mentioned that in many cases of cerebral hæmorrhage the urine passed after the attack has been found to contain small amounts of albumen or sugar. This symptom is usually ascribed to compression of the medulla from the effusion. There is usually retention of urine; in other cases there is involuntary micturition.

A certain number of patients never awake from the initial coma. Death may not be immediate, but they remain completely unconscious; the respirations become more rapid and irregular (sometimes of the Cheyne-Stokes character), and there is a rattling in the throat, because mucus and saliva run down into it; the pulse, which was at first retarded, now becomes accelerated; the face grows paler and more and more sunken; the eyes are deep in their sockets; the cornea becomes opaque; and at last, after the coma has lasted some hours, or even one or two days, death occurs, often attended by a considerable rise in temperature.

This termination is, however, by no means the usual one. More frequently the patient survives the attack. The bleeding ceases, the clot contracts, and begins to be disintegrated and absorbed. At the same time the pressure exerted upon surrounding parts grows less and less, the more distant parts of the brain gradually recover from the shock, and consciousness slowly returns. The patient begins to open his eyes when he is spoken to in a loud tone; he raises his hand to his head, sighs, and yawns; gradually the intellect clears up, he tries to talk, or to express himself by signs; memory returns, and he recognizes those about him once more. Exceptionally, recovery is interrupted by a fresh and perhaps a fatal relapse. This may result from a renewal of the hæmorrhage. Generally, however, improvement persists, the patient fully regains his consciousness at the end of a few days, and it now becomes possible for the first time to "estimate the damage."

The symptoms thus far described belong to severe apoplectic attacks. There are also, as we have said, cases of all degrees of mildness, as regards the first onset. In these there is no deep and persistent coma. The patient loses consciousness only temporarily, if at all. He is seized with vertigo, or with sudden headache, and is for a time stupefied. Nausea and vomiting are of frequent occurrence, just as in ordinary syncope. Yet cases presenting these comparatively slight early symptoms, and with few even of these, may exhibit the genuine focal symptoms referable to the hæmorrhage, such as hemiplegia, in all their severity. These latter phenomena must now be considered.

We term the symptoms of loss of function directly dependent upon the destruction of a definite portion of the brain the direct focal symptoms of cerebral hæmorrhage. Where the hæmorrhage takes place we have seen that a larger or smaller extent of the brain-substance is completely destroyed by the sudden and forcible escape of the blood. The dimensions of this lesion are represented later by the apoplectic scar or cyst, and its position by the nature and extent of the persistent, and for the most part irreparable, loss of function (*Ausfallserscheinungen*). But there are, in addition to these direct symptoms, other indirect focal symptoms, which outlast the apoplectic shock, and vary with the locality of the hæmorrhage. These do not, however, correspond to the territory actually destroyed. They are due to the influence exerted for a certain length of time by the hæmorrhagic focus upon the immediate surrounding structures. The pressure of the effusion, the disturbance of circulation resulting from it, the collateral œdema, and perhaps also the imbibition of the soluble products of disintegration of the apoplectic focus, are the chief factors in exciting these indirect symptoms. They do, indeed, outlast the initial shock, but are nevertheless temporary, vanishing sooner or later, at the end of several days or weeks, or even months. If the apoplectic shock is over and if the symptoms still exhibited have been minutely determined, yet we are unable to say, at first, whether the existing focal symptoms are direct or indirect. We can decide about this only after further observation. If the early symptoms disappear within the next few days or weeks, or after the first two or three months, we can then affirm, retrospectively, that they were indirect. Such as outlast the first six months are to be regarded as direct, and as not destined to improve much. From a practical point of view this distinction is of extreme importance. We shall revert to the subject when considering the course of cerebral apoplexy.

A minute description of all the focal symptoms which might occur after cerebral hæmorrhage, and of the light thus thrown upon the location of the hæmorrhage, need not be attempted here, for it would necessitate a repetition of all the facts enumerated in the preceding chapter. It is only requisite to describe, in detail, the chief and by far the most frequent results of a cerebral hæmorrhage — ordinary cerebral hemiplegia (see Fig. 175).

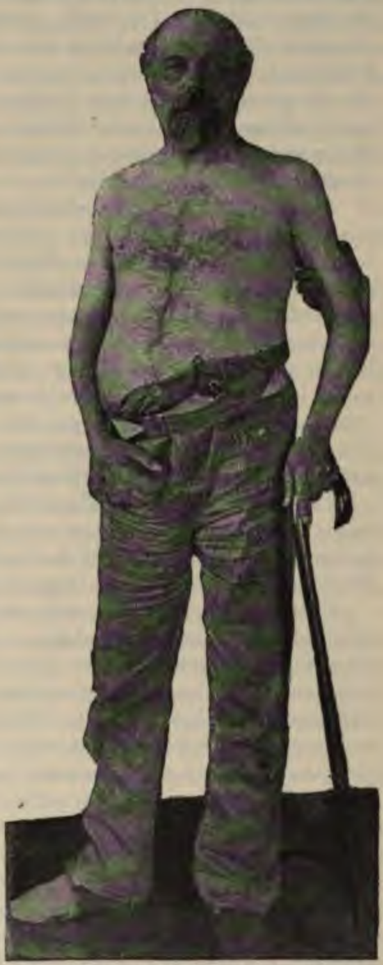
FIG. 175.—Patient with right hemiplegia. (Personal observation.)

It has been mentioned that most of these hæmorrhages occur near the lateral ventricles. Hence, in a majority of instances, the motor pyramidal tract, as it traverses the internal capsule, is either directly destroyed or at least indirectly affected. Consequently, most patients who survive the apoplectic shock present a paralysis of that half of the body which is opposite the seat of hæmorrhage. On minute examination we usually find, in the first place, that even in the distribution of the facial nerves there is a distinct difference between the two sides, the lower division of the facial (which supplies the muscles of the cheek, nose, and mouth) being evidently paralyzed on one side, while its upper division (going to the eyes and forehead) is entirely, or almost entirely, intact. The forehead can be wrinkled on one side as well as on the other, or only a little less on the paralyzed side than on the sound side; but if the patient tries to dilate his nostrils, or alter the shape of his mouth, or whistle, or blow, or talk, etc., the paralysis becomes evident. Often, indeed, while the face is quiet, it can be noticed that one nasolabial fold is obliterated, or that one corner of the mouth droops. It is an interesting fact that the paresis of the lower division of the facial is much more noticeable during voluntary efforts, as in showing the teeth, than when the patient smiles involuntarily. A patient will sometimes try in vain to draw back the corner of his mouth, then begin to laugh at his own awkwardness, and thereupon open his mouth in an almost perfectly normal manner. We have seen above, in the chapter on cerebral localization, that this condition can probably be explained by the relation of the optic thalamus to the movements of mimetic expression. The difference in the behavior of the upper and the lower part of the face in cerebral hemiplegia is probably connected with the fact that the muscles supplied by the upper division (frontalis, corrugator supercilii, and to a certain extent the orbicularis) are very seldom exercised upon one side alone, but always bilaterally. Perhaps both sides receive nervous fibers from each cerebral hemisphere, so that, if a single center be intact, it alone answers for the muscles on both sides.* In the distribution of the lower division of the facial, also, ordinary cases of cerebral hemiplegia present almost always a more or less marked paresis, and only exceptionally a complete paralysis.

There is quite often a slight impairment of the hypoglossus in addition to the paresis of the facial nerve. If the patient puts out his tongue, its tip deviates toward the paralyzed side. This is a result of paresis of one of the genio-hyoglossi. When both these muscles contract, they may be said to push the tongue forward. If this thrust be more vigorous on one (the healthy) side, the tongue is deflected toward the other (paralyzed) side. In ordinary cerebral hemiplegia this is almost always the sole way in which the movements of the tongue can be seen to be impaired. Sometimes, however, the slight paresis of half of the tongue, combined with the facial paresis, entails a noticeable difficulty in articulation. This, to be sure, is only exceptionally great, and is often appreciated by the patient alone, who is conscious that an effort is required in order to speak.

The soft palate is rarely much affected. It may, however, hang rather lower down on the paralyzed than on the affected side, and move less. The uvula is inclined sometimes toward the healthy and sometimes toward the paralyzed side. There are no special disturbances of function as a result of these changes.

The trapezius is the only muscle of the trunk which is ordinarily much affected in cerebral hemiplegia. As a result of the paresis of this muscle, the shoulder sags and it can not be raised as high as on the normal side. If the patient tries to take a very deep breath, it is in some cases possible to see that the paralyzed side lags behind a little in respiration, which is due to a paresis of the respiratory muscles on that side. It is perhaps due to this that the pulmonary diseases which attack hemiplegic patients frequently develop in the lung on the affected side, where respiration is deficient.

The most important element in the hemiplegia is the paralysis of the extremities. Immediately after the hæmorrhage it is often so complete that even the slightest voluntary motion in the affected arm and leg is impossible. Other cases, however, exhibit only a more or less severe paresis (hemiparesis) from the first; or the complete paralysis at any rate is confined to certain groups of muscles, the others still retaining vestiges of their normal contractility. Even when there is total hemiplegia at first some of the muscles almost always regain a certain amount of their old power later on (*vide infra*).

The behavior of the reflexes is comparatively constant in nearly all cases of cerebral hemiplegia. In general the rule is that the tendon reflexes are increased on the paralyzed side, but that the skin reflexes are diminished. The increase of the tendon reflexes is an almost constant symptom. Only when the symptoms of the initial shock are very severe, there may at first be no tendon reflexes whatever. In all cases of any duration they are, with few exceptions, exaggerated, and usually very much so. In the arm we find very lively periosteal reflexes on tapping the lower end of the radius and the ulna (see page 840), also on striking the tendons of the biceps and triceps, the clavicle, etc. In the leg the increase of the patellar reflex is most noticeable; but there is often a lively Achilles reflex also, an increase of the adductor reflexes, etc. This increase of the tendon reflexes is by no means limited to the affected side; it can almost always be detected on the sound side also, although to a lesser degree. Many have expressed the opinion that the increased tendon reflex upon the paralyzed side is a result of the secondary degeneration of the pyramidal tracts in the spinal cord. In our judgment this view is entirely unfounded, inasmuch as the exaggeration of the tendon reflex often appears within a few days, or even a few hours, after the apoplectic attack—that is, before a secondary degeneration in the spinal cord is to be thought of. We would rather seek to explain the phenomenon as being due to the suspension of certain reflex inhibitory influences because of the cerebral lesion.

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* There is a remarkable general rule which should be mentioned here, and which is perhaps to be explained in the same way, namely, that those muscles which are usually called into play in pairs are never completely paralyzed in cerebral hemiplegia. Furthermore, it is impossible for us to contract most of these singly, on one side alone, or at least not without special practice: this applies, for instance, to the corrugator supercilii, the frontalis, the motores oculi, and the muscles of mastication and respiration.

There is very often found, particularly in cases of long standing affected with well-marked contractures, an increase of "direct mechanical excitability" in the paralyzed muscles, such that a tap upon them causes them to contract vigorously. We are of the opinion that a part of these contractions are of reflex origin and are due to the mechanical irritation of the fascia of the muscles (fascial reflex). There is also the direct mechanical irritation of the nerves of the muscles.

The skin reflexes in hemiplegia behave in precisely the opposite way from the tendon reflexes, being almost invariably decidedly diminished on the paralyzed side. In the paralyzed arm it is usually impossible to excite any skin reflex whatever; and in the corresponding leg there is either no, or at any rate a greatly diminished reflex. The difference in certain other skin reflexes, especially the abdominal and cremaster reflexes (page 839) is still more evident than in the plantar reflex. [The plantar reflex, however, is often manifested by extension of the great toe—the Babinski reflex.—K.] These reflexes are almost always much diminished or wholly absent on the paralyzed side, while they can be elicited on the sound side in normal strength, a distinction which is not infrequently of service in determining the seat of the hemiplegia when the patient is stupid or even quite unconscious. There is often a marked difference in the conjunctival reflex also. On the paralyzed side we can touch the eyeball with the finger or the head of a pin without causing an immediate reflex closure of the eye, as is the case on the healthy side. It is not easy to explain the striking behavior of all the skin reflexes in cerebral hemiplegia, especially the peculiar contrast between them and the tendon reflexes. It is ordinarily assumed that certain inhibitory influences for the tendon reflexes are lost through the cerebral disease, while the inhibition of the skin reflexes is, on the contrary, put into a state of irritation. This explanation is evidently forced and unsatisfactory, and thus the suspicion is not wholly unwarranted that these skin reflexes, contrary to the physiological theories thus far generally accepted, have their reflex arcs not in the spinal cord, but much higher up in the brain. The absence of certain skin reflexes in cerebral hemiplegia is accordingly due to a direct injury of the reflex path. Of course it must be emphasized that further studies upon this important point are extremely necessary.

Sensation is but little impaired in most cases of cerebral hemiplegia, but on careful examination we often find a decided blunting of sensibility for simple contact, although the sensibility to pain and temperature and the muscular sense are retained. Slight paræsthesia is not infrequently complained of on the affected side, especially at first. Pain in the paralyzed parts is not uncommon. It is often to be referred to special peripheral conditions (contractures, etc.), but sometimes there is apparently an irritation of the central sensory tracts (Edinger). Any marked disturbance of sensation indicates, as has been seen (compare page 1084), that the posterior extremity of the internal capsule is involved. Such cases are rare. In them we may observe a complete cerebral hemianæsthesia combined with the hemiplegia. According to Gowers, a temporary hemianopsia is often present directly after the occurrence of a cerebral hæmorrhage. We have seen the initial hemianopsia combined with associated paralysis of the ocular muscles. It is also not uncommon to find hemiplegia associated with permanent hemianopsia; but as yet little is known about the pathological anatomy of such cases. The most likely thing would seem to be some lesion of the fibers of the optic nerve in the internal capsule or in the pulvinar of the optic thalamus. The muscular sense is not usually affected in hemiplegia. [The stereognostic sense (*vide* page 795) is not infrequently affected.—K.]

Turning now to the further course of hemiplegia, we find a new group of important symptoms. First of all should be considered the changes in the paralyzed muscles. If the hemiplegia be incomplete, even from the start, almost the normal degree of motility may in a comparatively brief period be regained by the affected side. At most there will persist a certain slight amount of weakness and stiffness; and even this will gradually diminish. From what has been already said, it is evident that in these cases the initial paresis is an indirect focal symptom, and accordingly vanishes as soon as the effusion ceases to affect the tissues not immediately involved.

Even where there is a complete hemiplegia it is exceptional for this condition to remain unabated throughout the entire region affected. After a few weeks, or even a few days, one part and another of the paralyzed half of the body begin to recover their former motility. The improvement goes on slowly, and in the most favorable cases the paralysis may for the most part have vanished at the end of some months. Usually, however, the improvement advances only to a certain point, and the condition then attained remains stationary. We now find the ordinary characteristic typical " hemiplegic," who can be recognized at the first glance by the practiced eye. The most noteworthy phenomenon in such cases is this, that, in spite of many individual differences in the final recovery of certain movements and also in the permanence of the paralysis of other groups of muscles, there is still a very striking regularity and uniformity. There is one chief fact which is very noteworthy, that almost invariably the leg regains greater power of movement than the arm. In regard to the condition of the individual groups of muscles, moreover, we almost always find the same manifestations. As has been shown chiefly through the interesting investigations of Wernicke and L. Mann, the permanent paralysis in cerebral hemiplegia, or the return of motion, never relates to individual muscles, but always to whole groups of muscles whose functions are closely connected. In the upper extremity there is usually permanent paralysis of all the muscles that rotate the arm outward (supinators of the forearm, outward rotation of the upper arm by the infra-spinatus and teres minor, and fixation and outward rotation of the shoulder-girdle by the lower portion of the trapezius and the rhomboidei), and of those that raise the upper arm (deltoid, serratus), and also paralysis of the movements at the elbow, especially of extension, of opening the hand (extensors of the fingers, fixation of the wrist by the flexors), and of opposition of the thumb (opponens, abductor brevis). On the other hand, inward rotation of the arm and closure of the hand are, as a rule, preserved. The patient can therefore not take hold of an object on account of the difficulty of opening the hand, but he can hold firmly what he has once grasped. Fine movements of the hand, however, are much impaired on account of the disturbances in the movement of the thumb. In the leg the flexors of the lower leg and the dorsal extensors of the foot usually remain permanently paretic, and also the abductors and inward rotators of the hip, while the ilio-psoas, the quadriceps extensor, and the plantar flexors of the foot often regain their power of motion. The return of mobility in the ilio-psoas and quadriceps is therefore of great practical significance, because by it the patient is enabled to walk again. Of course the patient needs the help of a cane, and the gait remains slow and labored, but the advantage of independent locomotion is extremely great. The gait of the hemiplegic is very characteristic and is always of the same type. The affected leg has the toe turned slightly outward, it is moved forward slowly, dragging on the ground, in a slight curve whose convexity is outward. We can not state with certainty the reason for these facts just mentioned—that both the improvement and the permanent paralysis almost always involve regularly the same definite groups of muscles. The muscles which regain the power of motion are probably those which can be most easily innervated from the healthy hemisphere on the same side of the body.

It is improbable, although of course not impossible, that fibers when once destroyed can undergo regeneration; but the fact above mentioned is of the greatest practical consequence, that hemiplegic symptoms can not improve materially after the first six months.

The permanently paralyzed muscles become very often contractured later on. The appearances produced exhibit considerable uniformity. The arm, which suffers more from paralysis than the leg, is also usually more contracted. The fingers are almost invariably flexed; the forearm contracted in a position of pronation, and usually flexed rather than extended; and the upper arm adducted (the pectoralis major being chiefly affected). These contractures correspond exactly to the natural positions which the paralyzed arm almost always assumes if left to itself, either from gravity or as a result of any remaining power of movement. The contractures are almost always found in the same groups of muscles which suffer relatively the least loss of power in hemiplegia (*vide supra*). Since the position once acquired from the paralysis of the antagonistic muscles can not be altered, so-called passive contractures appear in the permanently shortened muscles. The correctness of this theory is favored by the fact that the deformity can to a certain extent be prevented by the persistent use of passive motion, which renders any permanent shortening of the muscle impossible. Nevertheless, Charcot and his pupils, including Bouchard, hold an entirely different opinion—namely, that the contractures are due to the secondary degeneration of the pyramidal tract. In defense of their position no fact can be adduced, save that fatal cases exhibiting hemiplegic contractures do invariably present the secondary degeneration mentioned; but of course this is no proof that the two things have any causative relation to each other. A contracture occurs only in connection with persistent paralysis; a persistent paralysis is never seen unless the pyramidal tract be destroyed; and if it be destroyed, a secondary degeneration must result. The contractures and the secondary degeneration are therefore two sequelæ, independent of each other. That the degeneration should "irritate" the fibers, and thus excite muscular contractions, is extremely improbable; for, according to all analogy, fibers which are undergoing degeneration can not be stimulated, and can not therefore transmit any sort of stimulus to the paralyzed muscles.

If the contractures become marked in the lower limb, they may involve either the extensors or flexors, which depends in the main upon accidents of position, etc. At the ankle we almost always find a contracture of the muscles of the calf, because the dorsal extensors of the foot are paralyzed and the foot falls into a position of plantar flexion from its own weight. The fact that the muscular tonus is increased on the paralyzed side (analogous to the increase of the tendon reflexes) is a circumstance which is not wholly without influence on the strength of the contracture. We may mention here the fact brought forward by Hitzig that many contractures are slight in the morning, when the patient wakes from sleep, and become more marked after he has begun to move.

Among other symptoms we will mention first the interesting associated movements, which are of frequent occurrence in hemiplegics. The chief explanation of them is that the patient can no longer innervate the different paretic groups of muscles with ease. In order to do so, it is always necessary to make a forcible associated movement of the leg, when the patient is endeavoring with all his energy to move his arm, and *vice versa*. If the patient tries to raise the arm at the shoulder, he flexes his forearm at the same time. The common associated movement in the foot (dorsal extension from contraction of the tibialis anticus) on flexing the hip and knee, which was first described by the author, is especially striking. Not infrequently the patient is quite unable to extend the foot dorsally as an isolated movement, but, if he draws the whole leg up to the trunk, there is then always a simultaneous marked dorsal extension of the foot. Associated movements also occur on the sound side on movements of the affected side, and it is said that the opposite condition has also been observed.

Another peculiar phenomenon must be mentioned here. It is what Weir Mitchell has termed post-hemiplegic chorea. Some time after the paralysis begins the parts affected by it exhibit involuntary movements, reminding one of chorea or athetosis (*vide* page 836). Sometimes these movements are continuous, sometimes they occur only as associated movements in connection with voluntary motions of the paralyzed, or even of the sound, side. Hemiplegia due to cerebral hæmorrhage very seldom exhibits this phenomenon. It is said to occur chiefly in focal disease of the posterior extremity of the internal capsule, and of the optic thalamus (irritation of the adjacent pyramidal tract?). It is much commoner in cerebral infantile paralysis (*vide infra*).

It is interesting to observe the trophic and vaso-motor changes in the paralyzed parts. At first the skin may be somewhat redder and warmer on the paralyzed than on the sound side. Nothnagel has shown that, even in the distribution of the cervical sympathetic, symptoms of vaso-motor paralysis occur. They are partly temporary and partly persistent, and comprise increase of temperature and color in the paralyzed side of the face, swelling of the eyelids, and contraction of the pupil; but they are usually slight. We very frequently find, especially on the back of the hands, more or less puffiness, which is likewise usually regarded as of vaso-motor origin. It should, however, be considered that the natural movements of any part of the body greatly promote the nervous and lymphatic circulation, and that the quietude of paralysis may therefore have much to do with the œdema. In hemiplegia of some duration the extremities upon the paralyzed side are always cooler than normal, and the hand in particular is very often deeply cyanotic. The skin sometimes becomes harsh and fissured, and often is thickened. The internal surface of the hand, in case of contracture, is frequently quite damp with perspiration.

Among the specific trophic symptoms of hemiplegia Charcot includes "acute malignant decubitus." This sometimes develops with extreme rapidity within a few days after the shock, and usually occupies the buttock on the affected side. There appears a circumscribed redness with the formation of vesicles, which is soon succeeded by a deep-reaching necrosis of the soft parts. We have ourselves never met with this in a case which has been properly nursed, and we can not therefore help thinking that its development is not wholly due to trophic disturbance, but to pressure and to the penetration of septic matter below the skin. Of course, patients long confined to bed with hemiplegia are as liable to bedsores as are any others similarly situated.

If the hemiplegia has existed for some time, we almost always find the paralyzed muscles more or less atrophied in comparison with those on the sound side. On microscopic examination we also find an evident atrophy of the individual fibers, but this atrophy, as a rule, is not so extreme as in peripheral or poliomyelitic paralyses (see page 834). Furthermore, the atrophy is always "simple," not "degenerative," and accordingly the faradic and galvanic excitability of the paralyzed muscles is completely preserved, even if the muscles are very much atrophied. The electrical excitability does not even show a slight quantitative diminution, as careful examinations have taught us. In some cases, even in ordinary hemiplegia, the atrophy of certain muscles (for example, the interossei, the thenar muscles, the deltoid, etc.) may come on unusually early and become extremely marked, so that we may speak of a "cerebral muscular atrophy." In such cases certain trophic influences actually seem to be especially manifest. The joints of the paralytic extremities, and in particular the knee and shoulder, may exceptionally become inflamed. The arthritis is sometimes acute, and sometimes more of a chronic variety. Its cause is not evident. Charcot thinks that it is probably a neurotrophic symptom, and he is of the same mind with regard to the rarely seen swellings of the peripheral nerve-trunks of the paralyzed side ("hypertrophic neuritis").

Mental symptoms are rare, except the initial loss of consciousness. There is sometimes, however, a persistent general uneasiness of mind, accompanied by great excitability and wakefulness. In a large number of cases of persistent hemiplegia there finally come on, in the course of years, constantly increasing indications of mental weakness. The patient grows dull and forgetful. Very often he exhibits a peculiar tendency to weeping, bursting into tears at the slightest provocation; but frequently he is subject to quick alternations of feeling, weeping and laughing in the same minute. The patient sometimes becomes eventually confused, excited, etc. All such pronounced conditions are usually dependent upon the general cerebral atrophy that comes from arterio-sclerosis.

The general nutrition is often good for a long time. Sometimes there is even a decided tendency to corpulence. In other cases, and especially in the bedridden, marasmus gradually comes on and hastens the fatal termination, particularly if there be some intercurrent trouble, such as a bedsore or bronchitis.

We have detailed the peculiarities of hemiplegia somewhat minutely, because the statements will apply in the main to all cases of cerebral hemiplegia, no matter in what place the pyramidal tract is interrupted or by what sort of a lesion. It is needless to enter into the diverse symptoms which are caused by diversity in the exact location of the hæmorrhage. The hemiplegia itself is the same, whether the effusion be in the cortex, internal capsule, crus cerebri, or pons. It is easy to infer from the preceding chapter what the accessory symptoms are which would enable us to localize the trouble. We need mention here only the frequent combination of right hemiplegia and aphasia. This occurs when there is a large effusion in the left hemisphere, extending from the internal capsule to the neighborhood of the third frontal, or possibly the uppermost temporal convolution (compare page 1077 *et seq.*).

Diagnosis.—The diagnosis of cerebral hæmorrhage rests on the sudden onset of the apoplectic symptoms, and on the later symptoms (if there be any) of impairment of the cerebral functions. The diagnosis can scarcely ever be made with absolute certainty, for cerebral embolism may exhibit almost identical phenomena: the differential diagnosis between the two will be given in the following chapter. Occasionally there may be danger of mistaking other cerebral affections for a hæmorrhage, such as meningitis and tumors. The same may be said of a suddenly developed uræmia, and of constitutional sepsis. In these cases the rapid onset of grave cerebral symptoms of a general nature, such as unconsciousness, simulates the apoplectic coma.

Prognosis.—The first question is, whether the patient will survive the initial shock. The answer depends upon the severity of the early symptoms. The deeper and more persistent the unconsciousness, the more deficient the respiration and pulse, the less the prospect of recovery; but we can never decide absolutely. [A steady rise of temperature or a very high temperature is also of bad omen. —K.] If the patient have withstood the first onset and be hemiplegic, then the possibility of improvement hinges on the question whether the paralysis is a direct or an indirect focal symptom. Inasmuch as there are no means of knowing about this at first, we must speak very guardedly. [The early appearance of ankle clonus or greatly exaggerated patellar reflex is apt to portend permanent paralysis with contractures.—K.] It should never be forgotten that the hæmorrhage may recur. The predisposing disease of the blood-vessels renders individuals who have had one stroke liable to be visited, sooner or later, by another.

Treatment.—The treatment of the apoplectic shock demands, first of all, rest in bed, with the head and shoulders elevated. To avoid bedsores it is very important to maintain cleanliness, and to watch attentively that portion of the skin which is pressed against the bed by the weight of the body. An ice-bag should be put upon the head, and particularly over that side on which the hæmorrhage is supposed to be. Bleeding was formerly universally practiced, but of late its usefulness is doubted. It is at most indicated only when the deep congestion of the face, the violent pulsation of the carotids, and the full, slow pulse show increased arterial tension. In such a case, if the patient seem otherwise robust, we may bleed at the commencement of an attack, in the hope of checking the flow of blood by lowering the intra-arterial pressure. In similar conditions experience shows that the local abstraction of blood from the temples is sometimes advantageous. The bowels should be well emptied by enemata, and later on by drastic purgatives. If the respiration and pulse fail, we may try stimulants (ether, camphor).

[Horsley and Spencer have found, by experiments on animals, that if the internal carotid be tied the bleeding from the divided lenticulo-striate artery is checked. Horsley therefore suggests that in the very earliest stage of cerebral hæmorrhage ligature or compression of the internal carotid may check the bleeding, and prevent the formation of a large clot and extensive destruction of the brain. This procedure has been tried with some benefit in one or two cases of ingravescent hæmorrhage.

Where there is much difficulty in respiration during the period of coma, relief may sometimes be obtained by turning the patient upon the side.—K.]

When the patient is safely over the shock, our resources for aiding him in the further stages of his trouble are very limited. As long as headache and symptoms of fever persist, the application of ice to the head should be kept up, and other disturbances should be treated symptomatically. For uneasiness and wakefulness, small doses of morphine or chloral are given. Treatment of the hemiplegia must be deferred for the first three or four weeks, until all the initial symptoms of irritation are over. Then electricity plays the chief *rôle*. Local galvanization should be tried, the current being made to pass transversely through the head, with as much regard as possible to the position of the hæmorrhagic focus; the current should be feeble, and the application should occupy two or three minutes. With this may be combined galvanization of the sympathetic nerve on the side of the hæmorrhage; nor should galvanization (stroking with the cathode) and faradization of the paralyzed muscles and nerves be neglected. If favorable changes take place, it is, however, uncertain how much should be ascribed to treatment; since, as has already been stated, there is often improvement without treatment.

Passive movements and massage of the paralyzed limbs are very important as a prophylaxis against contractures. They should be commenced promptly, and be continued methodically. Massage, and later on systematic and appropriate gymnastic exercises, may contribute much to the restoration of voluntary motions. The same object is also promoted by rubbing with spirits of camphor, chloroform liniment, etc.

Internally, potassic iodide is frequently given, out of regard for its "absorbent" properties. We may also try the effect of strychnine; it is most adapted for cases of some duration.

As to baths, they should not be too warm—that is, not over 90°–93° (26°–27° R.). Moderately warm baths, medicated with common salt if it seems desirable, and employed three or four times a week, seem to be beneficial. If it be thought best to send the patient to a regular bathing-place, Wildbad, Ragatz, Teplitz, Wiesbaden, Oeynhausen, Nauheim, etc., may be chosen. At the first-mentioned places too hot baths should be avoided.

Hemiplegic paralysis often lasts so long that the physician must repeatedly change the details of treatment, so as to support the courage and patience of the sufferer. Particular care should be given to regimen, in order that any recurrence of the hæmorrhage may, if possible, be avoided. The diet should be simple; any large amount of alcohol should be forbidden; and there should be no severe bodily exertion or mental excitement.

CHAPTER IV

CEREBRAL EMBOLISM AND THROMBOSIS (ENCEPHALO-MALACIA)

(Softening of the Brain from Embolism or Thrombosis)

Ætiology and Pathology.—Occlusion of the cerebral arteries is one of the most frequent injuries inflicted by embolism. Usually the emboli originate in the left side of the heart, from thrombi in the left auricle, or from the thrombotic deposits which form in chronic endocarditis (mitral or aortic disease) upon the valves of the left ventricle. Chronic arterio-sclerosis may also lead to thrombosis in the larger arteries, particularly the aorta; and in case the cerebral arteries themselves are extensively atheromatous, the larger vessels at the base of the brain may furnish material for embolism of the smaller cerebral arteries.

Thrombosis of the arteries of the brain is always due to primary disease of the blood-vessels, the most common cause being the chronic arterio-sclerosis just mentioned. Wherever the atheroma has altered the normal structure of the intima, deposits of fibrine may take place. Their development is further promoted by the subnormal elasticity of the arteries, and by the occasional narrowing of their lumen; for thus the flow of blood is rendered slow, if not even completely checked. It is easy to understand that thrombosis and embolism may each give rise to the other. From every thrombus an embolus may be detached; and every firmly lodged embolus may form a nucleus for thrombosis.

Next to arterio-sclerosis, the most frequent cause of a cerebral thrombus is syphilitic endarteritis. In the chapter on cerebral syphilis this subject will be minutely considered. Whether thrombi ever form here independently of disease of the vessels is doubtful. An apparently spontaneous thrombosis is now and then seen in patients who are cachectic or severely ill (cancer, grave typhoid fever, etc.). Such cases are explained either by the existing cardiac weakness, or perhaps by an abnormal tendency of the blood to coagulate.

In whatever part of the arterial system complete occlusion has been produced by an embolus or thrombus, the results depend upon the possibility or impossibility of blood reaching by collateral channels the region thus deprived of its ordinary supply. If the collateral circulation prove efficient, no harm is done; if not, the tissues must perish and undergo "softening." It is thus a matter of the greatest practical import that the perforating arteries of the brain-stem, and particularly the branches of the middle cerebral artery in the fissure of Sylvius, which supply the great central ganglia and internal capsule, are all "terminal," in Cohnheim's sense—that is, they form few anastomoses with neighboring vessels. Now, the middle cerebral artery and its branches are known from experience to be peculiarly liable to embolism, above other cerebral arteries. Hence we see why the region they supply suffers so severely and so very frequently from emboli. It is noteworthy that the left middle cerebral artery is more frequently plugged by emboli than is the right. In the centrum ovale and cortex there is more opportunity for collateral compensation than in the central ganglia; but even here the supply of blood often proves insufficient, as is shown by the not infrequent occurrence of spots of softening in the cortex and the white substance of the cerebrum. On the other hand, embolic foci are much rarer in the crura cerebri, pons, and cerebellum.

The various steps in the process which begins with embolic or thrombotic occlusion and ends in softening of the brain-substance are essentially the same as occur in other organs (compare chapter on pulmonary embolism, page 273). The tissues which are deprived of their arterial blood perish, become disintegrated, and are transformed into a soft homogeneous mass. Into the empty portion of the artery, beyond the embolus, blood flows in the reversed direction from the veins, and, if the anatomical relations permit, from the minute arteries in the neighborhood; but the supply is not sufficient to nourish the tissues. The walls of the blood-vessels become abnormally permeable and delicate, so that some red globules invade the disintegrating region by diapedesis, and others in the way of minute but genuine ecchymoses. Actual infarctions are, however, never formed in the brain, perhaps because, as Weigert suggests, the nervous structures swell so much as to exclude any large amount of blood; but the little punctiform ecchymoses are in many instances so abundant that the whole softened spot seems distinctly reddish or yellowish. This red or yellow softening is also due in part to the tissues being stained by the dissolved pigment of disintegrated blood-globules. If the discoloration be not very striking, then we speak of a white softening.

Fresh foci of softening are seen through the microscope to be composed of drops of myeline, bits of swollen nerve-fibers, numerous fatty granular cells, and free fat globules. The time required for these changes is one or two days. If within that period a compensatory collateral circulation is set up, the nervous structures may be restored and resume their functional activity. If not, the tissues perish and become disintegrated. The white blood-corpuscles and leucocytes (and possibly also the endothelial cells of the blood-vessels, and the neuroglia and ganglion-cells) absorb the fatty detritus thus made, and come to form the fatty granular cells above mentioned. If the patient live, the dead and disintegrated tissue is gradually absorbed, and it may even finally be replaced by a cyst not differing in appearance from those which occur after cerebral hæmorrhage. A minute focus of softening may also be replaced at times by indurated cicatricial tissue. If portions of the surface of the brain become softened, quite a deep depression often results. This is filled up in part with serous fluid and in part by hyperplasia of the pia mater. Sometimes the convolutions are still recognizable in places; but they are atrophied, of a yellowish color, and of a greatly increased consistency, due to the growth of cicatricial tissue.

Clinical History.—The occurrence of cerebral embolism is attended with almost precisely the same sort of shock as is cerebral hæmorrhage. We do not need to enter into the particulars again here, but will refer to the preceding chapter (*vide* page 1091). In embolism, also, the intensity of the shock varies from transitory confusion of intellect or slight vertigo to the deepest persistent coma. One chief factor in determining the nature of the case is the size of the occluded artery; another is its position, according as the embolism has taken place in the hemispheres or toward the base of the brain. In general, however, the shock of embolism, especially the loss of consciousness, is not often so severe and long continued as that of hæmorrhage; and embolism does not so frequently give rise to symptoms of cerebral compression, including slowing of the pulse. The temperature does not usually show the decline which is seen in fresh cerebral hæmorrhage, but moderate fever sets in as a rule in the course of a few days. On the other hand, epileptiform convulsions are seen more often in embolism than in hæmorrhage. Embolism again may have a rather slow onset, where there is at first a small embolus and this becomes the nucleus of a gradually formed thrombus.

It is not easy to explain why there should be a shock at all in case of embolism. Perhaps the main reason is the diminution of pressure which the embolism occasions in the region directly affected and in its neighborhood. The portion of the artery beyond the plug becoming empty not only causes a draught upon the blood and lymph, but subjects all the soft surrounding structures to a diminution of tension and to a certain amount of strain (Wernicke). It is not impossible, however, that the disturbance of circulation occasioned in the surrounding blood-vessels, including the capillaries of the cortex, by sudden embolism of a good-sized artery is of itself enough to account for the symptoms of shock.

With regard, also, to the persistent symptoms of embolism we may be equally brief, inasmuch as they very closely resemble those which follow a cerebral hæmorrhage. As has been said, it is only when a compensatory collateral circulation is developed within the first forty-eight hours that the early symptoms of focal disturbance can entirely vanish. After this period, the tissues which have been deprived of their normal blood-supply must undergo necrosis; although there is still room for hope that some of the symptoms will prove indirect, and therefore capable of improvement, so that embolic hemiplegia, like that from hæmorrhage, may improve decidedly in the course of the first few weeks.

Inasmuch as the middle cerebral artery is by far the most frequent seat of cerebral embolism, and inasmuch as this artery supplies the internal capsule as well as the great central ganglia, the most frequent focal symptom of embolism of the brain is ordinary cerebral hemiplegia, all the features of which have been depicted in the preceding chapter. Aphasia is associated with it with comparative frequency, for, as already mentioned, the left middle cerebral artery is especially apt to suffer. Less frequent is paralysis of a single member, due to cortical embolism, or an occipital lesion with hemianopsia.

Where softening is the result of thrombosis, the symptoms are but seldom abrupt in their onset. Usually the focal symptoms and the more general ones (such as unconsciousness) are developed rather gradually. This is most often seen in so-called senile softening of the brain, a disorder almost always caused by sclerosis of the cerebral arteries. The various symptoms generally come on under cover of repeated relapses and fresh aggravations of the disease. A severe initial shock seldom occurs; but almost invariably there is a gradual and progressive dementia.

The further course and the final termination of cerebral softening vary as do those of cerebral hæmorrhage. Embolism of a larger artery may cause speedy death. If, however, the first shock passes away, the impairment of function which may be left behind may last for years without seriously affecting the general health. There is always danger of a recurrence when the source of the embolism continues unchanged, as in cardiac disease or atheroma.

Diagnosis.—Both the onset and the persistent focal symptoms are so much alike in hæmorrhage and embolism that in many cases it is utterly impossible to decide which caused the apoplexy and hemiplegia. The following are factors in making a differential diagnosis, if one can be made: 1. It is very important to find whether there be any source for an embolus. Thus, if the patient have valvular cardiac disease (particularly mitral), embolism is more probable than hæmorrhage. 2. A young person is, on the whole, more apt to have embolism than hæmorrhage. At a later period of life one is about as probable as the other. 3. When the shock is severe and persistent, with red face, strong pulsation of the carotids, and signs of cerebral compression (slow pulse), we should think of hæmorrhage rather than embolism, in which latter the initial symptoms are sometimes comparatively slight (*vide supra*). 4. Finally, it is sometimes possible to obtain support for a diagnosis of cerebral embolism by demonstrating embolism elsewhere, as in the fundus oculi, by means of the ophthalmoscope.

In rare instances, also, tumors of the brain, into the substance of which hæmorrhage takes place, may induce symptoms closely simulating a primary apoplectic shock, as may also abscesses which have been previously latent, and then suddenly burst into a ventricle. In such cases a correct diagnosis is seldom possible.

Softening due to a thrombus is the most readily diagnosticated in those cases where there is cerebral syphilis (*q. v.*). Senile softening is inferred from the age of the patient, the signs of general arterio-sclerosis, the abrupt advances of the disease from mildness toward severity, and the developing dementia.

For the prognosis and treatment of cerebral embolism we may refer simply to what was said in the preceding chapter.

CHAPTER V

INFLAMMATION OF THE BRAIN

(Acute and Chronic Encephalitis)

1. Abscess of the Brain (Suppurative Encephalitis)

Ætiology.—In most cases of cerebral abscess it is possible to demonstrate with certainty that infectious material capable of exciting suppuration has penetrated to the encephalon. This is particularly true of those not very rare abscesses which follow injuries of the scalp, cranium, or brain (traumatic abscess of the brain). Here the wound is probably almost always an open one, affording free ingress to the infection. In the rare cases of traumatic cerebral abscess, apparently without any open wound, small injuries of the skin have probably been overlooked. It is not essential that the bones should be injured, however, for experience shows that, even where the soft parts alone are wounded, suppuration may extend to the brain. The manner in which this extension takes place determines the question whether a suppurative meningitis (*q. v.*) or an abscess shall be developed. Not infrequently we find the two combined. Another source of traumatic cerebral abscess is foreign bodies which penetrate into the brain (for instance, through the orbit), and thus carry septic matter into the very substance of the organ.

Besides traumatic causes, any existing suppuration in neighboring parts may, by direct extension, occasion cerebral abscess. The same processes are prominent in this connection which we have already found to excite purulent meningitis (*vide* page 1055)—particularly suppuration in the middle ear and in the petrous bone (chronic purulent otitis media, caries of the petrous bone). As a rule, the suppuration attacks the brain by contiguity, after the disease of the bone extends to the dura, and this becomes infected. In rare cases extra-dural abscesses are also formed. If the thin roof of the tympanic cavity is broken through, the abscess usually forms in the temporal lobe, while suppuration in the mastoid usually leads to cerebellar abscess. The excitants of suppuration may also reach the brain along the lymph-sheaths of the acoustic and facial nerves or by means of the perivascular lymph-sheaths and veins. About one third of all abscesses of the brain are due to purulent ear disease. The danger of the development of a cerebral abscess arises especially when the discharge of pus is checked by the growth of granulations, by the formation of cholesteatoma, etc., and also in acute exacerbations of chronic purulent otitis. Abscesses of the frontal lobe, following suppurative processes in the nasal cavity and the ethmoid, are much less common.

In yet a third class of cases the morbific agents are conveyed from foci of disease situated in distant parts of the body. These are metastatic or embolic abscesses. They occur, for instance, in pyæmia and ulcerative endocarditis. Abscesses of this sort, however, are usually small, and seldom are conspicuous in modifying the grave general disease. Of more importance are cerebral abscesses connected with certain suppurative affections of the lungs and pleura. These are not so very rare. They are oftenest associated with fetid bronchitis, pulmonary gangrene, and empyema. Purulent meningitis (*q. v.*) may occur in the same way. There can be no doubt that virus is in some way conveyed to the brain, but just how is not yet known.

In some few cases of cerebral abscess no certain ætiology can be made out. To these the term idiopathic is applied. We met with several of them just at the time of an epidemic of cerebro-spinal meningitis; and it therefore seems reasonable to suspect that possibly many apparently spontaneous cerebral abscesses are referable to the same poison as is epidemic meningitis. In other cases we have to do with other forms of infection.

From the variety of exciting causes, it is evident that in cerebral abscess the special variety of the purulent infection is not always the same. We find streptococci or staphylococci most frequently in the pus of an abscess, or under some circumstances the pneumonia diplococci.

Pathology.—The pathological anatomy of abscess of the brain is precisely the same as that of abscesses in other organs. The size varies from minute foci hardly as large as a lentil to extensive cavities occupying nearly the whole of one lobe. The traumatic and otitic brain abscesses are usually single, but metastatic abscesses are not infrequently found in considerable numbers. The pus is usually greenish yellow. It may be odorless or offensive. Sometimes remnants of the destroyed ("melted") nervous tissue and red blood-globules are mixed with it. The walls of the abscess often bulge out irregularly. The cerebral parenchyma around the abscess for a greater or less distance exhibits white softening. This softening is due partly to the pressure and partly to the spread of the inflammation. An abundance of granular cells is generally to be found in the tissues near the abscess.

If the abscess be very large and reach nearly to the surface of the brain, it may sometimes be recognized externally by a distinct bulging and perceptible fluctuation. The convolutions on the surface of the affected hemisphere are almost always flattened. If the abscess extends completely to the surface of the brain, purulent meningitis is excited. The complication of cerebral abscess with purulent phlebitis and sinus thrombosis has also been quite frequently observed. Abscesses which are centrally situated not infrequently burst into a lateral ventricle. An abscess of long standing may finally become encapsulated—that is, become encased in a smooth, firm layer of connective tissue, which prevents further extension of the process. The pus inside gradually becomes thick and cheesy. Apparently, however, it is very seldom entirely absorbed, and a new acute outbreak of inflammation, excited, for example, by an injury, is always among the possibilities.

Clinical History.—Small abscesses, and even large ones, may for a long time have scarcely any symptoms. This is particularly true of apparently idiopathic abscesses, and of those which develop very slowly and insidiously after apparently insignificant injuries of the head or in connection with chronic inflammation of the middle ear, etc. There is sometimes a long "period of latency" or a longer period of indefinite mild precursory symptoms (occasional headache, vertigo, general malaise, a slight elevation of temperature, etc.) preceding the onset of severe cerebral symptoms.

In abscesses following more extensive injuries, the symptoms are more violent from the start; this is also true of many abscesses which develop acutely and enlarge rapidly. The symptoms can hardly be distinguished from acute meningitis. The patient is dull, or grows delirious; he has violent headache, vomiting, symptoms of motor irritation (general or limited convulsions), and fever. Sometimes the temperature repeatedly rises to a high point. Definite focal symptoms (hemiplegia or monoplegia, partial cortical epilepsy, aphasic disturbances, hemianopsia, etc.) may manifest themselves according to the special situation of the abscess. The loss of consciousness becomes more and more complete; and in a comparatively brief time (one or two weeks) there may be profound coma and death. Rarely the violent symptoms abate, and the acute passes into a chronic stage.

The symptoms of cerebral abscess, when it pursues a chronic course, may be divided into (1) the general symptoms and (2) the focal symptoms, resulting from the special position of the abscess. There is no other localized cerebral disorder in which the focal symptoms are so often absent, either for a long time or even throughout the illness. This is partly because the abscesses are often situated in parts of the brain, injury to which does not occasion any demonstrable focal symptoms. Such localities are the white matter of the frontal lobe, the hemispheres of the cerebellum, etc. A second reason is that an abscess is not very apt to excite indirect focal symptoms by its influence upon the parts around it.

Among the general symptoms, the most important is a persistent, deeply situated, dull headache. For a long time it may be the only symptom, especially when the abscess is gradually developed after an injury to the head or after aural disease of long standing. The pain is referred mainly to the seat of the abscess; but the exceptions to this rule are not infrequent. The skull is sometimes especially sensitive to percussion in the vicinity of the abscess; but we should not lay too much stress upon this symptom. Another frequent symptom besides headache is vertigo; and there may also be vomiting, either after meals or entirely independently of food. Another valuable symptom in diagnosis is the irregular fever, sometimes slight, and sometimes exhibiting great elevations with intervals between them; but in many cases also, particularly where the abscess is encapsulated, there may be no fever whatever. The pulse is often slow. An important diagnostic point is that choked disk and optic neuritis may occur in abscess of the brain, but they develop far less frequently and only exceptionally do they reach so high a degree as in tumors of the brain (*q. v.*).

The general health may be but slightly disturbed. Usually, however, there is decided indisposition. The patient is pale, has no appetite, loses flesh, and sometimes shows slight mental changes (a melancholy condition, temporary confusion, etc.).

As to the focal symptoms, little need here be added to what is contained in

Chapter II of this section. Abscesses involving the motor region of the cortex have been repeatedly found to cause limited epileptiform attacks and monoplegic paralysis. It is particularly characteristic that, as the abscess grows, one symptom of paralysis is added to another, and that often the advance of the paralysis is ushered in by epileptiform convulsions. Abscesses in the occipital lobe have repeatedly been observed to cause hemianopsia, and abscesses in the temporal lobe word deafness; and these facts have been used in making a diagnosis of the location of abscesses. Abscesses in the cerebellum may remain unsuspected for a long while; but in other cases characteristic cerebellar symptoms (staggering gait, vertigo) may be evident as well as severe general symptoms.

The duration of chronic cerebral abscess varies greatly. In most cases it lasts for months; and sometimes it has certainly lasted for years. Particularly when the abscess gives rise to no symptoms, or merely to slight and indefinite ones referable to the head, it may last a very long time. It is quite often the case that there are separate attacks of the severe symptoms, such as headache, vomiting, and fever, and that in the intervals of variable duration between these paroxysms the patient feels pretty well.

The final termination of cerebral abscess, unless there is surgical intervention, is almost always fatal. Recovery is not impossible, but it has been seen with certainty in only a very few instances. Death either comes on gradually, when, as the abscess grows larger, all the symptoms become correspondingly aggravated; or it may happen quite suddenly. Sometimes it is brought about by the abscess bursting into a lateral ventricle, or by the supervention of meningitis. Often a cerebral abscess terminates in a sudden and unexpected death, where no immediate cause for the event can be found.

Diagnosis.—Although it is often possible to make a correct diagnosis of cerebral abscess, yet there is usually a good deal of difficulty in arriving at such a conclusion, and entire certainty is seldom attainable. The most important points in diagnosis are: (1) The demonstration of some cause, such as trauma, chronic otitis, fetid pulmonary diseases, or empyema. (2) The presence of general cerebral symptoms, such as headache, vertigo, and vomiting; and the fact that these are sometimes better and sometimes worse. To aid in excluding tumor, we have (3) the febrile symptoms frequently caused by abscess, but rare in case of tumor; and (4) the rarity of optic neuritis, which is very frequently occasioned by tumors. The focal symptoms, if there be any, are not characteristic. They grow worse by fits and starts in tumor just as in abscess. One fact, however, is of value, namely, that while tumors (*vide infra*) frequently cause disturbances in the area of distribution of the nerves at the base of the brain (such as paralysis of the oculo-motor), an abscess does this only exceptionally. It is often quite impossible to make a differential diagnosis between acute abscess and purulent meningitis, unless focal symptoms are developed, which indicate the existence of an abscess.

The timely recognition of an abscess of the brain following purulent otitis is of the greatest practical importance. It is important to know that cerebral symptoms (headache, vomiting, vertigo, stupor, etc.) may also occur from retention of pus in the middle ear, and that they may disappear completely after evacuation of the pus by paracentesis of the membrana tympani. If we find no retention of pus in the ear, however, we must suspect an abscess of the brain, if any persistent cerebral symptoms arise. Since such abscesses of the brain are most frequently situated, as we have said, in the temporal lobe or in the cerebellum, we should pay special attention to the localizing symptoms of these portions of the brain. Abscesses in the left temporal lobe may be diagnosticated with the greatest relative certainty by the discovery of aphasic disturbances, word deafness, verbal amnesia, optic aphasia, etc. (Oppenheim). Deep-seated abscesses may also cause hemiparesis, hemianopsia, ataxia, and, by pressure on the base of the brain, paresis of the oculo-motor or abducens. Otitic abscesses of the cerebellum cause occipital pain, slight stiffness of the neck, vertigo, and an uncertain gait; but not infrequently definite cerebellar symptoms are absent. The general cerebral symptoms (headache, vomiting, disturbances of the sensorium, etc.) are the same in otitic abscesses of the brain as in other varieties of abscess. We may also consider the local tenderness of the skull on percussion, usually above the upper border of the ear. In other cases, however, the diagnosis of otitic abscess of the brain is very difficult, especially if we can find no pronounced focal symptoms. We often can not avoid confounding it with extra-dural abscess, purulent meningitis, sinus thrombosis, etc. The presence of meningitis can sometimes be established by lumbar puncture.

Treatment.—The only possible way in which to cure an abscess is by operation; the skull is trephined and the abscess laid open. The earlier and the more accurately the diagnosis of abscess can be made, the more favorable is the outlook for the patient. With the present antiseptic modes of procedure, the dangers of an operation are slight, and the series of successful results that have already been obtained urgently demand further surgical intervention. Many a life can be saved by timely surgical interference, especially in abscesses of the brain following chronic ear disease. For particulars we must refer to special works on surgery.

If operation be not justifiable, nothing but purely symptomatic treatment is left us. Ice to the head, narcotics, potassic bromide, electricity, and sometimes also the local abstraction of blood, are the main remedies beyond general hygienic measures.

2. Acute and Chronic Non-suppurative Encephalitis

While the spinal cord is quite frequently affected by idiopathic circumscribed inflammation (transverse myelitis), analogous cerebral disease is much more exceptional. The scanty information which we possess upon the subject is as follows:

1. **Idiopathic (Inflammatory) Softening of the Brain.**—In rare instances there are found in the brain quite extensive foci of softening, the pathological anatomy of which is almost identical with that of embolic softening, and yet the afferent blood-vessels do not furnish any explanation of the condition. They have accordingly been termed "foci of inflammatory softening." The mode of their production is unknown. The symptoms resemble closely those of softening from thrombosis.

2. **Curable Form of Encephalitis.**—In certain cases pronounced symptoms of focal disease exist for a time, and suggest a tumor or the like; but after some months, or even a still longer time, the symptoms gradually abate, and finally there is complete recovery. Such cases are usually explained by assuming that there has been a localized encephalitic process, capable of a restoration to the normal state. The nature of the symptoms, as we have observed them, would seem to imply that the lesion is generally in the neighborhood of the cortex, for there is usually paresis of some part of the body, often associated with certain symptoms of irritation and impairment of speech. Possibly the recovery of these cases may be assisted by electricity and potassic iodide. It is always hazardous, however, to class such affections as "encephalitis," since we can hardly ever exclude absolutely other affections (especially syphilis).

3. **Diffuse Cerebral Sclerosis.**—Diffuse sclerosis of the brain is a peculiar disease, which is usually classed as a chronic inflammation. The whole brain may be involved, or the disease may be confined chiefly to a large part of one hemisphere. There is a very marked increase in the consistency of the brain-sub-

stance, so that it cuts like a tough piece of leather. The microscope reveals in many cases, but not in all, diffuse hyperplasia of the neuroglia. In a case which we recently examined there was a decided atrophy of the nervous fibers lying in the white substance of the brain.—The disease is rare, and its characteristic symptoms can not yet be stated absolutely. They are developed slowly. The most constant among them seem to be hemiplegia or paraplegia without much change in sensation; symptoms of motor irritation, such as epileptiform convulsions (sometimes general and sometimes unilateral), tremor, or single twitchings, which may be rhythmical or like those of chorea; and, lastly, dementia. When both hemispheres are involved, there are usually marked spastic symptoms in the lower limbs.

The disease has been observed especially in children and also in elderly people. Alcoholism and hereditary syphilis seem to be among the causes, but nothing definite is known upon this point. Transitional forms between diffuse sclerosis and pseudo-sclerosis apparently occur (*vide* page 959). Treatment can be only symptomatic.

4. **The Acute Encephalitis of Children. Cerebral Paralysis of Children.** (*Spastic Infantile Hemiplegia of Benedikt.*)—Children not infrequently suffer from a definite form of hemiplegia, which deserves a brief special description.

The patient is usually between one and four years of age. The symptoms almost always begin acutely. Having been previously healthy, the child is suddenly attacked with malaise and fever. Very often there is nausea and vomiting, speedily or at once followed by grave cerebral symptoms. There is stupor, and convulsions are particularly frequent. This condition may last but one or two days; or it may continue, perhaps with unabated severity, for two or three weeks. Then the acute symptoms abate, and the child recovers comparatively fast; but it is noticed by the parents to be paralyzed; and this paralysis seldom entirely disappears, although it may diminish. Sometimes the paralysis follows the convulsions, without any other definite encephalitic symptoms.

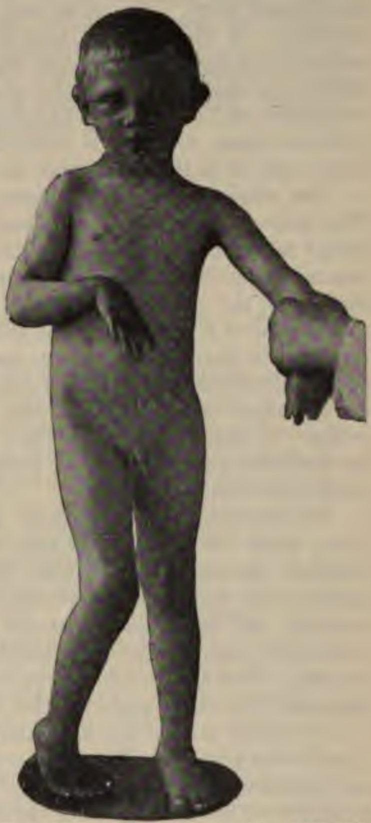

FIG. 176. — Cerebral infantile paralysis. Right hemiplegia. (Personal observation.)

If such children come under observation after the paralysis has lasted some time, as is usually the case, their condition is generally as follows: The cranial nerves, as a rule, are but little affected. The main change is in the extremities of one side; and the arm is almost always worse than the leg. The affected parts betray an arrest of development, more or less impairment of motion, in many cases a marked exaggeration of the tendon reflexes, and almost invariably contractures of more or less severity (compare Fig. 176). The muscles, although generally somewhat atrophied, never exhibit the reaction of degeneration. Sensation, as a rule, is unimpaired; but on careful examination we sometimes find in the paralyzed arm a slight diminution of the sensibility to touch, and especially a loss of the ability to recognize objects by the aid of the sense of feeling when the eyes are closed (see page 795). Permanent hemianopsia may also be associated with the hemiplegia. Symptoms of motor irritation are found upon the affected side with striking frequency; the commonest are athetoid movements (hemiathetosis, hemichorea); and associated movements are also not infrequent. The constant movements of the fingers resulting from the athetosis sometimes render the articulations so lax that it is possible to make the fingers assume an angle of ninety degrees or even less with the back of the hand, at the metacarpophalangeal joint. The child may keep making motions with its paretic arm while walking. It is not very rare for epilepsy to develop later. There are convulsive attacks, which usually begin on the paralyzed side, but which may later affect the whole body. With right hemiplegia we sometimes see co-existing disturbances and a defective development of speech. The intellectual development of many such children is tolerably normal. Others, however, are more or less demented, or betray defective moral sense.

The whole course of the disease suggests very strongly an acute encephalitis. The process is probably in most cases limited more or less completely to the motor region of the cortex, and hence it has been called "polio-encephalitis." * It reminds one forcibly of the acute poliomyelitis of children, with a difference merely in the localization of the disturbance. It is not impossible that the two diseases have a very similar, if not identical, ætiology. They can hardly be differentiated in their initial stage; but when further developed they can not be confounded, because the cerebral disease causes unilateral paralysis, leaves the electrical reactions unimpaired, and frequently causes exaggeration of the tendon reflex. It deserves mention that precisely similar phenomena may be presented by children during recovery from measles, scarlet fever, pneumonia, and other acute infectious diseases.

The pathological conditions of the early stage have not yet been studied. Long after the process has run its course, the affected portion of the cerebrum presents marked atrophy with cicatricial contraction. If the cortex has been affected we find a cicatricial depression, a loss of substance, a so-called porencephaly. At these places the pia is thickened. Sometimes limited cystic formations are found. The pyramidal tract exhibits a secondary descending degeneration. It is thus evident that the process is, from an anatomical point of view, precisely like the atrophy of the anterior cornua occasioned by poliomyelitis.

Of course not all the hemiplegias arising in childhood are due to an encephalitis, for in not a few cases infantile hemiplegia may be due to embolic softening, and perhaps sometimes to hæmorrhage. In some cases it is often hard to decide whether we should regard a cerebral infantile paralysis as the result of a previous encephalitis or of some other form of cerebral disease; but it seems to us highly probable that a large part at least of the very peculiar infantile hemiplegias must have their special cause, which we have assumed to be encephalitis. The chief thing to be considered in the diagnosis is the peculiar onset of the disease with fever, convulsions, etc., in a child who was previously perfectly healthy. This distinguishes encephalitic paralysis from hemiplegia existing since birth (congenital defect, injury at birth). [There is really little pathological evidence of acute polioencephalitis in children. Sachs and Osler, from the study of a large number of cases, find that the cause of the paralysis is almost always hæmorrhage or embolic softening.—K.]

The treatment at first is to be governed by the same rules as in the initial period of acute poliomyelitis (*q. v.*). The hemiplegic symptoms which persist

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[* This name, however, has been previously applied by Wernicke to that form of external ophthalmoplegia due to changes in the nerve nuclei in the pons (*vide* page 1045).—K.]

after the first few months will never improve much. The most we can do will be by means of electricity, massage, and cold baths with friction ("cold rubbing"). For the epileptic attacks large doses of bromide of potassium are decidedly beneficial. This may be combined with chloral, belladonna, etc.

5. **Acute Hæmorrhagic Encephalitis in Adults.**—Cases of acute hæmorrhagic encephalitis certainly occur in adults. They have been observed either as a sequel of other acute diseases (especially influenza) or as a primary acute disease. In such cases very severe cerebral symptoms (headache, loss of consciousness, hemiplegia) come on quite suddenly and lead to a speedy death with high fever. The autopsy shows several hæmorrhagic-encephalitic foci, usually confined to one hemisphere. The brain substance is yellow and softened, very œdematous, and studded with numberless little capillary hæmorrhages. Milder curable cases of acute encephalitis also seem to occur in adults.

CHAPTER VI

INSOLATION. SUNSTROKE. HEAT PROSTRATION. THERMIC FEVER

[**Ætiology and Pathology.**—Under exposure to undue heat, either in the direct rays of the sun, or, during hot weather, in engine-rooms, laundries, and the like, marked effects may be produced on the human organism, manifesting themselves in a widely different manner in different cases. The liability to these effects is much greater with us than in most portions of Europe or in Great Britain, and is enhanced by a moist atmosphere which tends to prevent evaporation from the surface of the body. Attacks may come on at night and under cover as well as by day. While excessive heat is the sole and sufficient exciting cause of the changes and symptoms to be described, exhaustion due to muscular exertion or other cause plays a very important secondary *rôle*. A vigorous person, however, of regular and temperate habits, can stand much greater heat and exertion than an individual who is debilitated or addicted to the use of stimulants. The frequency of attacks after a full meal has been noted. Those newly arrived in the country are, other things being equal, more likely to succumb than natives or those who have become accustomed to the climate. That high temperature due to solar or artificial heat, or a combination of the two, is the prime causative condition, has been clearly shown by experiments on animals. There is practically nothing to add to the researches of H. C. Wood.

Pathological Anatomy.—In cases of sudden death from shock there are no constant and peculiar lesions. The blood is dark, imperfectly coagulated, and collected in the veins; ecchymoses are frequently found.

After death, due chiefly to abnormally high temperature—that is to say, in cases of thermic fever—the heart, and especially the left ventricle, is firmly contracted from post-mortem coagulation of its myosine; the lungs are apt to be much engorged with dark fluid blood, and they may be the seat of hæmorrhage; extravasation of blood under the skin is more or less marked, and hæmorrhage into and about the cervical sympathetic ganglia has also been observed. The membranes of the brain and cord are often greatly congested, and there may be evidences of beginning meningitis. The blood-corpuscles are crenated and show a diminished tendency to the formation of *rouleaux*. Rigor mortis is marked and early in the voluntary musc [illegible] in the heart, and is attributable to the same cause. Parenchy [illegible] of the organs is sometimes found.

Symptoms and Course.—The onset of the attack is usually sudden, though there may be slight premonitions, such as dizziness, pain, or uncomfortable sensations in the head.

It will, perhaps, add to clearness to distinguish three leading forms of attack, it being understood that nature does not always classify cases as sharply as is done here. The first of these comprises cases of heat prostration, denoted by faintness, syncope, nausea, and sometimes vomiting, with marked muscular and general weakness. The surface of the body is cool, the pulse rapid and feeble. The great majority of these cases are mild, and the symptoms pass away more or less quickly on placing the patient in the recumbent posture in a relatively cool and quiet place with free ventilation. After a few hours the patient can be removed to his home, and in a day or two he has recovered perfectly, except for some sensitiveness to heat or the sun's rays. There may be transient insensibility, or, on the other hand, the case may terminate fatally very soon from general exhaustion and cardiac failure.

The second form includes cases with respiratory as well as circulatory failure, due especially to the exhaustion of the nerve centers presiding over these functions. Under this head comes cases of true sunstroke—patients suddenly losing consciousness while exposed to the sun. The skin is cold and the pulse is feeble; death may quickly ensue or recovery may follow, especially if prompt and suitable treatment be instituted. The after-effects of the attack may be very slow to pass away, and may never disappear entirely.

The most striking characteristic of the third form is the great increase in the temperature—a symptom which has given rise to the term thermic fever. In this form premonitions are more common than in the others. The thermometer may register 110° or even more; the skin is burning hot and generally dry; the pulse is slow and full, or quick and jerking; the respiration is quickened, sighing, or even stertorous; the pupils at first are usually contracted; there may be great restlessness; coma and convulsions sometimes occur; vomiting is common; and, toward the close of life, the sphincters are sometimes relaxed. A fatal result is due to apnœa and asthenia combined. What was said with regard to recovery from the second form holds good also for the third.

Diagnosis.—This is seldom difficult. The circumstances under which the attack comes on are generally patent; and the hyperpyrexia, if present, is distinctive. Acute alcoholism and meningitis are the chief affections which might lead to error.

Prognosis.—The mortality rate of the severer forms of the disease is very high, but the prognosis depends much on the possibilities of securing prompt and skillful treatment. Under this many a case recovers from a seemingly desperate condition. The tediousness and imperfection of recovery have been already alluded to. For long periods in some cases the mental and physical powers are much impaired, and great care has to be exercised as regards exertion, high temperature, and the sun's rays. Insanity is sometimes a sequel.

Treatment.—In the first place, much may be done by preventive measures to obviate the necessity for any treatment. A regular and temperate life will do much; and special precautions of an obvious nature should be taken during hot weather by those whose occupations involve a liability to exhaustion and exposure to unusual heat. It would be well if it were generally known that there is comparatively little danger of sunstroke so long as perspiration is free. Many an attack might be averted by noting the activity of the skin and seeking rest and shelter as sweating diminishes. A considerable responsibility rests upon militia surgeons and others in similar positions during hot weather.

Mild cases of heat prostration seldom require other measures than those already indicated in describing the symptoms. If there be nausea, and it seem desirable to give stimulants, they are better given under the skin or by the rectum. The clothing should be loosened; the cold douche and other refrigerating measures are not indicated unless there is fever, and they should then be used with caution.

For true sunstroke, treatment should be active and energetic, the indications being to reduce the temperature of the overheated centers and to stimulate their activity. If a cool or shady spot be near at hand, the patient should be removed to it and largely stripped of clothing; if not, no time should be lost before resorting to the cold douche on the head and body while stimulants are administered by enema or subcutaneously. External stimulation by mustard or flagellation, and purgative enemata, are said to be sometimes useful. The use of cold externally should not be prolonged in these cases unless there is fever. Nervous exhaustion is the prominent symptom, and all depressing measures are out of place.

The case is widely different with the third form of the attack. Here the immediate danger is from the hyperpyrexia, which must be combated by rubbing the patient with ice, placing him in a tub of water with lumps of ice, or similar measures, until the temperature in the rectum is reduced nearly but not quite to the normal point. In the application of the refrigerating measures the head must not be neglected. The sole indication at first is the reduction of the temperature. Antipyrine subcutaneously has been given in a few cases in the Boston City Hospital, and also in Brooklyn, with good results. After a reduction of the temperature, any symptoms of collapse or exhaustion demand stimulants.

With regard to the employment of bloodletting there is considerable difference of opinion. That cases occur in which this procedure is indicated is undoubtedly true, but they are exceptional; they are characterized by the evidences of great cerebral congestion without hyperpyrexia.

The subsequent management of convalescents from any form of sunstroke is often very important. Prolonged rest, and sometimes change of climate, may be demanded. A symptomatic and common-sense treatment is in place. It has seemed to the editor that quinine, especially in solution with a moderate excess of sulphuric acid, is distinctly useful in those suffering from mild or moderate aftereffects of undue heat.]

CHAPTER VII

TUMORS OF THE BRAIN

Ætiology.—The precise causes which lead to the development of tumors in the brain are no more definitely known than in regard to other organs. The new growths are usually formed insidiously and gradually without ascertainable cause in persons previously healthy. We are therefore disposed at the present time to assume that there is some abnormal embryonic predisposition of the tissues which acts as the special cause of the subsequent new growth. A circumstance which deserves mention is that sometimes the first symptoms come on immediately or a short time after some injury to the head. Especially in cases of sarcoma arising from the periosteum, the meninges, etc., is a traumatic origin (that is, the "setting free" of a predisposition to tumor by the injury) very probable, although, of course, hardly ever quite certain. Glioma is also referred at times to a traumatic cause.

Most cerebral tumors are found in persons in youth or middle life. Certain forms of tumor, however, particularly solitary tubercles, are comparatively frequent in children. Sex seems to exert a decided influence: men are much oftener affected than women.

Varieties of Cerebral Tumor.*—The most important forms of tumor seen in the brain are as follows:

1. GLIOMA.—Glioma is a kind of tumor peculiar to the central nervous system, but seen much oftener in the brain than in the spinal cord (*vide* page 1024). Apparently the new growth always originates in the neuroglia, which is the connective-tissue framework of the true nervous matter. As seen under the microscope, a glioma is made up of fibers and cells, the latter being like the normal cells of the neuroglia, while the fibers seem to consist mainly of the numerous cell-processes. Whether the ganglion-cells also take an active part in the new growth has not yet been proved. It is characteristic of gliomata that they are seldom sharply defined, but shade off gradually into the healthy tissue. The affected portion of the brain is usually enlarged, but yet maintains pretty nearly its original shape. On cross-section, the parts that have undergone "gliomatous degeneration" present a yellowish or reddish-gray surface. It is usually rather soft, and almost always is very vascular. This great vascularity is not without clinical importance, for variations in the fullness of the blood-vessels, and, above all, sudden hæmorrhages into the interior of the new growth, which not infrequently occur, may produce marked symptoms.

Gliomata are most frequent in the white substance of the cerebral hemispheres, but they are also found in the central ganglia, cerebellum, and elsewhere. They are usually single, seldom multiple.

2. SARCOMA.—It is very seldom that any form of sarcoma originates in the brain-substance. It usually begins in the connective tissue of neighboring parts, in the dura mater, the periosteum of the cranium, or the cranium itself (osteosarcoma). The sarcoma is found most frequently at the base of the skull, in the form of a circumscribed tumor of varying consistency, and by pressing upon neighboring parts, or by involving them in the diseased process, it may cause the gravest disturbances. Histologically, we have here such varieties as round-celled or spindle-celled sarcoma, fibro-sarcoma, etc.

3. GUMMA AND SOLITARY TUBERCLE.—Both gumma and solitary tubercle are very prone to attack the brain. We shall revert to gumma in the chapter on cerebral syphilis. Solitary tubercles may grow to the size of a cherry or larger. They may be single or multiple, and may occupy any part of the brain. They are found most frequently in the cortex, and in the cerebellum and pons.

Solitary tubercles and gummata, upon cross-section, have a yellowish, cheesy appearance, and are usually distinctly defined. Histologically, they are composed of granulation tissue. It was formerly no easy matter to distinguish gumma from tubercle in all cases; but now certainty is attainable by determining the presence of tubercle bacilli in the latter.

4. CARCINOMA.—Carcinoma completes the list of such cerebral tumors as have much clinical importance. It is almost always secondary in the brain. It has been our experience that secondary cancer of the brain is associated principally with primary cancer of the breast, or of the lungs and pleura; which fact bears a remarkable analogy to the occurrence of secondary cerebral abscess after primary empyema, pulmonary gangrene, etc.

5. Among the rarer varieties of tumor is *psammoma*, which usually starts from the meninges, is hard, generally comparatively small, and therefore often harmless, and contains calcareous matter, so that it grates under the knife.

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* From a clinical standpoint, the term "cerebral tumor" usually is meant to include also such tumors as originate in the neighborhood of the brain (as in the base of the skull), if they finally involve the brain itself.

The calcareous deposits are found in the form of roundish objects, which are composed of flat cells which lie over one another like the coats of an onion. Psammoma is probably of endothelial origin. *Cholesteatoma*, which has a sheen like mother-of-pearl, has also been observed in rare instances, and also *angiosarcoma, angioma*, etc. The not very rare growths arising from the hypophysis cerebri ("struma of the pituitary gland") remind us of adenomata in their structure.

6. In addition to the true tumors we may mention the *cysts* of the brain. They develop in very rare cases without known cause (perhaps as a sequel to cranial injuries?), they may grow to the size of an apple, and may cause precisely the same clinical symptoms as a tumor of the brain.

The General Symptoms of Cerebral Tumors.—As is the case with all focal diseases of the brain, some of the symptoms of cerebral tumors are connected with the special localization of the new growth. There are definite focal symptoms, varying with the part destroyed, or at any rate functionally impaired, and it is from these symptoms alone that we are enabled to determine the position of the tumor. But, in addition to these focal symptoms, there are certain general symptoms common to almost all cerebral tumors of any size. They are in large part referable to the general compression of the brain due to the enlargement of the new growth. In the first place, numerous clinical facts indicate that whenever there is a tumor of any great size, a large part of the entire encephalon is subjected to pressure; and, secondly, the anatomical appearances of almost every brain affected by a large-sized tumor lead to the same conclusion. The convolutions are flattened and obliterated, the dura mater is crowded against the cranium, and perhaps thinned or even perforated because of the persistent pressure, or, on the other hand, thickened as a result of chronic inflammation. Now and then the effects of pressure are visible even in the bones of the skull: they are worn thin, or even perforated, or their sutures are loosened. Another result of the general intracranial tension, through its effect upon the venous trunks of the brain, is serous effusion into the ventricles (internal hydrocephalus), which occurs very frequently. The largest effusions are caused by tumors in the posterior cerebral fossa, which directly compress the venæ Galeni.

The symptoms of cerebral tumors, referable to the effects of general compression, are as follows:

1. *Headache* is one of the earliest and most constant symptoms. It is usually persistent, although subject to temporary exacerbations and intermissions. The patient describes it as dull, deeply seated, and stupefying. Although it affects the whole head, it is sometimes (not invariably) referred mainly to the neighborhood of the tumor. It is particularly true of persistent occipital headache that it indicates a new growth in the posterior fossa. Sometimes also it is possible, by tapping upon the skull, to find an especially tender region. Considerable caution, however, should be used in drawing diagnostic conclusions from such observations. The headache usually lasts to the close of the disease, and, even after the patient has become completely comatose, the persistence of the pain is still evident from his low groans and the way in which his hand seeks his head.

2. Next in frequency to headache are *disturbances of the sensorium and intelligence*. Even the facial expression may be somewhat characteristic, being peculiarly languid, apathetic, and dull. The patient talks slowly, often having to think a long time before knowing what to say. Memory becomes impaired, especially with regard to the most recent events. The interest of the patient in those about him, and the things he used to care for, grows less and less. He has a sleepy, dazed look, and grows careless and untidy. There is sometimes pronounced somnolence. Of course, individual cases present various deviations from this outline; but in general, all cases are a good deal alike, although the degree of

mental disturbance may vary from the slighter forms of stupor to the highest degrees of mental weakness. We occasionally see other forms of mental disturbance in tumors of the brain, such as melancholy, hallucinatory confusion, states of excitement, abnormal attempts at wit (Witzelsucht), etc. In such cases, however, local disturbances (especially in the frontal lobe) are probably to be taken into account.

If unusual fullness of the blood-vessels, a hæmorrhage into the new growth, or some similar cause, induces a sudden temporary increase of tension, there may be such marked symptoms as syncope or an apoplectiform shock.

3. Other general cerebral symptoms are *vertigo, slowing of the pulse,* and *vomiting.* If, however, vertigo be a very prominent symptom, it implies that the cerebellum is especially encroached upon by the tumor. The retardation of the pulse is a frequent symptom, and not without diagnostic value. It has already been mentioned as one result of general compression of the brain, in connection with cerebral hæmorrhage. The rate may be put at about 50 to 60, or even lower. The pulse is also sometimes slightly irregular. Cerebral vomiting may be one of the earliest and most troublesome symptoms. It frequently occurs independently of the ingestion of food, especially in the morning, and is not infrequently associated with dizziness.

4. *Epileptiform convulsions* are sometimes excited by cerebral tumors, although many patients are free from them. Such attacks in all probability originate invariably in the cortex of the cerebrum, and it is therefore natural that they should be seen most frequently (as they are) in connection with tumors in the cerebral hemispheres. This rule, however, is not without exceptions. If the convulsions are not general, but are unilateral or confined to distinct portions of the body, they are to be regarded rather as focal than as general symptoms, and they may be utilized to determine the position of the lesion (*vide* page 1073). A certain amount of information in the same direction may also be obtained from those attacks which begin unilaterally or in one particular limb, and then quickly involve the entire body.

5. *Optic neuritis* ("choked disk") is one of the most important and common general objective symptoms of cerebral tumor, occurring in about eighty per cent. of all cerebral tumors. We should therefore never omit to make an ophthalmoscopic examination of the fundus oculi in a case of chronic cerebral disease. Some differences of opinion yet exist in regard to the special pathology of optic neuritis; but we may regard it as extremely probable that the main factor in its production is the purely mechanical one of general compression of the brain. This opinion is supported especially by the fact that even a marked choked disk may disappear completely in a short time, if a lessening of the intracranial pressure be brought about by trephining the skull and causing an abundant discharge of cerebro-spinal fluid. The original view of Von Graefe was that the increased intracranial pressure obstructs the venous return through the vena centralis retinæ into the cavernous sinus. The opinion which prevails at present is that advanced by Schmidt and Manz—namely, that the increased tension forces the cerebro-spinal fluid into the lymph-sheath of the optic nerve (Schwalbe), and that the "*hydrops vaginæ nervi optici*" thus produced compresses the nerve and the blood-vessels which traverse it. [Leber and Deutschmann have shown pretty conclusively that the increased pressure is not the chief cause of optic neuritis. It favors its production, but probably there must be the presence of some irritant as well, in order to produce the inflammation.—K.] At any rate, optic neuritis is not a focal symptom. The tumor occasioning it may have any position, if only it gives rise to general compression of the brain.

Disturbances of vision may be entailed by optic neuritis, but they are not a necessary consequence of neuritis; the patient may have amblyopia, defects in the field of vision, or even total blindness. Only in a few instances has amblyopia been one of the earliest symptoms of cerebral tumor, so that the patient has applied first of all to an oculist. Usually the sight is preserved for quite a long time, in spite of the abundant objective evidences of optic neuritis. The latter consist of swelling of the disk, marked distention and tortuosity of the veins, possibly hæmorrhages (from passive congestion), and cloudiness of the disk, although the retina still exhibits its normal transparency. It is not until the long-continued congestion impairs nutrition to such an extent as to cause atrophy of the optic nerve that vision is much impaired.

6. General *loss of flesh and strength* may sometimes appear comparatively early in cases of tumor of the brain. It is due in large part to the small amount of food taken, vomiting, and wakefulness; but it is not impossible that the grave cerebral disorder itself exerts a direct and unfavorable influence upon all the processes of nutrition. There is in most cases, also, a tendency to obstinate constipation.

7. In conclusion we may mention that, on examination of the *spinal cord* of persons who have died of cerebral tumor, degenerative changes have been found in the posterior columns and posterior roots (Dinkler, Mayer, and many others), besides the ordinary secondary descending degenerations. These changes are probably a consequence of the increased cerebral pressure and the stasis of cerebro-spinal fluid thus occasioned, and thus they may be classed among the " general symptoms " of tumor. As a rule they have no clinical significance; but an otherwise inexplicable loss of patellar reflex may perhaps often be referred to these secondary changes in the spinal cord.

Tumors in the Different Parts of the Brain and their Focal Symptoms.—The symptoms above discussed indicate the existence of a tumor, but not its particular location. Other phenomena are necessary to enable us to localize the disease, but it is not exceptional to have none but the general symptoms. Tumors in the white matter of the frontal lobe, or tumors of the corpus striatum or optic thalamus, as well as others, may run their course without any focal symptoms; but most cases afford evidence which points with more or less certainty to the exact position of the tumor. Almost all of these focal symptoms have already been fully discussed in Chapter II of this section, and their interpretation follows the rules for all focal lesions of the brain. We may therefore be brief here. It is necessary only to emphasize the fact that with regard to cerebral tumors, as well as other lesions, focal symptoms should be divided into the direct and the indirect. Direct focal symptoms are the immediate result of the destruction of nervous tissue by the new growth, while the indirect symptoms are excited by the pressure exerted by the tumor upon the parts closely surrounding it. This pressure varies with the amount of blood in the vessels of the new growth, and therefore the indirect symptoms may undergo temporary exacerbations and remissions. An intermediate position is occupied by those focal symptoms which occur in many cases as the result of certain anatomical changes secondary to the new growth. Not infrequently there is white softening of the substance of the brain around the tumor proper. Probably this condition is generally the result of a compression of the minute blood-vessels surrounding the new growth, but sometimes it is the sequel of an obliterative endarteritis (Friedländer). The latter cause is especially operative when the tumor is a gumma or a solitary tubercle. In vascular new growths also, especially in gliomata, hæmorrhages may occur which may often exert a destructive influence over a greater territory than the new growth itself.

1. *Tumors of the Cerebral Hemispheres.*—Tumors in the vicinity of the *central motor convolutions* are especially important, because they give the best opportunity for an accurate topical diagnosis and surgical treatment. The

first focal symptoms are usually attacks of partial epilepsy, such as have already been described in detail (page 1073). Monoplegia gradually sets in, which may gradually develop to complete hemiplegia. At first there is, for example, a brachio-facial or a crural monoplegia, and later the paralysis attacks the leg or the arm on the same side. The precise peculiarities of cortical spasms and paralyses have been described above (see page 1072). On careful examination, disturbances of the tactile and stereognostic senses can often be discovered in the paralyzed limbs. Ataxia and, with tumors of the left hemisphere, disturbances of speech may also occur. Tumors which involve the ***temporal lobes*** cause soul deafness or amnesic (sensory) aphasia. Tumors of the *occipital lobe* may be recognized by the occurrence of disturbances of vision (soul blindness, hemianopsia). In tumors of the *frontal lobe* Bruns and others have repeatedly observed a striking disturbance of the gait (a staggering gait, a tendency to fall to one side or backward, similar to cerebellar ataxia). This is probably connected with a disturbance of the motor centers for the muscles of the trunk, which are situated in the frontal lobe. Tumors of the brain not very infrequently arise in the *corpus callosum*, but these can hardly ever be diagnosticated with certainty. Characteristic focal symptoms are lacking; there are usually only the general symptoms of a tumor and especially deep disturbances of consciousness (stupor, somnolence, loss of intelligence, etc.).

2. *Tumors at the Base of the Brain.*—The base of the brain is a favorite seat of new growths. The symptoms are in a majority of cases quite characteristic. Some of the tumors spring from the base of the skull; among these are many sarcomata and syphilitic growths ("gummatous periostitis"). Other tumors originate in the meninges, especially the dura; and still others from the brain itself. Of these last, it is remarkable that some spring from the pituitary gland. The exact starting point is very seldom of much clinical importance, for all the parts mentioned are in such close proximity to one another that there is no great difference in the symptoms produced. We can decide merely that there is a tumor in this or that place at the base of the brain.

Tumors at the base of the brain owe their characteristic clinical stamp to the frequency with which the cranial nerves at the base are involved. The anatomical relations are such that these nervous trunks are often compressed by the new growth or actually incorporated in it. Of the symptoms thus occasioned, the most frequent is paralysis of the motores oculi (oculo-motor and abducens). This is at first usually unilateral, but it may later affect both sides. If one of the optic tracts be involved, hemianopsia may result, and pressure upon one optic nerve may produce unilateral choked disk with unilateral disturbance of vision. Tumors of the pituitary gland are especially prone to cause symptoms referable to the optic nerve at an early period (compare also page 902). Lesions of the trigeminus not infrequently cause disturbances of sensation in the face, and occasionally also paralysis of the muscles of mastication. The trunk of the facial often suffers. The facial paralysis thus occasioned throws considerable light upon the diagnosis, for the frontal muscle and the orbicularis oculi are involved (compare page 862), and there is usually to be found in the paralyzed muscles the reaction of degeneration, showing that the paralysis is peripheral. We have therefore reason to assume that the lesion is situated at the base of the cranium, rather than in the brain itself. Tumors arising from the posterior fossa at the base of the brain cause pressure paralysis of the lower cranial nerves (hypoglossus, accessory) and sometimes pronounced bulbar symptoms (disturbances of swallowing and articulation) from pressure on the medulla. As might naturally be expected, various degrees and forms of paralysis in the extremities are often found in combination with all the above-mentioned disturbances of the cranial nerves. Such conditions are most frequent when the crus cerebri, with its pyramidal tract, is affected.

There is no need of enumerating all the possible varieties of symptoms. We must consider them all carefully in each individual case, and then, by bearing in mind the anatomy of the parts, we shall, in a majority of instances, be enabled to determine with some approach to accuracy the place at the base of the brain where the new growth must be. Sometimes we may be led into error by tumors which, though situated in the brain-substance and at a comparative distance, yet by their pressure give rise to indirect symptoms referable to the cranial nerves at the base.

3. *Tumors in the Neighborhood of the Third Ventricle.*—Tumors sometimes develop, which are situated in a central position in the interior of the brain, especially in the neighborhood of the third ventricle, and therefore give rise to few well-marked focal symptoms. Besides the general symptoms of tumor (amaurosis from optic neuritis, stupor, headache, vomiting) there is general weakness of the extremities, often associated with spastic rigidity, which is usually only a little more developed on one side than on the other. As a rule, therefore, it is hard to make an accurate topical diagnosis.

4. *Tumors of the Cerebellum.*—Cerebellar tumors are comparatively frequent, and they can usually be diagnosticated with considerable certainty. The characteristic focal symptoms of all cerebellar diseases have already been fully described above (see page 1086). We will recall here in particular the feeling of vertigo and the staggering gait. But cerebellar tumors generally occasion also very strongly pronounced general symptoms—namely, headache, mainly occipital; sometimes a spasmodic and persistent stiffness of the neck; vomiting; and visual disturbances, due mainly to the frequent existence of optic neuritis. Analogous to this last symptom would seem to be disturbances in other nerves of special sense. Thus, when the general intracranial pressure is elevated, the acoustic and olfactory nerves seem liable to passive congestion. Tumors in the posterior fossa have several times been found to occasion bilateral anosmia and deafness; and they should always be considered when such a condition is found. Cerebellar tumors not infrequently as they continue to grow exert pressure upon the pons and medulla, and thus special clinical symptoms may arise: symptoms of paralysis or irritation on the part of the posterior cranial nerves, nystagmus, hemiparesis, hemiataxia, etc.

5. *Tumors in the pons, the crura cerebri, and the medulla* are to be judged according to the general rules for the localization of cerebral diseases.

We may also observe, by way of an appendix, that repeated attempts have been made of late to make use of percussion of the skull as an aid in the diagnosis of the seat of a cerebral tumor. Over a tumor which is not too deeply seated the bones of the skull are said to give sometimes on percussion a note which is not wholly dull but rather tympanitic. A sort of "cracked-pot" resonance has also been observed. Percussion of the skull, however, can hardly claim great practical significance, since the interpretation of the results is too uncertain. [A few attempts have also been made to employ the Röntgen rays to reveal the situation of the tumor, but the results thus far have not been very definite.

In view of the increasing importance of the focal diagnosis of tumors of the brain, it has seemed advisable to insert the following brief summary of the focal symptoms of tumors in different parts of the brain:

Prefrontal Region.—Marked mental impairment (abnormal attempts at wit [Witzelsucht] ?); symptoms of invasion of the central region (Jacksonian epilepsy, aphasia); staggering gait; disturbances of smell.

Central Region.—Jacksonian epilepsy; monoplegia; tactile hypoæsthesia and loss of stereognostic sense; motor aphasia.

Posterior Parietal Region.—Word-blindness; disturbance of muscular sense (?); homonymous hemianopsia.

Occipital Region.—Homonymous hemianopsia; soul-blindness.

Temporo-Sphenoidal Region.—Often a latent region. Word-deafness, if the left side be affected; disturbances of taste, smell, and hearing.

Corpus Callosum.—Often a latent region. Progressive hemiplegia, often bilateral from invasion; mental disturbances.

Optico-striate Region.—Hemiplegia; contracture. In posterior part, hemianæsthesia, homonymous hemianopsia, post-hemiplegic chorea, athetosis.

Crus Cerebri.—Crossed paralyses of the oculo-motor nerve and limbs.

Corpora Quadrigemina.—Oculo-motor paralyses; reeling gait; blindness (?); deafness (?).

Pons and Medulla.—Crossed paralyses of face and limbs, or tongue and limbs. Other cranial nerve lesions.

Cerebellum.—Marked cerebellar ataxia; marked vertigo and vomiting.

Base, Anterior Fossa.—Mental impairment; disturbances of smell and vision; exophthalmus.

Base, Middle Fossa.—Disturbance of vision; oculo-motor disturbances; hemiplegia.

Base, Posterior Fossa.—Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis.—Disturbance of vision, especially bitemporal hemianopsia; oculo-motor disturbances; acromegaly.

It also is of importance, in cases which may come to operation, to determine whether the growth be cortical or subcortical. Seguin has given certain rules which, however, are not absolute. "In favor of a strictly cortical or epicortical lesion are these symptoms, none of them having specific or independent value: localized clonic spasm; epileptic attacks beginning by local spasm, followed by paralysis; early appearance of local cranial pain and tenderness; increased local cranial temperature. In favor of subcortical location of a tumor: local or hemiparesis followed by spasm; predominance of tonic spasm; absence, small degree, or very late appearance of local headache and of tenderness on percussion; normal cranial temperature."—K.]

General Course of Cerebral Tumors.—The symptoms of these growths almost always cover a long period of time. Exceptionally a tumor remains latent till a hæmorrhage or some similar event takes place in it, giving rise to sudden and grave symptoms, and possibly to an equally sudden termination. The rule is, however, for the phenomena to develop gradually. According to the location of a new growth, either the general or the focal symptoms may come first into prominence. They generally occur in the order named. First of all is an ill-defined, deeply seated headache, and by degrees all the other general and focal symptoms follow after. The symptoms may vary repeatedly and greatly in severity, a fact due mainly to the varying pressure of the tumor on neighboring parts. Repeated mention has already been made of the sudden exacerbations which may come on, especially in case of the vascular gliomata.

The entire duration of the disease is usually at least some months, and may be one or two years or more. The termination is almost invariably unfavorable. Death may be rather sudden, or it may not come till after a long period of wretchedness. Fortunately, however, the lameness, blindness, and marasmus are frequently made less terrible to the patient because of his mental debility. Recovery is possible only when the growth is syphilitic. It is indeed possible that solitary tubercles may also end favorably, but the matter is still in doubt.

Diagnosis.—The main points in support of a diagnosis of cerebral tumor would be the gradual onset and continuous slow increase of the general symptoms above detailed—namely, headache, vertigo, vomiting, convulsions, dementia, etc. The most constant of these symptoms is the headache. They all indicate the development of some chronic brain trouble, a tumor being the most probable if there be no definite ætiology to suggest some other process, such as traumatism resulting in abscess, or syphilis. Much stress may also be laid on the optic neuritis, since this, as we have said, is much less often seen in other chronic brain diseases (abscess or softening) than in tumor.

The general symptoms indicate that a tumor of the brain exists; but, in order to learn the position of that tumor, we have to rely mainly upon the focal symptoms. Their gradual development and the way in which one new symptom is slowly added to another, also give further ground for the opinion that some continuously progressive disease exists, and most probably a cerebral tumor. Of diseases with a similar course, abscess is recognized especially by the slighter symptoms of general intracranial pressure and by the absence of optic neuritis, frequently by febrile symptoms, and by its ætiology (trauma, purulent otitis). Inflammatory and thrombotic softening, if they come on slowly, usually produce less general disturbance than do tumors; they hardly ever cause optic neuritis, and (unless of syphilitic origin) are much rarer before middle age than tumors of the brain. Sclerosis sometimes simulates cerebral tumor; but here also there is no choked disk; the disease lasts much longer (five or ten years, or more), and, inasmuch as the sclerosis is usually multiple, there is frequently too great a complexity of symptoms to warrant the assumption of one solitary lesion. [We must, however, bear in mind that, in about one seventh of the cases, tumors also are multiple.—K.]

Certain rare cases of chronic circumscribed meningitis can not be differentiated from a tumor. They generally occur at the base, lead to a considerable thickening of the tissues, and they may simulate all the symptoms of a new growth in this region. Occasionally, also, chronic hydrocephalus is confounded with tumor of the brain. We met with a case of dropsy of the fourth ventricle which presented during life a perfect picture of tumor of the cerebellum.

As to the nature of a new growth, we can not go beyond surmises. If the focal symptoms indicate that the tumor is in the substance of the brain itself, our first thought would be of a glioma, because it is by far the commonest sort of growth in that situation; and, as has been stated, certain peculiarities in the course of the disease (especially if new symptoms add themselves abruptly) would make glioma probable. If, on the other hand, the tumor be at the base, it is most apt to be sarcoma, which is the most frequent form of new growths here. When symptoms referable to the optic nerve occur noticeably early, a tumor of the pituitary gland is to be thought of. In all cases, and especially in tumors at the base of the brain, we should bear in mind the possibility of syphilis. The previous history and the entire body of the patient should be closely searched with this point in mind; its therapeutic importance need not be dwelt on.

One special form of tumor deserves a brief mention here—namely, large cerebral tubercles. The growth may be single or solitary, or it may be multiple. It is seen chiefly in childhood, and any chronic cerebral disorder in a child should suggest the possibility of such a growth. It is rendered all the more probable by the co-existence of the signs of tuberculosis elsewhere, as in the lymph-glands, lungs, or bones. The symptoms are analogous to those produced by other tumors. Among the most frequent phenomena are headache and convulsions. The latter are often unilateral. There may also be all sorts of focal symptoms, according to the locality of the lesion.

Prognosis.—Except gummata, all tumors of the brain have a very unfavorable prognosis. It is said that in very rare instances tubercular growths have been arrested or cured; but in practice we can never rely upon any such result. In all other cases recovery is next to impossible. The time intervening between the appearance of the first symptoms and death varies greatly, as has been said.

We should therefore be very cautious in predicting the duration of the illness. It seldom, however, exceeds one or two years, and sudden death without any warning may occur at any time.

[Tubercular growths are occasionally found at autopsies, which have become encapsulated, and have apparently existed for years without causing symptoms. In some cases, especially with tubercular growths in children, the symptoms diminish and the patient may live for years, suffering only from the blindness, etc., caused by the growth. Other cases may present only mild symptoms, and the patient may lead a fairly comfortable life for years.—K.]

Treatment.—Inasmuch as the nature of the tumor can not be determined with absolute certainty in any instance, antisyphilitic treatment should always be tried. Forty to seventy-five grains of mercurial ointment (grm. 3–5) should be used by inunction, and thirty to seventy-five grains (grm. 2–5) of potassic iodide should be given internally each day. If the new growth be syphilitic, much benefit may be obtained in this way. It must be confessed that the treatment is generally of little avail, because the tumors are not of a syphilitic character; although it may be that iodide of potassium sometimes has a temporary good effect upon other forms. A long-continued course of arsenic has also been recommended, in order to check the growth of the tumor. This remedy should be used especially in those cases where there is a suspicion of sarcoma or solitary tubercle.

Of late years many attempts have been made to remove tumors of the brain by surgical measures. In this respect the special favorable conditions of the individual case are chiefly to be considered. If we are able to make an approximately certain diagnosis of a tumor on the surface of the brain, especially in the motor cortex, surgical interference is warranted, and in some cases it has been attended with decided success, either permanently or at least for a time. Since there is no outlook for the patient's recovery without an operation, we should make an attempt at operation in every case where the tumor can apparently be diagnosticated with certainty and localized in the motor region of the cortex. We should, of course, never undertake an operation with too great expectations, since errors in diagnosis and unforeseen complications and incidents only too often arise. When there are marked general symptoms of pressure (headache, somnolence, optic neuritis) it may be well worth while, as a practical measure, to advise simple opening of the skull without further interference. In some cases the lessening of intracranial pressure that ensues has a very favorable effect, at least upon these general pressure symptoms of tumor.

Beyond what has just been indicated, treatment must be symptomatic. The headache is combated with ice-bags, narcotics, antipyrine, etc.; the convulsions require bromide of potassium or the inhalation of chloroform; the vomiting is lessened by rest in bed, opium, and bits of ice. The general care and nursing of the patient are very important, so that bedsores and the like may be avoided if possible.

APPENDIX

HYDATIDS OF THE BRAIN

It was stated on page 510 that the cysticercus cellulosæ, which originates from the tænia solium, may occur in great numbers in the brain. The cysticerci most frequently occupy the pia mater, but generally project downward into the cortex of the brain. The meninges not infrequently exhibit signs of chronic inflammation, and may present hæmorrhages, which are not always minute. If there are numerous cysticerci in the neighborhood of the ventricles, a varying degree of internal hydrocephalus usually develops. The individual cysticerci are usually enveloped in a capsule of connective tissue, but they may be entirely devoid of such a covering.

No characteristic clinical sketch of hydatids in the brain can be drawn, because the symptomatology of each case differs according to the number and position of the parasites. Sometimes cysticerci produce absolutely no symptoms, and are discovered incidentally at the autopsy. In other instances they cause a long and tedious illness. Epileptiform convulsions seem to be the most frequent symptom, and these must be due to the position of the cysticerci in the cortex of the brain. They may, like true epileptic attacks, come on only at certain times when the general condition is otherwise good; or permanent general cerebral symptoms may also appear, such as persistent headache, vertigo, and mental disorder. In a case recently under our observation the symptoms consisted solely of paroxysmal headache and uncontrollable vomiting. The diagnosis of suspected tumor was made. The autopsy showed a cysticercus the size of a cherry in the fourth ventricle with secondary hydrocephalus. Other cases have shown merely mental disturbances (confusion), staggering gait, vertigo, etc., so that the disease has been repeatedly confounded with hysteria. Special focal symptoms are only rarely present with cysticerci of the brain. It is a fact of great practical significance that death may not infrequently come on suddenly and unexpectedly in cysticerci of the brain. We have seen several such cases in persons who previously had not complained of feeling at all ill, or who had complained merely of slight headache, etc. In such cases the cysticerci are usually in the third or fourth ventricle, and probably cause sudden pressure upon the medulla.

The diagnosis can never be made with absolute certainty. The presence of cysticerci in the brain may be suspected when the above-mentioned severe symptoms occur in a butcher or other person who is from his calling especially exposed to infection, or who is known to have had or still to have a tapeworm, or in whom cysticerci have been demonstrated in some other part of the body, such as the skin.

We know of no remedy capable of destroying the cysticerci. Treatment, therefore, can be only symptomatic. [In a few cases, chiefly in Australia, the cysticerci have been successfully removed by surgical interference.—K.]

CHAPTER VIII

CEREBRAL SYPHILIS

Ætiology.—The importance of syphilis as an ætiological factor in many chronic diseases of the nervous centers has been repeatedly adverted to in preceding chapters; but, as we have already stated, the influence of syphilis upon the genesis of these diseases is shown in two essentially different ways. In one case there is a probable toxic, so-called post-syphilitic degenerative process of certain tracts of fibers and ganglion-cells (tabes, general paralysis); in the other case there is the formation of true tertiary syphilitic ("gummatous") new growths. Only this latter form of syphilitic cerebral disease will be spoken of here.

As a rule, genuine cerebral syphilis develops in the later stages of the whole syphilitic process, but sometimes cerebral symptoms come on by the end of the first year from the date of the initial lesion. Usually the interval is several years, and it may be ten or even twenty years after the beginning of the disease, before the first signs of the cerebral trouble develop. We therefore class cerebral syphilis generally among the "tertiary symptoms" of syphilis.

Liability to the disease does not seem to be influenced essentially by age or sex, except so far as pertains to the general distribution of syphilis. Even hereditary syphilis has been proved to cause diseases of the nervous system. But it can not be denied that a predisposition to cerebral syphilitic disease is often engendered by those influences which are apt to promote cerebral disease in general. Just as the position of syphilitic cutaneous lesions is often determined by external irritation at some one place on the skin, so the disease seems more liable to attack a brain which is exposed to certain unfavorable conditions than one which is perfectly normal and vigorous. Such conditions are inherited tendency to nervous diseases, various injurious mental influences, poisons, in a broad sense, and traumatism. It need hardly be said that even a previously sound brain does not enjoy immunity from the affection.

Pathology.—As far as has yet been ascertained, there are two chief forms assumed by syphilis in the brain: (1) a circumscribed, broad, flat syphilitic new growth like a tumor (gumma, syphiloma), and (2) a disease of the arteries of the brain, which is usually quite extensive. There is no essential difference underlying these two varieties. They may also occur in combination with each other. The disease of the blood-vessels is really a syphilitic new growth affecting the walls of the arteries.

The syphilitic new growths are yellowish or grayish-red, and frequently cheesy in the center. Their most frequent seat is the dura mater or the subarachnoid space whence they spread to the brain-substance or the neighboring vessels and nerves; but exceptionally they may originate in the substance of the brain itself. Histologically they are made up of granulation tissue of varying degrees of vascularity, presenting yellow spots usually visible to the naked eye. These spots are of firmer consistency than the rest of the growth, and have undergone coagulation-necrosis (have become cheesy). In the brain substance itself circumscribed, wedge-like gummata may sometimes develop, which closely resemble solitary tubercles; but the extensive, flat syphilitic new growths arising from the meninges (*meningitis gummosa*) are of much greater clinical importance. These are found most frequently at the base of the brain, especially developing from the region of the chiasma, or, more rarely, on the lateral portions of the brain (fissure of Sylvius) or on the convexity. In these more extensive new formations of tissue we usually find all the different stages of the process together, fresh granulation tissue, cheesy places, and finally the transition into contracting cicatricial connective tissue (scar formation).

The syphilitic disease of the arteries was first fully appreciated by Heubner, who has described it accurately. It is usually most pronounced in the arteries of the base of the brain, and especially in the middle cerebral artery and its branches. Even the unaided eye detects a grayish opacity in the arteries. They feel firm and stiff, and on cross-section their walls are found to be thickened, either uniformly or in some places more than in others. This causes no inconsiderable narrowing of the lumen, or even its obliteration, particularly if the last gap be closed by the formation of a thrombus. The microscope shows that the new growth originates chiefly in the intima of the vessel, where there is a hyperplasia of the endothelium, and a gradual transformation of it into a firm connective tissue. But the adventitia also undergoes a gradual thickening of considerable extent. Syphilitic endarteritis presents no distinctive histological characteristics. Entire certainty that the inflammation is syphilitic can be attained only by discovering other evidences of syphilis, either in the brain or elsewhere, or from the personal history and the previous course of the disease.

The great clinical importance of syphilitic endarteritis is due to its cutting off the normal supply of blood from the regions supplied by the diseased arteries. If the occlusion be complete, cerebral softening is inevitable, as in ordinary embolism and thrombosis of cerebral arteries; and, inasmuch as the middle cerebral artery is particularly liable to the disease, syphilitic softening is most often found in the region supplied by this vessel.

Clinical History.—The variety of the pathological processes and of their location produces a corresponding variety in the symptoms of cerebral syphilis; but certain types, already described in the main by Heubner, may be distinguished, and in many cases these present a picture which is very characteristic of cerebral syphilis.

1. *Basal Gummatous Meningitis. Basal Cerebral Syphilis.*—The development of a flat syphilitic new growth spread over the base of the brain, whose anatomical relations have been briefly described above, causes a form of disease which in many respects agrees with that of tumors of the base of the brain (*vide supra*, page 1120), but which also often shows certain characteristic peculiarities. The first symptoms consist of persistent headache, sometimes growing worse at night, vertigo, vomiting, and a certain mental dullness, which rarely, however, reaches such a high degree of stupor as is so often see in genuine tumors of the brain. States of marked maniacal excitement also occur at times. The patient often has an intense and striking polydipsia and polyuria. To these symptoms are soon added others, which are due to the invasion of the different basal cranial nerves by the morbid process. The optic nerve and the motor nerves of the eye (especially the oculo-motor) are almost always among the first to be affected. Disturbances of vision (limitation of the visual field, in some cases hemianopsia, blindness of one or both eyes), changes in the pupils, disturbances of motion of the eyeballs or eyelids, may now become manifest in various ways. Optic neuritis or choked disk may occur, but in general the ophthalmoscopic changes are decidedly less frequent in cerebral syphilis than in genuine tumors of the brain. It is a very characteristic circumstance that all the different symptoms on the part of the cranial nerves (especially the changes in the visual field, as Oppenheim has shown) may show great variations, which are undoubtedly connected with changes in the pressure from cicatricial contraction, etc. Of the other cranial nerves, the facial, acoustic, olfactory, and trigeminus are most readily affected, but we should always make a very complete examination in every way in order to estimate correctly the extent of the process.

The further course of the disease may vary very much. In fresh cases, which are recognized and treated in time, there may be decided improvement, and even complete recovery or at least an arrest of the morbid process; but, on the other hand, the symptoms may increase. If there is also syphilis of the arteries (*vide infra*), there may be a slowly developing hemiplegia, hemiplegia following apoplexy, epileptic states, bulbar symptoms, etc.—in short, a complicated form of cerebral disease, which is no longer capable of any material change.

2. *Gummatous Meningitis and the Formation of Syphiloma on the Convexity of the Brain and in the Vicinity of the Fissure of Sylvius.*—In this somewhat less frequent localization of the syphilitic new growth prodromata similar to those just enumerated precede the severer symptoms. Then appear violent partial or general epileptiform convulsions. These often come on very suddenly, and they may recur at considerable intervals or in quick succession. There are usually still other symptoms of cortical disturbance, especially paresis of one limb or even of one half the body; very frequently slight disturbances of speech (stumbling over syllables), referable to the cortex, and indications of mental impairment. Optic neuritis as a rule is not present. Many of these cases reach a fatal termination comparatively early. The epileptiform convulsions become more and more frequent, and unconsciousness increases to deep coma ending in death. But in cases of precisely this type prompt and energetic treatment may accomplish a great deal.

3. *Cerebral Syphilis Involving the Cerebral Arteries Chiefly.*—A third common and important variety of cerebral syphilis is characterized chiefly by syphi-

litic arteritis. Not infrequently there is a prodromal stage, which is sometimes only slight; then, as a result of the occlusion of some vessel, which often occurs quite suddenly, there is a pronounced apoplectic attack, followed in most cases by hemiplegia. The intensity of the initial shock may vary greatly; sometimes there is only a slight dizziness, sometimes there is a coma that lasts for days. Sometimes the shock is succeeded by a peculiar condition of mental confusion and dullness, which may persist for weeks. In severe cases, death is speedy, and is usually ushered in by a great rise of temperature. Other patients improve more or less rapidly, especially under proper treatment.

Apoplectic attacks of this sort may recur after temporary improvement has taken place, and may be associated with all sorts of nervous symptoms.

4. *Complicated Cases of Cerebro-spinal Syphilis.—Combination of Gummatous Syphilis and Syphilitic Degeneration of the Nerves (Tabes, General Paralysis).*—Besides the three types of cerebral syphilis already described there are also many cases which belong in part to one of the types mentioned, but which, on the other hand, exhibit much more complicated symptoms on account of the far greater extent of the anatomical changes. In the first place we will mention the combination of cerebral and spinal symptoms. The latter are often due to gummatous spinal meningitis also, which is very often situated in the cervical region of the cord, and which may lead to paraplegia, brachial paresis, symptoms of unilateral lesion (*vide* page 1036), shooting root pains, vesical disturbances, etc. With these symptoms, all of whose varying details it is impossible to speak of here, are not infrequently associated cerebral symptoms, which, for their part, correspond to the conditions above described. A considerable number of cases which have been studied anatomically of late are also of especial interest. In these cases the symptoms of gummatous syphilis were associated with genuine tabes, or in other cases cerebral symptoms ensued, which were of the type of general paralysis (weakness of memory, a peculiar disturbance of speech, twitching of the facial muscles, paralytic attacks, etc.). In such cases the multiplicity of possible details is inexhaustible; but the characteristic peculiarity of the type lies in this regular irregularity, which often admits a positive recognition of syphilis of the nervous system and is also capable of a clinical and anatomical interpretation, with proper consideration of the nature of the disease.

Diagnosis.—The most important diagnostic criteria have already been mentioned in the description of the symptoms. Here, too, the diagnosis is first topical and then ætiological and anatomical. The distinction between syphilis and genuine tumors is the most difficult. Absence of optic neuritis, a marked variability in the severity of the symptoms, and multiplicity of symptoms, especially the combination of symptoms demanding different localizations, are in favor of syphilis. The discovery of a previous syphilitic infection is, of course, always important. We can not here describe in detail the methods of determining this fact. The history of the patient, as well as previous specific affections—such as, in women, miscarriages, abortions, etc.—and the objective signs on other parts of the body, are the two sources of information. There may be scars on the skin or mucous membranes, enlarged glands, ulcers, tibial periostitis, or changes in the testicles. Age is also important: thus an apoplectic attack in a young person would suggest syphilis, because the other causes of such an attack operate chiefly upon the aged. The results of treatment often throw considerable light upon the diagnosis. As there is nothing to lose and much perhaps to gain, we should always give specific remedies in doubtful cases. If they prove successful, the diagnosis of syphilis receives strong confirmation.

Prognosis and Treatment.—There are few severe and dangerous diseases in which timely and appropriate treatment is attended with so much success as that achieved in many cases of cerebral syphilis. In order, however, both to understand the favorable results and not to be misled by the failures, we need to gain a clear idea of the way in which antisyphilitic remedies can be of benefit. They can accomplish this only by causing the dissipation and absorption of the new growth—that is, the gumma or the swelling of the intima. If this be effected, the surrounding parts are of course relieved from pressure, and the circulation becomes unimpeded. If the tissues still retain functional power, they resume their duties and all symptoms of disease vanish. But when the tissues have already been considerably impaired by the compression, or by the scanty blood-supply, the results are quite different. Even then the degenerated nerve trunks at the base of the brain may gradually become regenerated; but such parts of the true cerebral parenchyma as have undergone softening have lost their functional capacity forever. In such cases, antisyphilitic treatment is unavailing.

It is therefore obvious that the first essential of success is to begin treatment as early as possible. The sooner a correct diagnosis is reached, the sooner will existing symptoms be relieved, and further danger be averted. The method of treatment which will probably accomplish all that can be accomplished, and in the shortest possible time, is energetic mercurial inunction. At least a drachm (grammes 3–5) of mercurial ointment must be rubbed in every day at first, according to the ordinary method. We should not venture to restrict the diet unless the patient be well nourished and "full blooded." If he be anæmic and feeble, a generous regimen is demanded. Usually the internal administration of iodide of potassium is combined with the inunctions; we should give thirty to forty-five grains (grammes 2–3), or, in severer cases, even a drachm or a drachm and a half (grammes 4–6), daily [In this country iodide of potassium is given much more freely. In cases where syphilis is suspected, we may safely begin with doses of thirty grains (grammes 2) three times a day. If the symptoms be urgent, the dose should be rapidly increased until the patient takes three or even four drachms (grammes 10–15) three times a day. If the patient have syphilis, the danger of iodism seems less. The drug must be given in large amounts of water, and it is better borne if given in Vichy or Giesshubler water. Sometimes the stomach will not tolerate the extreme doses.—K.] The same remedy should also be given afterward, for a long time, in smaller doses. When there is no benefit at all after twenty or thirty inunctions, there is little prospect of any appreciable improvement. In favorable cases, the mercury often begins to produce some effect after the fifth or sixth inunction; and it may cause astonishingly rapid improvement. Potassic iodide alone is sufficient for the milder cases only, where there is merely headache, trigeminal neuralgia, or paralysis confined to the motores oculi. In many cases it may be desirable from other reasons to carry out the treatment at some health-resort rather than at home (Aix, Tölz, Hall in Upper Austria, Wiesbaden, etc.). These resorts are also suitable for after-treatment or for repeated courses of treatment, but the treatment by drugs is always much more important than the baths.

CHAPTER IX

PROGRESSIVE GENERAL PARALYSIS OF THE INSANE

(*Paralytic Dementia. Paretic Dementia. General Paresis*)

Preliminary Remarks.—Although the description of mental diseases is not a part of the plan of this book, we must nevertheless make an exception of one disease of the sort, namely, the so-called progressive general paralysis of the insane, or paralytic dementia, which in medical parlance is often abbreviated into "general paralysis." We consider it advisable to make this exception, because a great part at least of the symptoms of general paralysis are purely of a physical nature, and also because a knowledge of this disease, which is so common and so fatal in its results, is of the greatest importance for the general practitioner.

We must thank the French alienists Boyle (1822) and Calmeil (1826) for the first clinical descriptions of general paralysis, by which it was more sharply differentiated than previously from the diseases which resemble it. A more accurate knowledge of the different symptoms, and the anatomical changes to which the morbid symptoms must be referred, has, however, only of late years been rendered possible by the introduction of better methods of investigation. Accordingly, we must now say that general paralysis is a disease which may attack the most diverse portions of the whole central nervous system *—the brain and spinal cord—at the same time or successively, although we can, of course, make out certain rules as to the predisposition of individual portions to disease, and as to the order of the symptoms. General paralysis begins most frequently in those regions of the cerebrum which have an immediate relation to the regular course of the psychical and certain psycho-motor processes. Mental and motor symptoms accordingly form the introductory features of the disease in most cases. More extensive regions of the central nervous system are gradually involved in the morbid process, which goes hand in hand with a progressive degeneration of all the higher intellectual faculties, while at the same time many physical disturbances dependent upon nervous changes constantly increase.

Ætiology.—General paralysis is a common disease, and apparently demands a heavier quota from the better and more highly educated classes than from the lower classes. We may assume that, on the average, one tenth of all patients committed to the insane asylums are general paralytics. In most patients the beginning of the disease falls in the period between the thirtieth and fiftieth year. The disease is much rarer in advanced life. In young people under twenty it has hardly ever been observed. [Over one hundred cases of juvenile general paralysis have been reported, almost always in victims of hereditary syphilis.—K.] There is no doubt that the male sex is much more frequently affected than the female, but the number of cases of general paralysis among women is not very small.

What is the special cause of general paralysis? A generally acceptable answer can not be given; but the theory is constantly gaining ground, and we agree to it, from our own experience, that by far the most important causal factor is a previous syphilitic infection. Such an infection can be made out in at least seventy-five per cent. of all cases. In this regard precisely the same conditions exist, and, of course, the same difficulties in the interpretation of this relation are to be considered, as we have previously mentioned in the account of the dependence of tabes dorsalis on syphilis (see page 961), a circumstance which again is not without significance, since the most intimate points of connection are to be found between tabes and general paralysis (*vide infra*). If we consider that general paralysis depends upon a previous syphilis, we have an easy explanation of most of the other peculiarities in the onset of the disease, especially the above-mentioned influence of age and sex, the decidedly common occurrence of the disease in persons in certain callings—such as artists and officers—the frequency of the disease in large cities in distinction from its rarer occurrence in the country, etc.

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* At present hardly anything is known in regard to a primary implication of the peripheral nerves in the general process. The principle of such an implication would be very readily understood in view of the almost regular occurrence of degeneration of the peripheral nerves in tabes.

Besides the ætiological factors named, which, in our opinion, are the most important, all other " causes " of general paralysis may well be regarded as merely predisposing. Mental over-exertion has the greatest significance, especially if it be associated with psychical irritation. In merchants, civil officers, etc., who suffer from general paralysis, such a previous over-exertion can often be made out. In some cases injury to the head or insolation is claimed to be the cause. Hereditary predisposition to nervous diseases plays perhaps a certain part in the origin of general paralysis, but by no means a very large one.

Clinical History.—General paralysis usually begins so slowly and gradually that a definite period for its beginning can hardly ever be given. In addition, it is often clear, at a time when the disease is already fully developed, that certain early symptoms, whose nature was at first not correctly recognized, ought to have been regarded as the initial symptoms.

The first symptoms of the disease in the psychical domain usually consist of the gradual appearance of a change in the whole nature and in the mental individuality of the patient; wherein, however, the mental disturbance usually shows from the start the character of weakness—that is, of a lessened capability of mental exertion. The patient's ordinary mental work no longer goes on as easily as before. His memory is uncertain, and there are marked forgetfulness and inattentiveness, which were previously quite impossible for him to exhibit. The patient is often disorderly in his dress, and violates the ordinary social rules of decency and morality. Since his judgment as to the value and significance of things is uncertain, he commits purposeless actions, wastes money, commits crimes, is dissolute, etc. In these respects, too, the increasing mental dullness often appears, since the patient becomes incapable of any higher intellectual, æsthetic enjoyment, and since the sway of nobler feeling finally becomes feeble, and unable to exert any lasting influence upon his actions. Besides all these signs of beginning mental weakness, we often notice, on the other hand, an abnormal irritability. The patient easily becomes agitated, or gets angry; but these moods rapidly pass away without leaving a lasting impression. We easily understand how this change in the whole personality of the patient must distress and alarm his family, since the relatives at first can not understand at all why the patient is now " so different from what he was."

In the first period of the disease a subjective feeling of illness is frequently present. The patient himself notices that his mental capacity, especially his memory, is diminished, and he very often becomes extremely anxious on this account. It frequently happens that certain subjective sensations are also noticed, a feeling of confusion in the head, pressure in the head, dizziness, rheumatoid pains, etc. The sleep is disturbed, as a rule, and also the appetite and the digestion. If such a patient comes to the physician with his complaints, it unfortunately only too often happens that he is in the beginning regarded as " neurasthenic," and is treated accordingly.

Careful observation, however, may usually even now discover the disease with certainty. The beginning mental disturbance is usually more apparent to the family than to the physician, who has not known the patient before and who sees him only cursorily, but it is apt to become manifest on a somewhat more searching examination of the patient. We generally succeed best by making the patient reckon; he often makes the greatest mistakes in simple examples in multiplication; especially does he fail in mental arithmetic, etc.

Certain motor symptoms, however, which usually come on in the early stages of the disease, are of the greatest diagnostic significance, especially peculiar disturbances of speech and handwriting. The paralytic disturbance of speech shows itself first in the form of stumbling over syllables (*Silbernstolpern*), or literal ataxia. The individual sound (in distinction from bulbar paralysis) can be pronounced quite correctly, but the combination of different sounds in the whole word causes increasing difficulties. It is a good plan, in order to recognize the first beginnings of this symptom, to have the patient pronounce a few difficult words, such as "third riding artillery brigade," "representative government," "initiative," "electricity," etc. We often hear "artralleriry" instead of "artillery," and like blunders. In the later stages of the disease the speech is sometimes almost wholly incomprehensible. We also observe other more complicated aphasic disturbances, such as paraphasia, persistent repetition of the same word, etc. In such cases the patients are sometimes no longer able to read any sentence correctly. They at times put in entirely different words, so as to make utter nonsense; but they do not notice it themselves. The abnormal associated movements of the facial muscles on speaking are also often to be observed, and are very characteristic. The voice of general paralytics often loses its power of modulation and becomes weak and rough—symptoms which depend upon a defective innervation of the vocal cords. The change in the handwriting, to be observed in general paralytics, is even more characteristic than the disturbance of speech (see Fig. 177). This is at first purely of a motor nature; the letters are uncertain, irregular, and tremulous. A psychical factor, however, also shows itself; single letters are omitted, the dot on the *i* and the marks of punctuation are forgotten, the patient ceases to keep on the lines, or leave a free margin, etc. As the disease advances, the disturbance in the handwriting gradually increases, so that the writing may finally become wholly illegible, and may consist merely of senseless scratches.

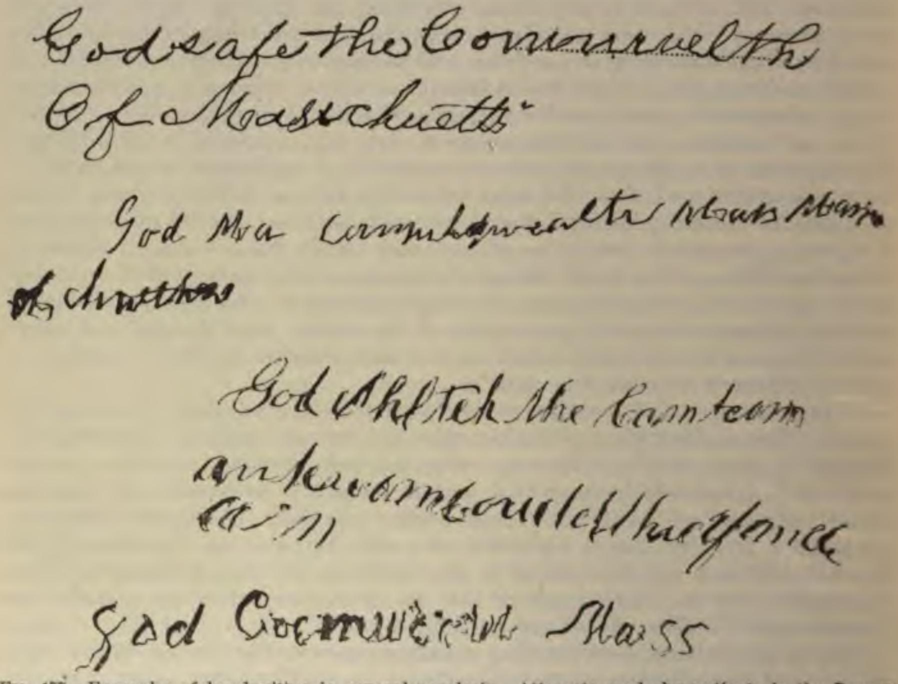

FIG. 177.—Examples of handwriting in general paralysis. Attempts made by patients in the Danvers Insane Hospital to write "God save the Commonwealth of Massachusetts." Besides the motor disturbance, the frequent omission of certain letters will be noticed, e. g., "Masschuetts" for "Massachusetts." In 3 and 4 the handwriting is almost wholly illegible.

Besides the changes in the speech and the handwriting, which we have just briefly described, other physical disturbances are often quite early symptoms, and prove in how many parts of the nervous system at once the disease may begin its work of destruction. The condition of the pupils especially is of importance in diagnosis. They are often unequal, and also show a reflex immobility (see page 972) in a large number of cases, especially in those in which other tabetic symptoms develop (*vide infra*). Transitory ocular paralyses are at times early symptoms. Not infrequently we find quite early changes in the tendon reflexes, very often a decided increase of the patellar reflex, or, in other cases, absence of the patellar reflex, which latter has a much more positive value in diagnosis—it is almost invariably a tabetic symptom (*vide infra*).

The association of reflex immobility of the pupils with an increase of the patellar reflex is not uncommon. In the distribution of the sensory nerves we may mention as repeatedly observed symptoms, neuralgia, attacks of migraine, and finally optic atrophy, the latter usually as one symptom of a co-existing tabes.

We can not give a generally applicable account of the further course of general paralysis, since it may differ decidedly in this respect. In what follows we can point out only the main features of the different types of the disease, and, in particular, we will sketch very briefly the groups of mental symptoms.

We often say that those cases belong to the "classical form" of general paralysis where an initial "stage of depression" with a melancholy tendency is followed by a second stage of "maniacal exaltation." This is the stage where the delusions, which are already quite pronounced, assume more and more the character of "grand ideas," and thus exhibit the "delusions of grandeur" which have for a long time been generally regarded as ominous. The first signs are often found in the patient's statements that he is now much better, that he is "very well," that he feels "very strong," etc. These delusions, however, often assume gradually a more exaggerated form; the patient considers himself enormously rich, he owns thousands of palaces, millions of dollars, has made the greatest inventions, considers himself the Emperor Napoleon, Christ, a "higher God," etc. Any judgment as to the absurdity of these ideas, and as to the sad contrast between his claims and the reality, has already become impossible to him; but, of course, there are even now occasional remissions in this condition, when the patient is clearer and recognizes temporarily the morbid character of his delusions.

We must not think, however, by any means, that the delusion of grandeur is necessarily an invariable symptom in general paralysis. In many cases (the so-called "depressive" form of general paralysis) the initial melancholic-hypochondriacal condition continues. The delusions that appear possess the same coloring; the patient claims that he can no longer eat, that he is poisoned, that he has lost his head or an arm, that he is very small ("*delire micromaniaque*"), etc. Sometimes acute and severe conditions of anxiety come on. In other cases, again (the agitated or maniacal form of general paralysis), there are states of violent excitement, in which the patient raves loudly, cries, and tries to destroy whatever falls in his way. Such states sometimes alternate with delusions of grandeur. Finally, we see cases of patients—and this is the most frequent form—who, in their mental relations, present simply the symptoms of a mental enfeeblement gradually increasing to complete dementia, without ever showing, in any notable form, states of excitement, the development of delusions, etc.

While the intellectual life progresses to utter ruin in the ways just stated, the physical disturbances of the disease, as a rule, gradually advance from bad to worse. In many cases ataxia of the extremities develops, and also loss of sensibility and vesical disturbances—in short, the symptoms of tabes. In these cases the tendon reflexes are almost always lost, and the pupils are often immobile. In other rarer cases, however, there is actual paralysis, first in the lower and then in the upper extremities. In these cases the tendon reflexes are often increased, so that the picture of "spastic paralysis" develops. Again, in other cases bulbar symptoms appear, such as disturbances in swallowing or masticatory paralysis, and also ocular paralyses, usually as a part of the tabetic symptom-complex, etc.

Peculiar attacks, which are among the commonest and most characteristic symptoms of general paralysis, are, however, of special interest, and sometimes even of marked importance in diagnosis. These "paralytic attacks" in their milder degrees sometimes appear even in comparatively early stages of the disease. Then they usually consist of attacks of vertigo, an obscuring of consciousness, or even a loss of consciousness, coming on quite suddenly, and lasting from a few minutes to half an hour or more, and they are not infrequently associated with mild hemiplegic or monoplegic symptoms. We very often see, besides the vertigo, a temporary feeling of weakness in the right arm, associated with a marked aphasic disturbance of speech. To this there is often added some slight twitching in the affected extremities or in the face. In the further course of the disease the attacks usually increase, and are termed apoplectiform or epileptiform paralytic attacks, according as the conditions of paralysis or spasm predominate. The epileptiform attacks may often be repeated with great frequency—thirty or forty attacks a day or more—during which time the patient remains in an unconscious state. If the patients gradually return to consciousness, sometimes only after a week or two, we very often see, as a result of such severe attacks, a permanent impairment of the general condition, an increase of the dementia, etc.

The other organs, apart from the nervous system, are only secondarily implicated in the morbid process. It may be mentioned briefly here that formerly, for a long time, stress was laid upon certain changes in the pulse, the pulsus tardus especially being thought to be characteristic; but the numerous investigations of the pulse have so far not given us any significant or sure results.

The temperature as a rule is approximately normal, or often somewhat subnormal, but very marked changes in the temperature occur in connection with the paralytic attacks—sometimes elevations and sometimes very deep declines.

The whole duration of the disease is in some cases only a few months (the "galloping" form of general paralysis), usually it is two or three years, and sometimes much more. The most rapidly fatal form is that in which there is very soon a marked emaciation and a rapid loss of strength as a result of the sleeplessness, the constant unrest, and the refusal of food. In other cases death ensues from the gradual and general loss of strength, or in a paralytic attack; or, finally, as is frequently the case, from the onset of secondary conditions, such as severe bedsores, pyelo-cystitis, tuberculosis, intestinal diseases, etc.

Pathological Anatomy and Nature of the Disease.—Considering the great difficulty of an accurate microscopic examination of the brain, it is not strange that our knowledge of the pathological anatomy of general paralysis is still very defective. If we except occasional immaterial changes in the skull, such as hyperostoses and the like, or in the meninges, such as hæmatoma of the dura or secondary thickening of the pia over atrophied portions of the brain,* the first striking and at any rate most important anomaly seems to be the atrophy of the brain which affects chiefly the anterior half, especially the frontal lobes. In this region the convolutions are very much diminished, and the fissures are wide; the weight of the anterior portion of the brain may be reduced to one fourth or one third of the normal. If we examine the convolutions microscopically we find that the diminution of the whole organ depends chiefly upon a loss of nervous elements. The cortex usually shows the greatest changes. In fresh cases we sometimes see the signs of a mild " inflammation "—that is, we find vascular dilatation and little disseminated foci of round cells about the vessels, but the changes in the nervous elements themselves are far more important, and consist essentially of a degenerative atrophy. We must mention especially that in the cortex of the frontal lobes, especially marked in the straight convolution and in the island of Reil, and also in other portions, we can make out with certainty, by the aid of good methods of examination, a very considerable loss of the fine medullary nerve-fibers, mainly those which run parallel with the surface, and hence are termed " association-fibers " (Tuczek); but signs of degeneration and atrophy are very often to be seen in the ganglion-cells themselves. The destruction of the nervous elements is regarded by many investigators as secondary, since they lay the chief stress upon the marked changes in the interstitial tissue, numerous spider-cells, or thickening of the vascular walls, which are almost always to be found in old cases, and hence they speak of an interstitial encephalitis (Mendel). We ourselves, with Tuczek, Wernicke, and others, are much more inclined to the theory that we have to do mainly with a primary process of degenerative atrophy of the nerve-fibers and nerve-cells, to which the increase of the connective tissue is only a secondary addition.

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* [An adhesion of the pia mater to the brain substance is very characteristic. This adhesion may be so firm that bits of the cortex are torn away on endeavoring to separate the pia from the brain.—K.]

The anatomical affection in general paralysis, moreover, is by no means limited to the cerebral cortex. We can often make out the loss of fibers in the deeper parts also, in the white substance, and the central ganglia. The co-existing changes in the spinal cord, first accurately described by Westphal, and since then recognized as almost constant, are of especial interest. They usually consist of fascicular systemic degeneration of the lateral columns (the pyramidal tract), or the posterior columns. A large part of the physical disturbances of general paralytics, such as tabetic symptoms or spastic paralysis (*vide supra*), are certainly due not to the cerebral disease, but to these accompanying changes in the spinal cord.

Accordingly, we believe that, on the basis of our present knowledge, we can best conceive the nature of general paralysis in the following way: By the action of certain injurious influences, which usually seem to stand in some connection with syphilis (see page 961), there is a gradually progressive destruction of nerve-tissue in the most diverse portions of the nervous system. The clinical symptoms must naturally differ according to the significance and function of the affected fibers or cells. As a rule, certain cortical regions of the cerebrum are first diseased. The disturbances of speech are probably dependent upon the loss of fibers in the left island of Reil, the disturbances of intelligence upon the destruction of fibers in the cortex. We may also make out corresponding anatomical changes, either cerebral or spinal, as an explanation of the later motor, tabetic, and other symptoms; but in many cases the order in which the different sections are affected varies considerably. We have seen in a previous section (see page 976) that the whole process may begin with a spinal disease, especially tabes dorsalis, to which the paralysis is " added " later; but we must understand that the two conditions are wholly analogous to and co-ordinate with each other. Both are parts of the same degenerative process, which can accomplish its work of destruction in the most diverse regions of the nervous system.

No coarse anatomical lesions can be made out, as a rule, to explain the paralytic attacks, but it is very probable that they depend, at least in great part, upon the changes in the motor central convolutions.

Diagnosis.—Since the diagnosis of beginning general paralysis is of the greatest practical importance, we will once more mention briefly all those symptoms which are especially to be considered in diagnosis: Striking alteration in the nature and the behavior, rapid and motiveless change in the disposition, disturbances of memory, loss of intelligence (failures in reckoning, etc.), the characteristic changes in the speech and handwriting, and finally the co-existing somatic symptoms: inequality of the pupils, immobility of the pupils, loss, or, more rarely, increase of the tendon reflexes, and mild paralytic attacks, such as vertigo, disturbance of speech, temporary disturbance of motion in one arm, etc.

We would also mention, as especially common and disastrous mistakes, that the symptoms of general paralysis are often misunderstood at first, and are regarded as the signs of immorality, the failure of the sense of duty, etc. It also frequently happens that general paralysis is at first regarded as simple neurasthenia or hypochondriasis, and treated accordingly.

As a rule, general paralysis can be certainly distinguished from other organic nervous diseases by careful attention; but of course we must add that in some cases cerebral tumors, gummatous processes, and especially certain cases of multiple sclerosis, may show a type of disease very like general paralysis. We should also remember that gummatous syphilis may be combined with genuine paralytic symptoms (*vide* page 1128).

Prognosis.—The prognosis of general paralysis, like that of all chronic degenerative conditions of the central nervous system, is wholly unfavorable. At present we know of only a small and decreasing number of (doubtful) recoveries, but there are many cases where there is a temporary improvement in the condition, a "remission," sometimes of a considerable degree and lasting a long time. The earlier the patient comes under proper care and treatment the sooner may we hope for such a favorable turn. Of course, as we have said, relapses of the disease almost always come on later. Those cases especially are to be regarded as unfavorable in which frequent paralytic attacks come on early, in which other physical symptoms, especially of a spinal nature, soon set in, and in which the whole nutrition of the body rapidly suffers.

Treatment.—As soon as the disease is recognized, the first and imperatively necessary injunction must be to remove the patient from all physical and intellectual exertion as well as from all mental excitement. The patient must therefore, if possible, withdraw from business, which up to that time he may have tried to carry on. His methods of life and his diet must be regulated, and every excess must be forbidden. For the cases which even at first are associated with states of great mental excitement, the commitment to a proper asylum is often most urgently to be recommended, while for cases that during their course show simple mental weakness, care at home is often sufficient. Only by a timely recognition of the disease and by adopting the appropriate measures can the patient's relatives be spared the many disagreeable experiences which would otherwise undoubtedly ensue.

In regard to the treatment of the disease itself, we should advise inunction with mercurial ointment, especially if we can discover a previous syphilitic infection. As a rule, we ought not to expect much success from this any more than in tabes (see page 978), but we may perhaps check the advance of the disease. We should therefore try the antisyphilitic treatment chiefly in the initial stages of the disease, and also in the cases where the suspicion that there is also gummatous syphilis is justified. We may combine the internal use of iodide of potassium with the inunction.

Furthermore, we should try tepid baths, with cool sponging, and also a cautious application of electricity (galvanization of the head and spinal cord), and prescribe internal remedies, especially ergotine. We need not go more fully here into the numerous symptomatic details.

CHAPTER X

CHRONIC HYDROCEPHALUS

Ætiology and Pathology.—Repeated mention has been made in preceding chapters of the occurrence of dropsy in the ventricles as a sequel to other cerebral diseases, such as meningitis and tumors. Besides this "secondary hydrocephalus," a collection of fluid in the ventricles may be a symptom of an apparently idiopathic primary disease. This is observed most of all in the newborn, or at least in young children.

Little is known with certainty about the causes of chronic hydrocephalus. The assumption is very frequently made that the condition is the result of an inflammation of the ependyma of the ventricle, which itself occurs either before birth or very soon after, but the autopsy often fails to support this idea. Our knowledge is equally uncertain as to the existence of a stasis due to mechanical obstruction (obliteration of the foramen of Magendie, etc.). Syphilis, drunkenness in the progenitors, etc., have been regarded as predisposing causes; whether justly or not, is uncertain. It has been repeatedly observed that the disease has attacked several children of a single family.

The most important physical sign of hydrocephalus in children is enlargement of the head. The circumference of the skull may even in the first year of life be sixty to eighty centimetres. Usually the frontal bones and the parietal eminences are especially prominent. The cranium becomes gradually almost as thin and translucent as paper. The fontanelles and sutures gape widely. The brain is flattened out, so as to seem almost like a bag, filled with the hydrocephalic fluid. In well-marked cases the entire thickness of the hemispheres is frequently not more than an inch. The space within, containing the serous effusion, represents the enormously distended ventricles, particularly the lateral ventricles, although the third and fourth ventricles are quite often distended also. The walls of the ventricles are often strewn with minute granulations, or they present a reticular hypertrophy. The hydrocephalic fluid usually has the appearance of colorless serum, and contains a very slight amount of albumen, if any. The specific gravity is about 1004 to 1006. The amount of fluid may be a quart or more; but, of course, there is a great variation in this respect in different cases.

Congenital hydrocephalus is often associated with other peculiarities or defects in the structure of the brain, which we can not mention here.

Clinical History.—Sometimes a child is born with hydrocephalus so far developed as to occasion dystocia. Usually, however, the parents notice nothing peculiar about the child for some weeks. Then they are alarmed by the gradual swelling of the head. As a basis for determining abnormal size, we may mention that under normal conditions the circumference of the head at birth is about thirty-five to forty centimetres, at the end of a year about forty-five centimetres, and from that age to puberty there is a gradual approach to a circumference of about fifty centimetres. The possible dimensions in chronic hydrocephalus have been already stated. The increase in circumference is often quite rapid, amounting in a fortnight or three weeks to one or two centimetres. Usually the swelling is tolerably symmetrical; but sometimes the greater increase is in the antero-posterior diameter, making the skull dolichocephalic. At times the rate of expansion may be particularly rapid, and then at other times it may seem to be suspended. That the fontanelles and sutures remain widely open has already been mentioned; sometimes it is even possible to get fluctuation through them. An intravascular murmur can now and then be heard in the head, but it has no great importance with regard to diagnosis. The veins are often so greatly distended as to form a bluish network underneath the scalp. The face remains small, in striking contrast with the great, heavy cranial portion of the head. The head almost always hangs over forward, from its own weight. The eyes generally look down, partly because the roof of the orbit is depressed, and partly because of impairment of the nervous supply to the motores oculi.

A very important symptom is the defective intellectual development of hydrocephalic children. They can not learn to talk well, if at all. If they play, it is in a silly manner. They can not concentrate their attention upon anything, and they are heedless and dirty. It must, however, be mentioned that sometimes, in spite of considerable hydrocephalus, the patient now and then evinces an unexpected activity of mind—thus, he gradually becomes able to distinguish the different objects and individuals about him.

There is almost invariably motor disturbance also. The legs, more rarely the arms, are decidedly paretic, or there may be even complete paraplegia. There are usually spastic symptoms and increased tendon reflexes. Few patients learn to walk or stand alone. The arms seldom present any great paresis, but their movements often betray an awkwardness and uncertainty suggestive of ataxia. It is noteworthy that sensation almost always remains intact; at least the patient reacts vigorously to the prick of a pin, etc. Of the special senses, sight is most frequently affected; choked disk and atrophy of the optic nerve have been observed repeatedly. Symptoms of motor irritation are of very frequent occurrence, such as general convulsions and spasm of the glottis. General nutrition is pretty well maintained in many cases, but, as a rule, hydrocephalic children are atrophic and ill developed.

The chronic hydrocephalus of children almost always terminates unfavorably. Only a few patients survive the fifth year, although now and then striking exceptions occur. Death is generally the result of marasmus; or a convulsive seizure may prove fatal. The possibility of recovery has not yet been demonstrated. The progress of the disease may, however, be arrested, and the child continue for years *in statu quo*.

Hydrocephalus in adults is a very rare, chronic, and apparently idiopathic disease. Its cause again is assumed to be chronic inflammation of the ventricular ependyma (so-called *meningitis serosa ventriculorum*). The symptoms are sometimes very like those of a tumor of the brain (with predominant general symptoms), and sometimes there is a remarkable absence of characteristic cerebral disturbance, except that spastic paralysis of the extremities (compare page 1012) is gradually developed.

Diagnosis.—A pronounced case of congenital hydrocephalus can be recognized without difficulty, inasmuch as the excessive size of the head betrays the disease upon the first glance. Less extreme cases may indeed be somewhat obscure, and we have especially to avoid confounding the condition in question with rachitic enlargement of the skull. We should always, therefore, take into consideration the intellectual powers, the presence or absence of motor disturbances, and other similar symptoms, as well as the cranial peculiarities. In the hydrocephalus of adults there is often no enlargement whatever, so that a diagnosis can hardly ever be made positively.

Treatment.—Thus far, no remedy has been applied with success in chronic hydrocephalus. The following may be tried: Applications of mercurial ointment and of tincture of iodine to the scalp, methodical compression of the skull, and iodide of potassium internally. The hydrocephalic fluid has often been drawn off, to a certain extent, by tapping, but only rarely with permanent success, and usually with merely temporary benefit, if any. Quincke was the first to suggest drawing off the hydrocephalic fluid by so-called lumbar puncture; that is, puncture of the vertebral canal. This can be done most easily in the region of the second, third, or fourth lumbar vertebra. Some favorable results from this method have been reported.

In most cases we confine ourselves to purely symptomatic treatment, and to recommending intelligent care for the child.

CHAPTER XI

MÉNIÈRE'S DISEASE

(Vertigo ab aure læsa. Labyrinthine Vertigo)

In 1861, Ménière, a French physician, first called attention to a peculiar affection which may be apparently primary or may result from chronic aural disease and which is characterized mainly by excessive vertigo and loud tinnitus aurium. At first the symptoms appear in distinct paroxysms. These are ushered in by a shrill ringing in the ears, which is often compared to the whistling of a locomotive, and which is perceived in but one ear. At the same time, or shortly after, comes on a very pronounced dizziness, of a unique sort. The patient has a feeling as if his whole body were moving, as if he were falling forward or were whirling around. Consciousness is unimpaired, but the patient feels wretched, the skin is pale and cool, and the face is bathed in cold perspiration. Frequently there is vomiting toward the close of the attack. The first paroxysms are of brief duration. As the disease progresses the attacks become more and more frequent, and at last the vertigo may be constant, being extremely annoying to the patient and perhaps confining him to bed. Even now there are occasional paroxysmal exacerbations of the disorder, usually ushered in by the shrill tinnitus. The tokens of chronic aural disease on one side, or less frequently on both sides, also persist. Sometimes there is purulent otorrhœa; often the aural speculum reveals lesions of the drum or of the middle ear; and almost invariably there is more or less deafness on the affected side. This condition may persist for years, until finally it ceases of its own accord, after the deafness on that side becomes complete.

The apoplectiform occurrence of Ménière's symptom-complex in persons whose ears were previously healthy is especially characteristic and interesting, but very rare. The disease begins suddenly, or at least very acutely, with an attack of vertigo or with loss of consciousness. In a very short time total deafness sets in, with extreme tinnitus, vertigo, and, as we have seen in one case, a very unsteady gait (as in cerebellar ataxia). In such cases we may suspect an effusion of blood into the labyrinth; but there may also be, perhaps, an acute inflammatory process. Later the symptoms may gradually become less severe, but there is hardly ever complete recovery.

We possess scanty information as to the origin of these subjective phenomena. That they are due to a disease of the internal ear (labyrinth) can scarcely be doubted; and it is further probable that in every case the vestibular nerve and the semicircular canals are involved. Numerous experimental investigations have demonstrated that these last-named structures bear a part in maintaining the equilibrium of the body. Acquaintance with this variety of vertigo is valuable to a nervous specialist, inasmuch as Ménière's disease has been more than once confounded with epilepsy or disease of the cerebellum and other parts of the brain. On the other hand, of course, we must also add that purely neurasthenic and hysterical conditions may sometimes simulate the type of Ménière's disease very perfectly, so that a correct diagnosis often can not be made until the patient has been carefully observed for a long time.

Treatment is not wholly unavailing. Charcot has discovered that the persistent use of quinine gives great relief—at least in many cases—and it may even completely cure the disease. Eight to fifteen grains (gramme 0.5–1) of quinine should be given daily, in two or three doses, and continued for at least several weeks. Persevering galvanic treatment (anode to the ear, cathode to the back of the neck) also seems to us to be at times of decided service. The particulars of such special treatment of the ear as may be necessary must be sought elsewhere.

VI.—NEUROSES WITHOUT KNOWN ANATOMICAL BASIS

CHAPTER I

EPILEPSY

(*Falling Sickness. Morbus sacer*)

Ætiology.—Epilepsy is a peculiar disease of rather frequent occurrence, the main symptom of which is paroxysmal loss of consciousness. In typical cases the unconsciousness is associated with violent general convulsions; but there are many anomalous and rudimentary forms of epilepsy without any symptoms of motor irritation. "Genuine epilepsy" is a functional neurosis—that is, with our present means of investigation we can discover no constant objective lesion of the nervous system as its basis. It is, indeed, true that attacks similar to those of true epilepsy not infrequently occur in the course of tumor, syphilis, and other diseases which do present an anatomical lesion; but such attacks are merely symptomatic, and are therefore termed "epileptiform," in distinction from the genuine epileptic paroxysms.

Of the actual causes of epilepsy we know nothing. We are acquainted only with certain factors which are favorable to the development of the disease, and are to be regarded, therefore, as predisposing or exciting causes. Heredity is decidedly the most important of these. About one third of all cases of epilepsy occur in persons who have inherited a nervous diathesis, and one or more of whose blood-relations have suffered from diseases of the nervous system. It should not be understood that we must find other cases of genuine epilepsy in the family, in order to establish the fact of congenital predisposition. The question is merely whether the ancestors have exhibited a general tendency to nervous disease. The more accurate and careful our investigations, the oftener do we find among the relatives of the patient instances of nervous trouble—sometimes genuine epilepsy, sometimes insanity, hysteria, or general "nervousness." Of course these "nervous families" present, besides those that are actually ill, others who are more or less peculiar and odd, and yet others gifted with extraordinary talents, although frequently somewhat ill-balanced. It is said that the children of parents who are related to each other are somewhat predisposed to epilepsy, as well as to other nervous diseases; but certainly this factor is very rarely of importance. Perhaps drunkenness in the parents is somewhat more prejudicial in this regard. It is said to have been repeatedly observed that children begotten while the father was intoxicated became epileptic.

There are other influences which are assumed to have ætiological importance, but whether justly or not is difficult to decide. Alcoholic excesses can seldom act in this way (although epilepsy is said to attack absinthe-drinkers in France quite frequently). Venereal excesses probably have still less importance. It should also be borne in mind that not infrequently excesses in these directions are the result of neurotic tendencies already existing. Syphilis has no direct connection with genuine epilepsy. Epileptiform convulsions may, as we have seen, be symptomatic of syphilis, being due to the cerebral lesion caused by this latter disease. Certain factors may determine the onset of epilepsy, although they can not be said to cause the disease (except the symptomatic epilepsy which develops after a previous encephalitis). Such are over-exertion of mind or body, repeated emotional disturbance, certain general conditions of the system, such as anæmia or malnutrition, on the one hand, and plethora on the other; and, in particular, acute febrile diseases, such as scarlet fever, measles, and gastric diseases. [The individual attack is often brought on by some disturbance of digestion or indiscretion in diet.—K.] Another important point is that the first attack is sometimes brought on by great mental excitement, especially fright. But here, too, it is probable that the terror is merely the exciting cause, a tendency to the disease already pre-existing. We must also be on our guard against confounding genuine epilepsy with the convulsive form of hysteria (*q. v.*), which very frequently develops after fright.

In some instances there is an evident connection between epilepsy and a previous injury to the skull at birth or from a fall, or blow, etc. At a certain interval after the trauma, attacks begin which seem precisely like those of genuine epilepsy. This is known as "traumatic epilepsy." These are not cases of genuine epilepsy, however, inasmuch as there is really some anatomical lesion of the cortex cerebri which, in some way as yet unknown, causes irritation of the motor centers of the cortex (*vide infra*). It is often the case that this variety of epileptiform attacks is peculiar in that the convulsions are at first unilateral, or confined to a single limb, corresponding to the seat of the injury in the opposite cerebral hemisphere.

"Reflex epilepsy" remains to be mentioned. This name is applied to cases where each convulsive attack seems to be excited by reflex influences originating in some remote part of the body. Most cases have followed injury of peripheral nerve trunks (retained splinters, or scars), and have ceased upon removal of the exciting cause. Other causes are new growths in the nerves, foreign bodies in the ear, otitis, intestinal parasites, and, apparently, diseases of the female sexual organs. It seems probable that sufferers from these attacks have had a tendency to disease of the nervous system. We must hesitate to rank reflex epilepsy in the same class with the genuine form. The whole subject of "reflex epilepsy" needs a new and thorough investigation. It has been in the past so often confounded with hysterical ("traumatic-hysterical") attacks, that we are personally disposed to doubt the occurrence of any real reflex epilepsy.

Both the traumatic and the reflex varieties of epilepsy have repeatedly been the object of experimental investigation. Brown-Séquard has shown, by a great number of experiments, that epilepsy can be excited in rabbits by injuries to the medulla, the spinal cord, and the sciatic, as well as other peripheral nerves. A certain time after the operation the animals undergo spontaneous convulsive paroxysms. These occur at frequent intervals and for a long time, and they may be voluntarily excited at any time by irritation of a certain portion of the skin called the "epileptogenous zone." An interesting observation in this connection has been made by Brown-Séquard, which is that sometimes the progeny of these animals, who have been made epileptic, suffer from spontaneous epilepsy. Westphal induced epilepsy in guinea-pigs by blows upon the skull. Immediately after the blow general convulsions occurred, but soon entirely ceased. Afterward, however, there were repeated epileptiform attacks. Westphal thought that the causative lesion in these instances was the minute hæmorrhages which were found in the upper part of the cervical division of the cord and in the medulla.

These and other experiments, which will be spoken of later, interesting as they may be in themselves, have contributed absolutely nothing to our knowledge of genuine epilepsy. In regard to the latter we have nothing left but to assume that there is a congenital, abnormal predisposition of the brain, and especially of its motor centers, but the nature of this predisposition is still wholly unknown.

[Various writers have maintained that the epileptic attack was due to an accumulation of toxic products in the blood—in many cases an auto-intoxication from intestinal disturbances. An increase in the toxicity of the urine has been noticed before the attack, and the attack may sometimes be provoked by the ingestion of certain toxic substances. The toxic origin of the trouble, however, is not yet established.—K.]

Clinical History.—In describing the symptoms of epilepsy, we shall first consider the various forms of the epileptic paroxysm, and then describe the general course of the disease.

1. The fully developed epileptic paroxysm is usually described, for the sake of greater clearness, as made up of several stages. First is the prodromal stage, or, according to Galen's expression, still in vogue, the stage of the epileptic aura (*aura* = breath). Not infrequently, however, there is no aura whatever, the convulsions coming on without warning; but in many cases the prodromata are well marked, and are repeated with noticeable regularity and similarity before each individual attack, although the different cases of epilepsy differ greatly as to the special phenomena of the aura occurring in each.

The best manner in which to distinguish the various forms of aura is according to the nature of the nervous phenomena, whether sensory, motor, vaso-motor, or psychical. Of these the most frequent is, beyond a doubt, the sensory. Here we have peculiar paræsthesiæ, beginning in the arm or leg, or perhaps in the region of the heart or stomach, and thence usually "mounting to the head." It is seldom that the peculiar sensation is actually like a "breath" or puff of air. The aura which proceeds from the epigastrium is sometimes associated with a very disagreeable feeling of oppression and anxiety, and often also with nausea and vomiting. The aura may be referred to the nerves of special sense. In certain instances the patient perceives an unpleasant odor, which he likens to some familiar one. An aura of taste also occurs, but it is very rare. An optical aura is much more frequent, consisting of a subjective sensation of color or light (usually a sensation of red appears first), of an apparent increase or diminution of the size of surrounding objects, or finally of actual hallucinations of vision, such as beholding all sorts of human or brute shapes. An auditory aura is not very rare: it produces a sudden feeling of deafness in one ear, or various subjective sounds, like whistling, humming, roaring, etc.

The motor aura takes the form of mild premonitory contractions, affecting the head, face, arm, or leg. There may be aphasic disturbance at the same time; or we may observe symptoms of irritation of the unstriped muscles (strangling, or a desire to go to stool). Sometimes there are prodromal vaso-motor phenomena, where the aura consists of a sensation of cold or warmth, often associated with excessive pallor or redness of the face or hands. An attack may be ushered in by chilliness, perspiration, or palpitation.

Finally, the name of psychical aura is applied to those initial symptoms which consist of vertigo, confusion of thought, or other pronounced disorders of consciousness. A particularly frequent form for this to assume is excessive mental uneasiness and excitement. We may also add that various forms of aura are not infrequently seen in combination.

The aura lasts sometimes only a few seconds. It may persist long enough for the patient, who knows from experience what is coming, to lie down or take other precautionary measures (*vide infra*). In some few cases the aura may last hours, and even days. This is especially true of the psychical variety. Sometimes the aura passes away without being succeeded by any true epileptic fit; but it is usually followed by the second stage of the attack—the convulsive stage.

The convulsive stage of the epileptic attack almost invariably begins abruptly. Perhaps there is no aura, or only a very brief one, before the patient falls suddenly to the ground, usually on his face, although sometimes on the side or back. Consciousness is entirely suspended. Insensibility is complete, and often the patient sustains severe injury from his fall. Some patients utter a loud "epileptic cry" at the commencement of the attack, but they are already entirely unconscious.

The convulsive attack begins with a brief period of general tonic spasm of the muscles. The head is usually strongly extended, the teeth are pressed firmly together, the trunk is curved backward in opisthotonos, the extremities are extended, and the fingers are clenched over the flexed thumb. Inasmuch as the respiratory muscles participate in the seizure, breathing stops, and the original pallor of the face soon gives place to deep cyanosis. This general tonic convulsion ordinarily is but brief, perhaps fifteen to thirty seconds. It is followed by the second period of the convulsive stage—that of the clonic convulsions. The facial muscles now exhibit the most violent contortions; the eyeballs roll, or occasionally present a conjugate deviation toward one side; the tongue is thrust out and retracted convulsively; the head beats violently against the floor; and the muscles of the arms, legs, and trunk undergo the severest clonic spasms. The pupils are probably contracted for a short time at first, but during the convulsive stage they are widely dilated and do not react at all. The pulse is somewhat accelerated, but not greatly. The temperature is normal, or elevated a small fraction of a degree. The cutaneous reflexes are still suspended directly after an attack; but the tendon reflexes are generally somewhat exaggerated, although sometimes they also are diminished or absent. Not infrequently an involuntary dejection takes place during the fit, or the bladder is emptied; and in men there may be a seminal emission. During these violent convulsions the body is often severely injured. The tongue is frequently bitten. The face, which is usually pale at the beginning of the attack, becomes extremely cyanotic. The veins in the neck are distended, and the venous stasis is so extreme that sometimes minute hæmorrhages occur into the conjunctiva, the skin of the face, and other parts.

The convulsive stage usually lasts several minutes. Then the contractions cease, often after a deep, long-drawn sigh; and the patient passes into the third stage of post-epileptic coma. He lies unconscious, but his respiration grows quiet, and the cyanosis vanishes. Gradually the coma yields to slumber, which may persist for some hours; but some patients remain only a very brief time in this stage, and recover from their attack with surprising rapidity. It is, however, not infrequently the case that for some days after-pains are felt; there are headache, languor, and exhaustion, and mental despondency and irritability. For some time there may be severe pain in the muscles, particularly those of the trunk. There may be slight paresis of one limb or one side of the body after an attack, but this speedily vanishes again in cases of pure epilepsy. In the first urine passed after the seizure is often found a trace of albumen, and perhaps a few hyaline casts. Not infrequently there is also decided polyuria for some time subsequent to the fit. Almost always after recovery of consciousness there is no recollection of what has happened, or it is very imperfect. On careful investigation we often find that the memory of events immediately preceding the attack (for fifteen minutes to two hours before) has also entirely disappeared. A student, for example, who had an epileptic attack at the author's clinique, later had absolutely no knowledge of how he came to the clinique or of what he had seen or heard there before his attack. In a few cases (especially after traumatic epilepsy) this retrograde amnesia may extend over a much longer period.

2. *The Milder, Rudimentary Forms of Epileptic Seizure. Petit Mal.*—Besides these violent paroxysms just delineated ("*grand mal*"), there are very often witnessed in epilepsy milder attacks of so-called "*petit mal.*" Sometimes there is only a transitory dizziness, or slight faintness, or perhaps a brief loss of consciousness, but without accompanying symptoms of motor irritation. These

milder attacks may or may not be preceded by an aura. Cases have been repeatedly seen where the patient suddenly pauses in the midst of conversation, card-playing, piano-playing, or other occupation, stares absently for a moment, and then, with equal abruptness, goes on with what he was doing, as if nothing had happened. In other instances the patient pursues his occupation during this brief suspension of consciousness. For example, if seized while upon the street, he walks on mechanically, but takes the wrong turning, or goes into a strange house, when suddenly he comes to himself and wonders to find himself where he is. Cases of "sudden somnolence" are also almost all of them ascribable to epilepsy. There are all sorts of transitional forms between the slight attack of vertigo and the typical epileptic fit. Not infrequently the patient falls down unconscious, but he has only a slight twitching of the face or arm, and in a few minutes is entirely himself again. Very often, especially in children, the attack consists of a short, piercing cry, loss of consciousness, a few convulsive movements of the arms—and in a few seconds all is over. Such attacks come on in the daytime and often in the night as well. In other patients the attacks consist of sudden pallor, springing to the feet, rushing to the door, the ejaculation of some short words, etc. In short, it is hardly possible to describe all the different manifestations of the rudimentary and atypical epileptic attacks; but persons who suffer from such slight attacks not infrequently have perfectly typical and fully developed epileptic attacks besides, and this is of especial importance in diagnosis.

[The medico-legal bearings of epilepsy, in its mild as well as in its severe form, are very important, but they can be only alluded to here.]

3. *Epileptoid Conditions* (*Epileptic Equivalents*).—Cases of petit mal are generally rudimentary forms of the typical epileptic paroxysm, and consist merely of a simple impairment of consciousness, possibly associated with slight motor symptoms; but in the epileptoid state the characteristic features of the typical epileptic attack are quite subordinate. The disturbance is paroxysmal, and it can often be shown to be connected with genuine epileptic seizures, else its undoubted relation to epilepsy would never have been recognized. The greatest practical importance attaches to the "psychical equivalents of epilepsy" (Samt). These are attacks of mental disturbance, which either immediately succeed a typical epileptic fit ("post-epileptic insanity") or occur independently. These attacks are manifested by states of complete mental confusion, in which the patient may do the strangest things—may strip himself, steal, jump into the water, or commit incendiarism. Besides these "states of epileptic confusion" there are also attacks of violent psychical excitement, associated with terror, frightful hallucinations, and resultant maniacal excitement. Not infrequently the patient is led to acts of violence against those about him. In the young the attack may take a peculiar form: the child runs about in a peevish way, collects all sorts of things together, makes strange motions, etc. We have also frequently seen in children brief states of disturbed consciousness of sudden onset, with dominant ideas of apparent anxiety, often associated with some twitching. Many cases of so-called *pavor nocturnus* are certainly epilepsy, although it is sometimes not easy to exclude hysteria. Numerous and valuable particulars upon this subject, and a consideration of its great medico-legal importance, must be sought in text-books on insanity, as we can not discuss them further here.

Among the rarer forms of epileptoid attack are the epileptoid sweatings (Emminghaus), a spontaneous outbreak of excessive perspiration in epileptics, which may or may not be associated with impairment of consciousness, and also attacks of epileptic tremor (Féré), procursive epilepsy (attacks when the patient runs impulsively forward), etc.

General Course of the Disease.—In a large majority of cases epilepsy begins before the thirtieth year. Often the disease appears in early youth, and sometimes even in the earliest years. Many a child has "convulsions from teething," which later on are seen to have been epileptic. It is only in rare instances that the first appearance of trouble occurs in advanced life.

It is impossible to give any general rule as to the frequency of the paroxysms. Different cases differ very much. There are persons who during their whole life have no more than three or four seizures, at intervals of ten or fifteen years, while in most cases there is an attack every two to eight weeks. In severe types the fits may even recur daily. One very often sees certain variations in the course of the disease; at some periods the intervals between the attacks will be longer, and at others shorter. In severe cases the patient may have for several days very frequent seizures, so that he does not regain consciousness at all between them. This is termed the epileptic state (*état de mal, status epilepticus*). The condition is quite rare. It is often fatal, death being ushered in by a great rise of temperature.

External influences sometimes affect the frequency of epileptic attacks. Alcoholic or sexual excess, [indiscretions of diet,] mental excitement, and physical over-exertion almost always exert a malign influence. An opposite effect is often experienced where a quiet life is led, with every attention paid to hygiene and pure air. In women, the appearance of the catamenia is not infrequently the signal for the occurrence of an attack. In many instances the disease begins with the first establishment of menstruation. Sometimes, however, epileptic girls grow better when they arrive at puberty. Pregnancy sometimes increases and sometimes diminishes the frequency of the paroxysms. Intercurrent diseases seem frequently to exert a beneficial influence upon the frequency of the attacks.

There is a practical distinction between diurnal and nocturnal epilepsy. Many patients have attacks only during the day, while others again have them only at night. A case of pure nocturnal epilepsy may go on for a long time unsuspected, particularly if the patient sleeps alone. He seldom has, on the next morning, the slightest recollection of his attack during the night. He usually perceives, however, from a confused feeling in his head, or from certain injuries, such as a bitten tongue, or from the disordered state of the bed, that something must have happened to him during the night. In some cases of nocturnal epilepsy the patient wakes up out of sleep before he enters into the epileptic state of unconsciousness. Probably he is aroused by the aura. Besides cases where the fits occur during the day or the night only, mixed forms are frequently seen.

With regard to the occurrence of the different varieties of epileptic seizure, all sorts of combinations are possible. Many cases never have any but the typical convulsions; but in many others there are also a greater or less number of attacks of petit mal. The latter may even be for a long period the sole indication of the disease. Often there are no epileptoid conditions whatever, while in other instances the "psychical equivalents" are the most prominent feature of the disorder.

During the interval between the individual attacks many epileptics seem perfectly well, both physically and mentally. They are not infrequently, to be sure, somewhat peculiar and nervously excitable, or again dull and lethargic; but this does not by any means apply to them all. Many epileptics, and particularly such as have comparatively infrequent paroxysms, are very capable; and history furnishes numerous examples of eminent men who suffered from this disease—for instance, Cæsar, Mahomet, Rousseau, and Napoleon I.

Much effort has been devoted to the discovery of "signs of physical degeneracy" in epileptics. Relying upon numerous measurements, Benedikt believes that a majority of epileptics exhibit craniometric anomalies, such as asymmetry of the cranium, macrocephalia, or steepness of the vertex. It is also not unusual to meet with anomalies of the ears, teeth, or hands * in such cases. Indeed, all peculiarities of this sort are, in general, more frequently observed in neuropathic families than in healthy ones.

When the disease has lasted some time, and particularly if the attacks come at very short intervals, the general condition of the patient often undergoes a gradual but marked change. This rule is by no means invariable. The mind becomes more and more affected; the intellect grows feeble; memory grows weaker, and occasionally there is at last dementia. In such cases the body also suffers. There are emaciation, paresis, tremor, and other persistent disturbances of cerebral origin.

In regard to the duration of epilepsy, it must be termed a life-long disease. To be sure, it is no rare thing for the paroxysms to cease and not return for years. But one can never rest satisfied that all trouble is at an end; some cause or other may excite another attack after a long interval. In general, an epileptic has a shorter life-expectancy than healthy persons, especially as he may be carried off by chronic pulmonary or other intercurrent disease.

The prognosis is obvious from what has been already said. The individual seizure is only exceptionally dangerous of itself. The so-called "*status epilepticus*" often ends fatally, as above stated. In general, those cases may be called the most favorable where the separate paroxysms are infrequent and mild; but even here the disease may suddenly assume an aggravated form. With regard to the distinction between nocturnal and diurnal epilepsy, the nocturnal is, in our opinion, the milder of the two.

Pathology.—The very fact that in the intervals between attacks the patient often betrays no sign of disease, shows that epilepsy can not be due to any persistent macroscopic lesion of the tissues. Indeed, in many cases nothing is found at the autopsy, or, at most, changes which can not be regarded as essential, such as osteosclerosis of the cranium or thickening of the cerebral meninges. Epileptic subjects who were during life decidedly demented usually present atrophy of the hemispheres. Meynert states that changes in the pes hippocampi major are noticeably frequent in epilepsy; but these changes are not at all constant, and their significance remains to be established.

We must therefore, for the present, be content to assume that the cause of the epileptic seizure is an intermittent functional condition of irritation. A natural question is, Where shall we locate this irritation, and what may be its nature? The opinion was long current that the medulla oblongata must be regarded as the true seat of the disease. Schröder van der Kolk was the first to express this opinion. It afterward received support from the experimental investigations of Nothnagel, who demonstrated that irritation of a particular spot ("convulsive center") in the pons, in rabbits, invariably excites general convulsions. Nevertheless, most pathologists have now abandoned this view, because experiment and clinical observation indicate with increasing distinctness that the origin of epileptic convulsions is to be sought, at least in the great majority of cases, in the cerebral cortex. The clinical evidence is the invariable combination of convulsions and impairment of consciousness; the circumstance that the milder and the masked forms of epilepsy, now known to be intimately related to the true epileptic convulsions, also, almost without exception, indicate psychical disturbance; that attacks, the symptoms of which are perfectly analogous with those of epilepsy, are often found to be the result of anatomical lesions of the cerebral cortex; and, finally, that these convulsions in man and the convulsions of "cortical epilepsy" (*vide infra*), experimentally produced in animals, extend over the different groups of muscles in a way which corresponds precisely with the anatomical position of the different motor centers in the cortex (Hughlings Jackson). For example, if the convulsion begins in the distribution of the facial nerve, it extends from this point to the arm before it affects the leg.

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* We have seen an epileptic who had six fingers on each hand.

There is also experimental evidence that epileptic paroxysms are of cortical origin. A great number of observers (Hitzig, Ferrier, Albertoni, Luciani, Franck, and Pitres) have proved that electrical irritation of the motor regions of the cortex in animals will produce epileptiform convulsions. Unverricht has made some of the latest and most thorough investigations in regard to this point upon dogs. He found that when a motor center is stimulated the convulsions spread from the corresponding group of muscles to others in a way which corresponds precisely to the anatomical position of the separate centers. If one of the centers in the cortex be destroyed, the convulsions of the corresponding muscles cease at once. This proves that the motor centers must be intact in order to render the occurrence of epileptic seizures possible. Just how the stimulation extends from one center to another we have as yet no certain information. Probably it travels horizontally through the cortex.

We see, therefore, that in all probability the seizures in man also originate in the cortex of the brain. The phenomena of the aura are likewise referable to some stimulation of the cortex, probably of the sensory region in most cases, as in the optical aura. The starting-point of the irritation is probably usually in the motor cortex, but it does not seem impossible that an irritation developing in the posterior cortical regions may extend secondarily to the anterior motor portion of the cortex (Unverricht). The manner in which the irritation is created is as yet entirely conjectural. Kussmaul and Tenner proved that epileptiform convulsions can be excited by a general cerebral anæmia; and this fact was the main foundation for the assumption that the genuine epileptic convulsions are also due to a temporary cerebral anæmia, caused, it may be, by local vaso-motor constriction. Definite proof has not yet been furnished on this point. In the artificial epilepsy which Unverricht produced, and that also which Magnan excited by absinthe, the cortex of the brain was not strikingly anæmic. In regard to the occurrence of the attacks in true genuine epilepsy we can therefore provisionally make the imperfect assumption that, as the result of a diseased condition, the motor cortex of the brain has lost the power to store up its motor energy and to give it out again in normal fashion, and that therefore there arise, from time to time, morbid "discharges of energy" and an outbreak of the convulsive movements already described. We will omit a further discussion of this theory, since we would soon be lost in the realm of mere hypothesis.

Diagnosis.—Most cases of epilepsy are easily recognizable. It needs only to be borne in mind that epileptiform convulsions may also occur as a symptom of cerebral diseases which do have an anatomical basis, such as tumor, abscess, multiple sclerosis, and hydatids. As a rule, however, such diseases are readily distinguished by the state of the patient between the seizures, or by the further course of the illness. It should also be understood that unilateral convulsions, or such as are confined to a single member (Jacksonian epilepsy, *vide supra*, page 1073), are usually not true epilepsy, but symptoms of some circumscribed affection of the cortex. We will return once more to the differentiation from hysterical convulsions in the chapter on hysteria. Weight should be laid on the general character of the attack, the complete loss of consciousness in epilepsy, the dilatation of the pupils, which do not react to light, the initial pallor not infrequently observed, and the subsequent cyanosis of the face. We may also consider the character of the onset and the duration of the attack (in true, genuine epileptic attacks the convulsive stage seldom lasts longer than a few minutes), the biting of the tongue, the condition of the patient immediately after the attack (deep, continued sleep is in favor of epilepsy), the possibility of exciting the hysterical attacks voluntarily (by suggestion, by pressure on a "hysterogenous" zone, etc.), which we can not do with epileptic attacks. The same factors (especially the condition of the pupils and the biting of the tongue) are the chief means of discovering simulated epileptic attacks.

Treatment.—Although no remedy is capable of working a certain and permanent cure of epilepsy, yet a favorable influence can be exerted upon the disease in many ways, so as to lessen the frequency and severity of the paroxysms, and to avert many of their evil results.

In the first place, regimen is of great importance. Any excessive exertion of mind or body must be forbidden. Temperance must be exercised in eating and drinking. Alcohol, strong coffee, and tea, can be used only moderately; nor is too much smoking permissible. The diet should be simple and unirritating, and vegetable rather than animal. It is said that in some cases decided improvement has been brought about by confining the patient to milk and vegetables. In summer the patient should live quietly in the country or the mountains. We have also the individual constitution to consider: a weak and anæmic person must have iron and abundant nourishment; and a full-blooded, corpulent individual should drink the natural aperient waters, and live abstemiously.

Proceeding to the treatment of the disease itself, we shall only rarely, when dealing with "reflex" epilepsy (*vide supra*), have to consider ætiological conditions. The excision of old scars, the extraction of foreign bodies, or trephining the skull, where the disease has followed an injury, are said to have brought about permanent recovery in a few cases. We should also deal with any nasal or aural disease, in order, by rational treatment of any such trouble, to exert, if possible, a favorable influence upon the convulsive attacks. If we discover any intestinal worms they should be expelled. In genuine typical epilepsy we have no such indications to fulfill, and we must have recourse to such treatment as experience shows can influence favorably the outward manifestations of the disease.

Among these remedies, potassic bromide has an undisputed right to the first place. It should be the first remedy tried in any severe case. Apparently it acts by directly lowering the sensitiveness of the motor centers of the cortex to irritation. Rather large doses are requisite. Beginning with about one drachm (grammes 4–5) a day, we may find it advisable to increase up to two or two and one half drachms (grammes 8–10). It may be prescribed in water (1 to 10 or 15), or in powders which the patient himself is to dissolve in water, sweetened if desired. The remedy in almost all cases needs to be used for months and years, and it is therefore often advisable for the patient to buy a half pound or a pound at a time and weigh it out himself into the proper doses. It should always be taken in a good deal of water, half a tumbler or more, to avoid irritating the stomach. The total amount for the day is usually divided into two or three portions; but the whole may be dissolved in a large amount of water (seltzer water) and drunk gradually through the day. The other bromide salts, the bromides of sodium and ammonium are also frequently employed. They have the advantage of disturbing the stomach less than does the potassium salt. It is well to combine the various bromides. Erlenmeyer strongly recommends a mixture of two parts each of potassic and sodic bromides with one of ammonic bromide in so-called "bromine" water.

In using the bromides, persistence is necessary for at least months, and often, with occasional interruptions, for years, if benefit is to be obtained. In case there are unpleasant symptoms due to the remedy, such as excessive acne, a bad breath, muscular lassitude and tremor, cardiac weakness, dyspepsia, impotence, or mental depression and weakness of memory, we must diminish the dose, or even omit the medicine for a time. Many patients are greatly annoyed by pustules due to the bromide; this can sometimes be avoided by giving Fowler's solution at the same time. If the attacks are decidedly abated, the dose may be gradually diminished, to be increased again, however, if there be any tendency to a relapse.

There is no doubt that the frequency of the attacks can be lessened by the continued use of bromides. This action is of course manifest only in those cases where the individual attacks occur rather frequently (every three or four weeks or oftener). If we are treating epileptics who have intervals of six months to a year or more between the individual attacks, it is hardly possible to decide with certainty whether the bromide treatment has any efficacy, and the long-continued use of bromide which is necessary is often attended by unpleasant accompaniments. We are therefore usually disposed in such cases to abstain wholly from the bromide treatment. Flechsig recommends combining the bromide treatment with the use of opium. We should give at first simply the extract of opium, beginning with two and a half grains (gramme 0.15) a day, and gradually increasing to six or eight grains (gramme 0.25–0.35) or more, always divided into two or three doses in the day. After using opium for six weeks we stop it abruptly and begin with a bromide treatment, continuing about two months. Whether this method has any special advantages can be told only after longer experience.

Recourse to other remedies is seldom had, unless potassic bromide fails, or for some cause must be discontinued. We may then try valerian in powder, eight to thirty grains (grammes 0.5–2) several times a day, or as an infusion. It is a very good plan to give patients who are taking bromide a cup or two of valerian tea at bedtime. Belladonna may also be exhibited, or a pill of atropine, grain $\frac{1}{125}$ (gramme 0.0005), three to five times a day; or zinc oxide in the dose of one to three grains (gramme 0.05–0.20), perhaps combined with one fourth of a grain of extract of belladonna [U. S. P.], and fifteen grains of valerian, as a powder, three times a day, and finally chloral in small doses (five to ten grains a day, grammes 0.3–0.5, divided into several doses). Combinations of the bromide salts with some one of the remedies mentioned have seemed to us very advantageous in many cases. We have sometimes seen a decidedly favorable action from a mixture of the bromides with small doses of chloral or with atropine, especially where there are frequent rudimentary attacks. Bechterew recommends the combination of bromides with adonis vernalis (an infusion of two or three parts of adonis vernalis in 180 parts of water, to which eight to twelve parts of bromide are added). Many remedies formerly recommended are not worth mentioning, as they are wholly inefficacious.

Electricity is apparently beneficial in occasional instances, and it may be tried in connection with other remedies. The galvanic current should be cautiously applied to the head and the sympathetic nerves. Still greater benefit is sometimes obtained from a carefully conducted cold-water cure. Cold sponging with friction at night helps most cases, and it is sometimes very advisable to send the patient in summer to some appropriate establishment for cold-water treatment. We can not hope for any benefit from the surgical treatment of epilepsy except in those cases where the epilepsy is symptomatic, and we can expect, by the operation, to remove or render harmless some actual local lesion in the brain (a depression of the skull, a cicatrix, a cyst, a meningeal adhesion, etc.). [In some cases of Jacksonian epilepsy an attempt has been made to relieve the attacks by removing the cortical center in which they apparently originate. After the brain is exposed this center is determined by faradization of the cortex. In a number of cases the center has been excised even when there was no macroscopic evidence of disease. The convulsions have often ceased for a time, but they have usually returned later, and the removal of the cortex is apt to cause permanent paralysis.—K.]

Treatment during the Paroxysm.—In most cases we can do little during the seizure except to take such precautionary measures as common sense suggests. We possess no means of suppressing an attack when once under way; nor, indeed, is it often dangerous. In individual instances the patient finds out from experience some method to cut short the paroxysm during the aura. For instance, there are cases where tightly bandaging or vigorously rubbing the part in which the aura originates will avert the convulsions. A number of cases have also been known where the ingestion of a generous quantity of common salt during the aura has had the same effect. In these cases the aura usually starts from the epigastrium. A patient of our own, whose attacks began with a feeling of rectal tenesmus, maintained that she could almost invariably suppress the convulsions by promptly going to stool, if she had time and opportunity. It was formerly a frequent manœuvre to seek to ward off the attack by compressing the carotids; but this usually fails. Berger recommends the inhalation of nitrate of amyl at the commencement of the fit, having repeatedly seen benefit follow its use.

In the "status epilepticus," narcotics are the most deserving of trial, and in particular chloroform or ether given by inhalation, and the internal use of chloral (forty-five to seventy-five grains a day, grammes 3–5). Amyl nitrite may also be of service.

[It is sometimes desirable to withhold the knowledge that he is an epileptic from the patient, whose ordinary life should be interfered with as little as is possible.

Especially in cases characterized by headache and heat in the head, Brown-Séquard finds the application of ice directly to the back of the neck and between the shoulders useful.]

APPENDIX

INFANTILE CONVULSIONS (ECLAMPSIA INFANTUM)

Convulsions in childhood are of such frequency and importance as to justify brief special mention here.

Every practitioner learns from daily experience that the young are especially predisposed to convulsions. Probably this is due in part to excessive reflex excitability of the brain in childhood. Thus children not infrequently undergo convulsions under circumstances in which adults would very rarely have them. They sometimes are seen in children in the beginning of acute febrile diseases, such as pneumonia, scarlet fever, measles, febrile sore throat, etc. They also occur from indigestion, particularly when the stomach has been overloaded; sometimes on account of teething; or because of intestinal worms. In these cases they are usually regarded as of reflex origin, although they may possibly be due to the action of toxines (especially with intestinal worms).

Convulsions may occur in very early life without ascertainable cause. In many cases they are really the commencement of epilepsy, as is seen afterward. Again, they may be due to some actual lesion in the brain. For example, if one recalls the initial stage of the acute poliomyelitis and acute encephalitis of children (*vide* pages 1015 and 1111), it will not seem unlikely that many rapidly fatal cases of "convulsions" are really instances of the diseases mentioned. This point has not yet been at all satisfactorily investigated by pathologists. At any rate, it does not seem satisfactory to us to regard the "œdema of the meninges" found in such cases as an independent disease and the sufficient cause of death. Often convulsions occur suddenly in children and then cease, never to recur, without our being able to find any explanation of the attack. Experience shows that rachitic children are especially liable to suffer from eclampsia—possibly because of cranial rachitic changes (?).

The symptoms of the eclamptic attacks are on the whole analogous with those of epileptic paroxysms. The child's eyes become staring and fixed, and there are tonic and clonic spasms of the face, trunk, and extremities. Such seizures may continue for days with brief intermissions. In such cases the prognosis is dubious, particularly if the child be weakly, but it is by no means absolutely bad. The cause and the significance of the convulsions can seldom be determined immediately.

Symptomatic treatment consists of applying cold to the head, wet packs, sinapisms on the chest and the calves of the legs, and perhaps an enema (to which vinegar may be added. These measures generally answer for mild cases. If the fits are very frequent and violent, we may allow even small children to inhale chloroform, often with great advantage. A dessertspoonful is to be poured upon a handkerchief and administered cautiously.

Of course, we must also try to discover and remove the cause (acute diseases, intestinal worms). The attacks due to overloading the stomach usually occur in not very young children, and are apt to be greatly benefited by a prompt emetic or purge.

[A bath at a temperature of 90°–95°, while cool water is applied to the head, seems often to be of service; if the child be exhausted by diarrhœa, the cold to the head should be omitted. In a teething child it can do no harm, and it sometimes has a very marked beneficial effect to lance the gums thoroughly. Enemas containing chloral with or without bromide of potassium are more used in this country than is chloroform by inhalation.

A drop of nitrite of amyl by inhalation is said by Eustace Smith to exert a controlling effect on the muscular movements.]

CHAPTER II

CHOREA

(*Chorea Minor. Sydenham's Chorea. St. Vitus's Dance*)

Ætiology.—Centuries ago the name *chorea* (dance) was applied mainly to those strange states of "dancing mania" which were endemic in certain places, being due to excessive mental excitement and to the innate propensity to imitation. The specific for this condition was held to be a pilgrimage to some shrine of St. Vitus. At the present time, however, chorea is used to designate a perfectly definite disease, of which the characteristic symptom is the appearance of certain peculiar motor phenomena due to irritation of the nervous centers. It is sometimes called *chorea minor*, in contradistinction from what was formerly termed *chorea major* or *magna*. This latter is, however, not a genuine, independent disease, but a manifestation of hysteria (*q. v.*), or apparently in many instances of epilepsy.

Chorea proper is mainly a disease of childhood. It occurs most often between the fifth and fifteenth years, although it may be seen both earlier and later. There is a slightly greater liability to it in girls than in boys. Hereditary neuropathic tendencies are also a factor in its ætiology, but not a very important one. (See the appendix to this chapter for chronic hereditary chorea.)

As to causation, in many cases nothing definite can be made out. Mental excitement, as from fright, seems in some few instances to favor the onset of the trouble. It is also certain that the imitative impulse will often lead to choreic movements in healthy children who come in contact with choreic patients, but it is doubtful whether this "imitative chorea" can be regarded as true chorea. It seems to us far more correct in such cases, which are very amenable to psychical treatment, to speak of a hysterical chorea. There is a very interesting connection between chorea and acute articular rheumatism. Although the statement of certain authors, that almost every case of acute articular rheumatism in children is followed by chorea, is far too strong, yet this sequence is comparatively frequent. Chorea is sometimes seen also in children who have a mild form of chronic rheumatism, or especially in such as have valvular cardiac disease, whether preceded by articular rheumatism or not. This undoubted and close connection existing between chorea, articular rheumatism, and valvular heart disease, indicates the possibility that in true chorea there is an intoxication arising from some infection.

Pregnancy exerts a special influence upon the development of chorea in women. *Chorea gravidarum* is most frequent in comparatively youthful primiparæ. The nature of the connection between chorea and pregnancy is still utterly obscure.

Clinical History.—Chorea usually begins gradually, and without any special prodromata. Sometimes, however, there are prodromal symptoms, chiefly a certain mental depression and irritability, with indisposition to intellectual effort. There may be slight rheumatic pains or anorexia, and other evidences of constitutional disturbance.

Ordinarily the peculiar motor disturbances are the first thing to attract the attention of the patient or its parents. There are involuntary and irrepressible movements in the most diverse groups of muscles. Both single contractions and also complicated movements occur, independently of the will, and in all parts of the body, now in one place, now in another, sometimes in a single member, and sometimes in several at once. The movements may be made in rapid succession, or may be separated by long intervals of quiet. The facial muscles may be involved, causing an occasional wrinkling of the brow or distortion of the mouth. When there is marked chorea of the facial muscles, as usually happens in severe cases, the patients often make the strangest grimaces, sometimes with some particular expression of emotion (laughter, anger, etc.). The eyes also may take part in the involuntary movements, being moved from side to side, or opened and closed. The pupils are frequently dilated. If the patient be asked to protrude his tongue and keep it quiet, it will often be involuntarily withdrawn into the mouth or thrust to one side. The tongue may even be sufficiently affected to impair speech, which is explosive and indistinct. The laryngeal muscles have also been observed to make choreic movements. The arms are frequently the most affected of any part; they are twisted, flexed, elevated, put behind the back—in short, moved in every conceivable way. The trunk is generally but little disturbed in the milder cases, but in severe ones the whole body participates. The patient stands up, lies down again, turns upon his side, etc. The legs are seldom as much disturbed as the arms and face, but slight movements of the lower limbs are very frequent—the foot is thrust forward or extended, the knee is flexed, and so on. In general, it may be said to be characteristic of chorea that the abnormal motor irritation usually affects a considerable number of muscles simultaneously, thus exciting all sorts of combined movements; and, secondly, that choreic movements, for the most part, are not short twitches, but take place in a manner decidedly similar to that of voluntary movements.

The vigor of the movements varies greatly in different cases. At first they may be too slight for the unpracticed eye to catch. Many children in an incipient stage of chorea are unjustly punished at school because they write ill or do not sit quietly. Many cases are mild throughout, never having very severe disturbance. Others, though considerably annoyed, can nevertheless walk or stand alone. In the severest cases, however, the whole body is continually in active motion. The patient throws himself about in bed, and all the extremities exhibit constant violent and irregular movements. The ingestion of food is extremely difficult, sleep is disturbed, and the patient's flesh and strength are rapidly and greatly diminished.

Further, each individual case presents variations in severity at different times. If the patient be left quietly to himself, the contractions are comparatively very slight. As soon as he is conscious of being watched or as soon as any one speaks to him, his condition usually becomes much worse. On the other hand, of course, the movements may also be somewhat influenced by the will. The patient sometimes becomes quieter if the body is completely supported and gently restrained, etc. During sleep the choreic movements usually cease altogether.

In many cases all the voluntary muscles are involved; but sometimes the disease is limited to certain groups of muscles. Very frequently the disturbance is mainly unilateral (hemichorea); the opposite side of the body then exhibits few involuntary movements, or, it may be, none. As already stated, the face and upper extremities are often more affected than the trunk and lower limbs.

These motor disturbances often constitute the sole or the predominant symptom of chorea. There is hardly ever muscular weakness or paralysis. It is remarkable how little feeling of fatigue there usually is, despite the incessant activity. In a few cases only, of genuine chorea, have we seen actual paresis, affecting, for instance, one arm, or in hemichorea the choreic half of the body. Sensation is unimpaired. The reflexes do not exhibit striking peculiarities. The tendon reflexes we have usually found normal, more rarely very weak or even a little increased. There may be isolated spots in the spinal column tender on pressure, but this is not at all constant. That chorea may be complicated by arthritis and valvular cardiac disease has already been stated. Some caution should be exercised in making a diagnosis of cardiac lesion here, for experience shows that choreic patients are apt to have functional murmurs and slight irregular cardiac action. [Osler has found that the endocarditis of chorea is very apt to lead to organic valvular disease. Of 110 choreic patients that he examined more than two years after the attack, 54 presented signs of organic heart disease.—K.] The temperature is not elevated, in spite of the constant muscular contractions, nor is the amount of urea excreted by the kidneys increased.

Slight mental disturbance is frequently observed. The patient is often rude, peevish, capricious, incapable of mental exertion, irritable, and inclined to tears. More marked mental disturbances (confusion, hallucinations) are sometimes met with in the severer cases, especially in chorea gravidarum. [These mental disturbances sometimes become very severe (*chorea insaniens*). The patient becomes excited and delirious, and is very apt to die in a few weeks of general exhaustion, with considerable fever, 104–105° F.—K.]

The entire process generally occupies several months. In mild cases, however, recovery may ensue at the end of a few weeks, while, on the other hand, cases may last a year or even longer. Variations in the intensity of the chorea are often witnessed. These are sometimes spontaneous, and sometimes are due to outward influences. Even when the disease is apparently extinguished, we must be prepared for a possible relapse. The disease may appear repeatedly in the course of a few years, in such a way that it is not easy to determine whether the different attacks are relapses or new illnesses. The protracted cases are, as a rule, comparatively mild; and many cases that begin with great violence end comparatively early. In adults, and still more frequently in the aged (chorea senilis), however, the chorea sometimes assumes a chronic form and becomes stationary.

The termination of chorea, in the great majority of cases, is favorable. Now and then severe cases do occur which end in death. In these the choreic movements are extremely violent. The patient is tossed about in his bed, and can eat little and sleep none. We have ourselves observed three such cases, in girls fourteen to seventeen years of age, which proved fatal within the first two or three weeks. Two died from general exhaustion and collapse, and the third from gangrene affecting numerous cutaneous traumatic lesions, which had occurred despite every possible precaution.

Nature of the Disease.—All cases of genuine chorea thus far examined by pathologists have failed to furnish any lesions which can be regarded as essential. In the three cases above mentioned the autopsy revealed absolutely nothing abnormal in the central nervous system. We are at present, therefore, obliged to classify chorea as a " neurosis "—that is, as a disease that produces functional disturbances, for which latter there is no anatomical basis known to us. The symptoms themselves show that the disorder must affect principally some motor region of the nervous system; but just which motor region is involved can at present only be conjectured. It seems very probable, however, that the true seat of chorea is to be sought in the brain. In the first place, the frequent occurrence of hemichorea would indicate this; as would also the fact that slight mental anomalies are frequently combined with chorea; and, finally, " choreiform " movements may occur as the sole symptom of undoubted cerebral disease, as in post-hemiplegic hemichorea. We have, however, no hint as to whether the motor regions involved are those located in the cortex, or others. The surmise has quite often been expressed that chorea is due to embolism of a mild type; but, in our opinion, this view entirely lacks proof, and is even extremely improbable. As has been said above, we believe that the most probable theory is that genuine chorea is of toxic and usually post-infectious origin.

Diagnosis.—The diagnosis is almost always easy, and it can often be made at a glance. The motor symptoms of athetosis, paralysis agitans, and of alcoholic, senile, saturnine, and mercurial tremor are so characteristic as to be readily distinguished from the movements of chorea. It is not difficult to perceive the difference between genuine idiopathic chorea and the symptomatic choreiform movements occasioned by some other cerebral lesion.

Prognosis.—As has been stated, the prognosis of ordinary chorea is almost invariably favorable, although the disease may prove very tedious. The possibility of relapses should be borne in mind. The prognosis is doubtful, only in the worst cases of acute chorea, where there is great and rapid failure of the general health. Chorea gravidarum must also be regarded as of far more serious prognosis than the ordinary chorea of childhood.

Treatment.—Even in mild cases the patient must be kept from school and at home, in order to avoid all unnecessary excitement from ridicule and the like. If the chorea be only moderately severe, it is not necessary that the child should be in bed; we may even allow moderate exercise in the open air. Where the motions are violent, we should seek to guard the patient from self-injury by means of pillows and cushions.

Among the remedies recommended for chorea, the chief place is occupied by arsenic and potassic bromide. Arsenic in particular seems often to be of value. We give Fowler's solution alone, or with equal parts of peppermint water or compound tincture of cinchona; we may begin with five drops, in water, two or three times a day, gradually increasing to eight or ten drops. [In severe cases larger doses may be given. It should be pushed up to twenty drops or more, stopping for twenty-four hours if symptoms of poisoning appear, and beginning again at the same dose. It should be freely diluted.—K.] In children under six, the dose should be made somewhat smaller. If the child be anæmic, iron may be given in addition; or, if there be great restlessness and loss of sleep, narcotics may also be administered. Antipyrine, in doses of seven to fifteen grains (grammes 0.5–1) several times a day, is sometimes of decided service in severe cases. We give it especially in cases associated with rheumatism. Many observers have seen good results from sodic salicylate, fifteen to thirty grains three times a day (grammes 1–2). If these remedies have no effect we may also try bromide of potassium in large doses, a drachm or more (grammes 3–5) a day. In severe chorea we have seen frequently undoubted benefit from the bromides. Numerous other drugs have been recommended: oxide of zinc, valerianate of zinc, nitrate of silver, and sulphate of copper. The efficacy of all these remedies is doubtful and at present they are very rarely used. Reiss has lately recommended physostigmine, one sixth to one third of a grain a day (gramme 0.001–0.002), best subcutaneously. [Sulfonal, three to five grains (gramme 0.2–0.4) three times a day, acts well, as an adjuvant to arsenic, in cases where there is much excitement. Exalgine, two or three grains (gramme 0.1–0.2) three times a day, has also been recommended.—K.] Narcotics should be employed very cautiously in chorea. Although chloral has been recently recommended for grave cases, there are instances known where this remedy has been followed by unfortunate results; but we may try the continued and cautious use of small doses of chloral, giving to children two to five grains (gramme 0.1–0.3), and to adults ten to fifteen grains (gramme 0.5–1) daily, or a few larger doses at night.

Hydrotherapeutics of a mild kind do good, and can easily be carried out in most instances. Thus, we may use lukewarm baths, a wet pack, or gentle sponging with water at 72°–82° (18°–22° R.) to great advantage. In some obstinate cases sweating seems of service. We have also repeatedly seen decided benefit from the use of methodical gymnastic exercises, consisting of regular flexion and extension of the arms, marching in time, etc.

Electricity may also be tried. A feeble current of galvanic electricity is applied to the head (in the region of the motor centers), or the spinal cord is galvanized. If there be points along the spinal column where pressure causes pain, it is said to be an excellent plan to apply the anode to them; but the results of electrical treatment are seldom very brilliant.

In the chorea of pregnancy, which sometimes is a very violent disease, the same remedies may be employed. If they do no good, artificial delivery may be required; after which, as we can attest, there may be a rapid abatement of all symptoms.

APPENDIX

CHRONIC HEREDITARY CHOREA. ELECTRICAL CHOREA. PARAMYOCLONUS (MYOCLONIA)

1. **Chronic Hereditary Chorea (Huntington's Chorea).**—Chronic chorea also manifests itself by severe choreic movements of the body, but in its nature it is a wholly different disease from ordinary chorea. It is notably of family (generic) or hereditary occurrence. The symptoms of the disease begin as a rule between thirty and forty. The choreic movements gradually become more severe and increase to the most curious and strange movements of the face and the whole body. The ability to walk and to use the arms in eating, dressing, etc., is retained for a long time; but every action is accompanied by distressing associated movements and twitchings of the whole body. The sensibility and the other nervous functions are unaffected, but mentally we notice a decided but gradual failure as time goes on, which may increase to complete dementia. The anatomical basis of this remarkable disease is still unknown. The disease is incurable. The remedies used in ordinary chorea never mitigate the symptoms.

2. **Electrical Chorea.**—Henoch gave this name to a form of chorea in children which differed from the ordinary chorea by the much quicker, lightning-like contractions coming on in single muscles (especially of the neck and shoulders).

The contractions give the impression that the muscles have been put in contraction by single galvanic shocks. They come on at rather long intervals of three to five minutes. The same name, "electrical chorea," has also been given to a peculiar disease, said to be endemic in Lombardy, which was first described by Dubini, and which is therefore known by the name of "Dubini's disease." In this affection also muscular twitchings occur, usually beginning in one arm and gradually involving all the extremities. After several weeks or months paralysis and muscular atrophy set in, and not infrequently, it is said, epileptiform convulsions and fever. Nothing definite is known as to the anatomical changes.

3. **Paramyoclonus Multiplex (Myoclonia).**—This name was first used by Friedreich for a case observed by him, in which there were clonic spasms in symmetrical groups of muscles of the arms and legs, which came on in paroxysms without any disturbance of consciousness. There was also a considerable increase of the tendon reflexes. Such cases have since been described in considerable numbers, but undoubtedly very different morbid conditions have been confounded with one another. We can regard as genuine paramyoclonus—that is, as a specific disease *sui generis*—only those cases where lightning-like contractions come on in single muscles, often without any visible effect of movement. The muscles of the trunk and extremities are most frequently involved. This peculiar morbid condition is on the one hand most closely allied to electrical chorea mentioned above, while, on the other hand, according to our own experience, it has a decidedly close relation to the different forms of *tic convulsif* (clonic facial spasm, clonic accessory spasm, *q. v.*). A large number of the cases described as myoclonia or paramyoclonus, however, are undoubtedly to be classed as hysteria (*vide infra*).

Unverricht and others have described a special form of myoclonia. It is characterized by its occurrence in various members of one family, by the extension of the twitchings to the lingual and pharyngeal muscles, and by its complication with epileptic attacks.

CHAPTER III

PARALYSIS AGITANS

(*Shaking Palsy. Parkinson's Disease*)

Ætiology.—In 1817 Parkinson described for the first time a disease which he named "shaking palsy." It is not of very frequent occurrence, and as yet little has been ascertained with regard to its ætiology. In most cases it develops very gradually, without any demonstrable cause. It almost always attacks elderly persons, being very rare before the thirty-fifth year. Sex does not seem to exert any great predisposing influence. Hereditary neuropathic tendencies can indeed be traced in some instances, but they are certainly of less potency in paralysis agitans than, for instance, in epilepsy. Special exciting causes have sometimes been observed, such as catching cold, violent emotional excitement, and traumatic influences (blows, falls, injuries, etc.). Berger reports two cases where the first symptoms appeared after an acute febrile disease (typhoid fever).

Clinical History.—Paralysis agitans has two characteristic symptoms, viz., (1) peculiar evidences of motor irritation, evinced by tremor, and (2) a condition of stiffness and persistent shortening of certain muscles, consequent upon which is a series of peculiar motor phenomena.

The trembling is generally the earliest symptom to attract the patient's attention. It usually begins in the hands, especially in the right hand, and then gradually involves the arm and leg on the same side, next the other arm and leg, and finally, in well-marked cases, the entire body. The form of tremor is very characteristic. There are rapid, uniform, oscillatory movements of varying extent. The tremor is usually greatest in the hands and arms. At the same time the thumbs and half-flexed fingers exhibit a movement which suggests spinning or pill-rolling. The forearm is generally flexed and extended in rapid alternation, but it is always very difficult to determine just what muscles contract. With regard to the trunk, it is often a question whether its tremor is of independent origin, or due merely to the motion of the extremities. Charcot states that the head and the facial muscles are never implicated, but there is doubt about this point. We ourselves, as well as other observers, have repeatedly seen independent tremor of the head. As to the face, the muscles about the chin seem to suffer chiefly.

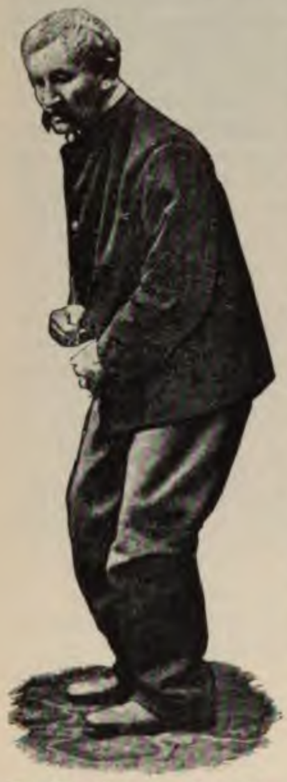

FIG. 178.—Characteristic position of the body in paralysis agitans.

The trembling of paralysis agitans is almost continuous. It may, indeed, cease for a moment in a limb, but only to recur immediately. The quieter the patient is in mind and body, the less violent are the movements. If he is excited, or begins to speak, or is watched, the tremor at once becomes exaggerated, and may be violent enough to jar the whole body vehemently. Active motion does not intensify the tremor. On the contrary, it may often be observed that the trembling abates when the muscles undergo vigorous voluntary contraction, as when a weight is lifted, or the hand of another is firmly grasped.

The second symptom is almost more characteristic than the first. It consists of a peculiar rigidity of the muscles. We generally notice, even in the face, a peculiar tension of the muscles. This often produces a stolidity of expression, so that the emotions are less clearly depicted than upon the countenance of a healthy person. We have been especially impressed in several cases by the infrequency of winking. The head gradually becomes more and more flexed. When the disease has lasted some years, the chin may even rest upon the sternum. The muscles of the trunk and extremities also stiffen gradually, and lead to peculiar and almost pathognomonic appearances. The body is bent over forward; the arms cling to the trunk, and are flexed at the elbow joint; the fingers are flexed, especially at the metacarpo-phalangeal joint; the thumbs rest against the fingers, as if holding a pen, or else are flexed inward upon the palm; and the legs are somewhat bent at the knee. The accompanying picture (Fig. 178) is from the photograph of a patient who was for a long time under observation at the clinique in Leipsic, and gives a good representation of the characteristic posture.

The stiffness of the muscles also operates to impair motion in various ways. In particular all movements of the trunk are greatly impeded. In advanced cases the patient can not get upon his feet, if he is lying in bed, without help. Inasmuch, however, as the muscular strength usually remains good (*vide infra*), he requires to exert but a slight traction upon some helping hand in order to attain an erect posture. On the other hand, the patient is often utterly unable to turn in bed from one side to the other. In severe cases, therefore, it is often necessary to alter the patient's position several times in a night, especially as lying long in one attitude makes him feel very uneasy. If the patient is in a chair, he can not get up of himself, because it is impossible for him to bend his body forward in the necessary manner; but with just a little help he can stand up, and he is then able to walk alone or even to run. Then, since the flexion of the trunk forward brings the center of gravity forward also, and the trunk can not be sufficiently bent backward, he is apt to "get going," so that he can not stop until he brings up against a post or a wall. If such a patient, with a considerable degree of anteflexion and rigidity of the trunk, is slightly pushed from behind, he will have to start into a run to avoid falling. This phenomenon is termed "propulsion." A push backward, which brings the center of gravity behind the point of support, is very likely to make such a patient fall, as he will seldom succeed in moving backward fast enough to recover his balance (retropulsion). Both propulsion and retropulsion are conceived by Charcot to be "forced movements" (see page 836) in the strict sense of the term. We are, however, convinced, as the result of numerous observations, that these phenomena can in every case be explained simply by the mechanical conditions arising from displacement of the center of gravity and the muscular rigidity. Again, the reason why many patients are prone to keep their arms behind them as they walk is that such a position contributes slightly toward bringing the center of gravity farther backward.

The movements of the extremities are less impaired than those of the trunk, but they often betray a certain slowness and stiffness. The strength of the muscles may be preserved for a long while, but often there is at last evident paresis. Even in the early stages of the disease the muscles may become easily fatigued. The impairment of facial expression has already been referred to. In many cases the muscles of the eye also seem to participate in the rigidity, making it difficult for the eye in reading to follow rapidly along each line, or to pass from the end of one line to the beginning of the next. [The voice is often affected; the patient talks in a high-pitched monotone.—K.]

The muscular rigidity is almost more characteristic of paralysis agitans than is the tremor. Indeed, there would seem to be cases, as we have ourselves observed, where, at least for a time, the posture of the patient is typical, and yet there is no trembling. Such cases might be called *paralysis agitans sine agitatione.* In uncomplicated cases all other nervous functions remain perfectly normal. Sensation is never impaired. Sometimes some pain is felt at the commencement of the disease, particularly in the shoulders. There is no striking disturbance of reflex action or of the bladder. In a few cases of paralysis agitans, cerebral and mental symptoms have been observed; but they are so rare that it is impossible to say whether they belong to the disease or are merely accidental complications. It is also noteworthy that many patients complain of a subjective feeling of excessive warmth. The internal temperature is normal; but it is said that the temperature of the surface of the body is frequently somewhat elevated. Sometimes there is a tendency to excessive perspiration. [Mosse and Banal have found that the excretion of urea and the total excretion of phosphoric acid is increased in paralysis agitans, but that the excretion of imperfectly oxidized phosphoric acid is diminished, thus showing that there is increased combustion.—K.]

The disease runs a very chronic course, perhaps for twenty years or more. From the first, it keeps on slowly but gradually developing. The symptoms rarely exhibit marked alterations of mildness and severity, but for long periods the progress of the disease may be apparently arrested. Recovery has never yet been observed. The final and fatal termination is not brought about by the disease itself, but is due to some intercurrent affection or to general marasmus. The original of the picture on the opposite page came to a pitiable end by tumbling face downward into a puddle of water. He could not get up, and was drowned.

Nature of the Disease.—The true nature of the disease is unknown. Inasmuch as the disorder is purely a motor one, the corresponding lesions must be sought somewhere in the motor system. As yet, however, post-mortem examinations of the nervous system, even with the microscope, have revealed no characteristic changes; at least we can ascribe no significance to the statements in this respect made by some observers. We must, therefore, confess that we have even had a doubt whether it is justifiable to claim that paralysis agitans is an affection of the nervous system at all, or whether it may not possibly be of purely muscular origin. Certainly it would not be impossible for abnormal processes in the muscles to excite the tremor and tonic contraction. But, as has been said, there is at present no ground for deciding this question; we would merely suggest it.

[Gordinier has found some perivascular sclerosis in the posterior portion of the spinal cord, with pigmentation and degeneration of the ganglion-cells and some dilatation of the vessels. He thinks the symptoms are due to irritation from an increase of the neuroglia in the posterior columns, causing excitability of the reflex collaterals of the posterior nerve-roots which arborize about the motor cells in the anterior horns. This causes an increased discharge of energy, which gives rise to tremor and rigidity. Other observers have found somewhat similar changes in the white matter of the cord, and also in the gray matter.—K.]

Diagnosis.—Any typical case of paralysis agitans can be easily and certainly recognized, often at the first glance. The important factors are the peculiar tremor, the characteristic carriage, and the rigidity of the muscles of the trunk and extremities. It was formerly a difficult matter to distinguish between paralysis agitans and multiple sclerosis; but to-day the peculiarities of the two diseases are better known, and confusion is seldom possible. The character of the tremor varies in the two. In paralysis agitans it persists even when the patient is quiet, and it is decidedly oscillatory. The motion in multiple sclerosis (*q. v.*) is almost always an intention tremor only; and, what is of still greater importance in distinguishing between them, the general appearances of the two diseases are essentially unlike.

Treatment.—As has already been implied above, we possess no means of controlling the disease. In most cases, therefore, the treatment is confined to general hygienic measures. Good may be done by lukewarm baths of considerable duration, and by gentle massage of the muscles. Among internal remedies Erb has lately recommended hyoscine, as having the most palliative and soothing influence. It is best given subcutaneously, and we must always begin with very small doses, as otherwise unpleasant symptoms, such as vertigo, headache, etc., may arise. We inject one two-hundredth to one one-hundredth of a grain (milligramme $\frac{1}{3}$–$\frac{1}{2}$) in aqueous solution (solution of hyoscine muriate, 1 to 1,000, seven to fifteen minims). If well borne we can cautiously increase the dose. [Atropine and hyoscyamine have also shown good results, especially when given subcutaneously.—K.] Arsenic, ergotine, potassic bromide, physostigmine, and curare may also be tried. Francotte praises the action of sulphate of duboisine (subcutaneously or in pills of $\frac{1}{120}$ grain, gramme 0.0005, three to six times a day); this also must be used with care. If electricity is to do any good at all, the case must be a recent one. It is said that in some instances stretching of the nerves has diminished the tremor considerably; but our own observations would not lead us to recommend the procedure in this disease.

CHAPTER IV

ATHETOSIS

IN 1871 the American neurologist Hammond described under the name of athetosis (ἄθετος, without fixed position) a peculiar symptom of irritation of the motor centers, differing in a characteristic manner from all other forms of involuntary movements, including the epileptiform and choreic. The movements of athetosis (see page 836) are often very complicated and peculiar. The part affected by them is in continuous unrest. If the facial muscles (usually those of the lower division of the facial nerve) and the muscles of mastication be attacked, the face and mouth are constantly being twisted and distorted. If the tongue suffer, as in one case which we saw, speech is difficult and indistinct. If the muscles of the back of the neck be implicated, the head is usually drawn backward or to one side, and is turned and twisted in all sorts of ways. Most characteristic of all, however, are the movements exhibited by the hand and fingers when affected. The fingers are incessantly being separated, extended, flexed, and intertwined, assuming the oddest positions. The accompanying pictures may serve to illustrate this (Fig. 179). The character of the movements reveals that the interossei must be chiefly involved. It is a very frequent result of the unceasing stretching of the articular ligaments of the fingers that at last the articulations become relaxed to

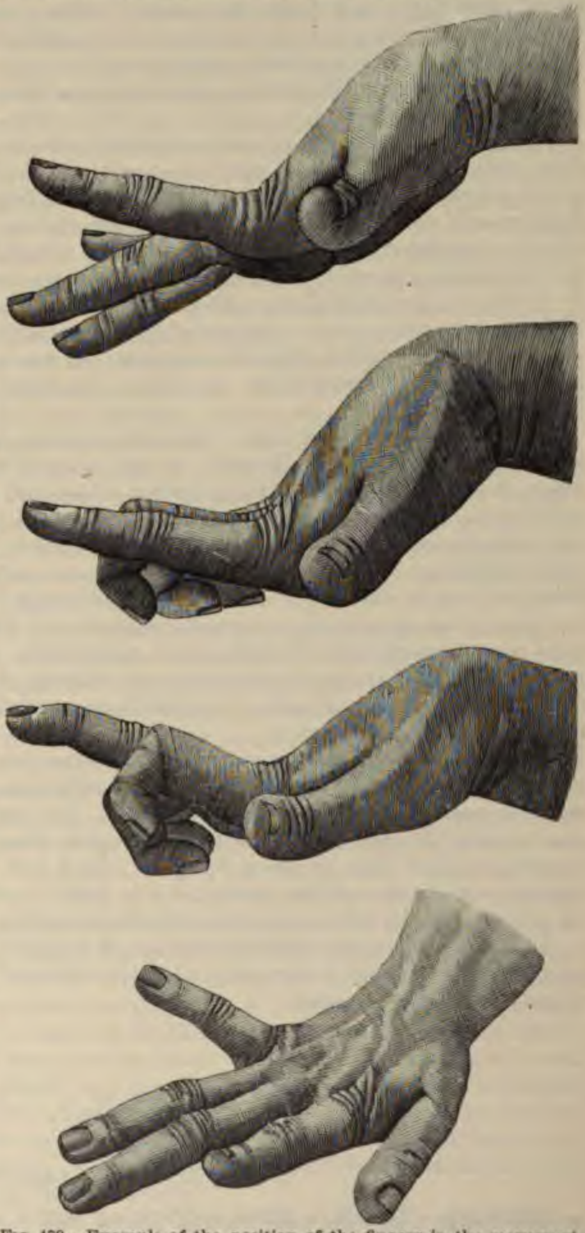

FIG. 179.—Example of the position of the fingers in the movement of athetosis (personal observation).

such a degree as to permit of such a hyperextension of the fingers, as it is impossible for a healthy person to imitate. The arms are generally less severely affected than the hands; and in the lower limbs the trouble is not often so severe as in the upper. The toes may, however, exhibit motions analogous to those of the fingers.

Although in general the movements are continuous, their vigor frequently varies. Thus they almost always are aggravated if the patient becomes excited. During sleep they generally cease, although in certain instances they have persisted even then, only being diminished. When voluntary motions are made they ordinarily grow feebler; but they may, on the contrary, become exaggerated, taking the form of sympathetic movements.

We must distinguish between genuine idiopathic athetosis and a symptomatic form, which also occurs.

Symptomatic athetosis is seen in various nervous diseases. The first observations reported by Hammond were, most of them, made in cases of epilepsy, or in severe psychoses, and the like. [In Hammond's original case the athetosis was symptomatic and due to a lesion involving the optic thalamus, the lenticular nucleus, and the most posterior portion of the internal capsule.—K.] By far the most frequent source of the phenomenon, however, is hemiplegia, producing what is known as post-hemiplegic chorea, or better, post-hemiplegic hemiathetosis. This is, to be sure, a very rare sequel to the ordinary hemiplegia of elderly persons, but follows rather infantile paralysis of cerebral origin (*vide* page 1112). Some traces of athetosis are seen in a majority of the cases of infantile hemiplegia.

Idiopathic athetosis is rare. Here the peculiar movements are the chief, if not the sole, symptom of disease. A few cases of this sort have been reported where the athetosis began without known cause, and usually was limited to some one region. It attacked elderly individuals who were previously healthy. Of especial importance is an apparently congenital form of athetosis, dating from the earliest infancy. Of this we have ourselves seen several instances, which closely resembled one another. The condition is a permanent one, not progressive, nor, on the other hand, capable of any great amelioration. The movements are almost always most pronounced in the face, head, and fingers. Other nervous disturbances, paralysis or impairment of sensation, are entirely absent or are associated with the athetosis in various ways. The intelligence of the patient is sometimes, but not always, diminished.

Of the nature of athetosis, or the locality or nature of the irritation, we possess no information as yet. It is extremely probable that the lesion is in every case a cerebral one. Perhaps it is in the cortex. In symptomatic athetosis we find post mortem the changes caused by the original trouble. In idiopathic athetosis no changes have thus far been reported. In a case of our own which came to autopsy, absolutely nothing abnormal was found in the brain. The patient was an elderly female, who displayed typical movements of the arm and hand.

It is not yet known whether recovery is possible. A certain amount of improvement sometimes follows the administration of Fowler's solution, bromide of potassium, or galvanism.

CHAPTER V

TETANY

(*Intermittent Tetanus. Tetanilla*)

Ætiology.—Tetany, a name originating with Corvisart, is applied to a peculiar neurosis, characterized mainly by paroxysmal tonic convulsions, usually painful, in certain symmetrical and bilateral groups of muscles. The disease attacks by preference children and young adults between fifteen and thirty years of age. The physiological processes peculiar to the female sex seem to have an especial tendency to excite the disorder. It is comparatively so frequent in nursing women that Trousseau has called it "*contracture des nourrices*." It is also remarkable that tetany is especially common in certain occupations, particularly in cobblers and tailors.

Among exciting causes, catching cold deserves particular mention. Hence the earlier observers describe the disease as "intermittent contracture of rheumatic origin." In other cases the disorder has appeared as a sequel to other acute diseases, such as typhus or typhoid fever, small-pox, and intestinal troubles. A very remarkable fact was pointed out by N. Weiss—namely, that tetany is apt to follow operative extirpation of goître. We have once observed attacks of tetany in a patient with exophthalmic goître. Its occurrence in gastric diseases is also interesting, it being seen especially in dilatation of the stomach due to cicatricial stenosis resulting from ulcer of the pylorus with hypersecretion of gastric juice. The tetany of childhood is seen chiefly in patients with dyspeptic and intestinal symptoms. All these observations suggest the action of some toxic substance, and it is therefore worthy of note that attacks precisely like tetany have been observed in different intoxications (ergotine, alcohol, chloroform). In chronic alcoholic subjects tonic spasms like tetany are not very rare. Reports from various quarters indicate that tetany may sometimes be epidemic. Most cases ordinarily occur in the winter and spring (February and March). On the other hand, endemic influences also seem to us to have some importance. At any rate, the published accounts would seem to indicate that tetany is much more frequent in Heidelberg (Erb, F. Schultze), Breslau (Berger), and Vienna (N. Weiss), than in Leipsic, for instance, where it is one of the very rarest nervous diseases. [It is also very rare in America. Crozer Griffith a few years ago was able to collect only seventy-three reported cases, while forty or fifty cases have been treated at the hospital in Vienna in a single year.—K.]

From what has been said it is evident that we should probably not regard tetany as one single disease ætiologically. Just as we distinguish in epileptic attacks between genuine epilepsy and symptomatic epileptic (epileptiform) convulsions, so we may distinguish between "idiopathic" and symptomatic tetany.

Clinical History.—The paroxysm of tetany usually has certain prodromata, consisting of slight general discomfort and pain, and of a feeling of weakness and stiffness, most marked in the arms. These symptoms last some hours (at least) before the true convulsive stage begins. The extremities, and more particularly the fingers, almost always suffer first; and then, after the arms, the lower extremities become involved. The spasm usually affects the toes before it seizes upon the other parts of the leg. The symptoms are almost invariably bilateral and symmetrical. Exceptionally, the disturbance commences in the leg, or is confined to one side of the body. In most cases the flexor muscles are predominantly affected, giving rise to very characteristic postures. The fingers are in apposition with one another, and placed as if holding a pen, or, as Trousseau says, as if the hand were about to be thrust into the vagina, during labor; but in the attack the fingers may also be closed in a fist. The hands are flexed, the elbows are also slightly flexed, and the upper arm in severe cases is pressed against the chest. In the lower extremities, the toes are flexed, and the feet are in the posture of talipes equinus. The muscles of the thigh are less frequently affected, as are also the muscles of the trunk, the muscles of mastication, the facial and ocular muscles, and the diaphragm.

The intensity of the tonic spasm is very great. The affected muscles feel as hard as a board, and are usually rather sensitive to pressure. The attack sometimes continues only a few minutes, but not infrequently it may occupy several hours or days. Other nervous symptoms are less prominent; the most noticeable are paræsthesiæ, tearing pains, etc. Anæsthesia is very rare. The reflexes are generally normal. There is no impairment of consciousness. In only a few cases has a combination of tetany and hallucinatory confusion been seen. In a few instances slight œdematous swelling and other trophic disturbances have been observed, and sometimes profuse perspiration. The temperature is normal, or sometimes subnormal or slightly elevated (100.5° to 101° [C. 38°–38.4°]), and the pulse is often quite rapid. Polyuria has sometimes been observed.

When the attack ceases, which it always does gradually, and never suddenly, the patient feels perfectly well, save for a slight pain and stiffness in the muscles, but all the muscles often feel weak and uncertain during the whole period of the disease. Even in the interval between the paroxysms there are also some few objective symptoms, which have a most important bearing on the pathology of tetany. In the first place, the peripheral nerves are generally abnormally sensitive to electricity (especially to galvanism, less regularly to faradism). The complete demonstration of this fact we owe to Erb. The weakest current will frequently produce violent contractions. The different sensory nerves and nerves of special sense also seem often to exhibit a similar abnormal irritability (Hoffmann), and finally we very often find a considerable increase in the mechanical irritability of nerves, which is seen with especial frequency in the facial (Chvostek, N. Weiss). Thus, if the face be vigorously stroked from above downward, with the finger or the handle of a percussion hammer, almost all the muscles contract energetically, one after the other. Similar contractions are, of caurse, obtained by light percussion of the different branches of the facial. The direct mechanical excitability of the muscles, on the other hand, is not increased (F. Schultze).

Another very characteristic symptom was discovered by Trousseau—"Trousseau's sign." It is found in most cases, although not in all, and is this: a fresh paroxysm can at any time be artificially excited by pressure upon the larger arteries and nerves of the arm (particularly the median nerve and the brachial artery). It is not definitely known how compression accomplishes this; but compression of a nerve seems to be the main point, as the corresponding experiments on animals show, in which an artificial tetany is produced by extirpation of the thyroid. Berger found that mechanical or electrical irritation of certain painful points situated along the spinal column produces the same result.

The frequency of the attacks varies greatly in individual cases. As a rule, there are several paroxysms daily; but the intervals may last for days, or again may be almost inappreciable. The entire duration of the disease is generally several weeks, but many cases last for months or even years. It is noteworthy that when the paroxysms grow less frequent and violent there is also a gradual diminution in the hypersensitiveness of the nerves and in the reaction to Trousseau's test. As long as these symptoms persist, spontaneous attacks are also possible. Many persons seem especially disposed to tetany, so that the affection repeatedly recurs in them.

The termination of tetany is, as a rule, favorable, but in some cases death results, which of course is usually due to complications. In children the disease may have an unfavorable termination if the spasms involve the diaphragm or the laryngeal muscles. Nothing definite is known as to the special nature of tetany. No essential or positive anatomical lesions have yet been detected. The mptoms leave us in doubt whether the disease affects the peripheral nerves or nervous centers.

Diagnosis.—The diagnosis is not difficult if we only consider carefully the ptoms presented, the nature of the paroxysms, and the other phenomena above umerated. It is of fundamental importance, however, after determining that the attacks are tetany, to discover, if possible, their special cause, and especially to determine whether we have to do with "idiopathic" or symptomatic tetany. It should also be said that tonic spasms, like tetany, may also occur in the hands in hysteria. The peculiar tonic spasms of young children are not, in our opinion, to be regarded indisputably as tetany; a special form has already been described (*vide* page 881) under the name of arthrogryposis. This is characterized by persistent tonic contracture, not paroxysmal, and most pronounced in the distribution of the ulnar nerve on both sides. In such cases there is no incerased mechanical irritability of the nerves.

Treatment.—The main treatment, besides general hygienic measures (especially rest and nursing) and the consideration of all discoverable causes (gastric and intestinal diseases, chronic intoxications, excessive work with the hands, etc.), is electricity. The stabile current is passed upward through the nerves affected; the galvanic current is also applied to the spinal cord, and the anode is applied to the various nerve-trunks, with the cathode on the sternum. This last procedure sometimes dissipates a spasm actually present. Internal remedies, such as bromide of potassium, arsenic, and belladonna, rarely produce brilliant results. Tepid baths and the cautious use of cold sponging, with friction and massage, may be tried. The internal exhibition of thyroid preparations may sometimes be attended with good results.

CHAPTER VI

TETANUS

(*Lock-jaw*)

Tetanus is an acute infectious disease whose chief symptom consists of the occurrence of severe, extensive, tonic ("tetanic") muscular spasms. The specific cause of tetanus, the tetanus bacillus, was first discovered by Nicolaier in garden earth. Rosenbach first cultivated the same bacilli from the secretion of the wound of a man who died of traumatic tetanus. Since then the specific significance of the tetanus bacillus has been positively confirmed beyond any doubt by numerous investigations. These bacilli are distinguished by a little head (spur) at one end. If a small amount of these bacilli be injected under the skin of mice, the most violent tetanic spasms ensue in the animals thus treated. Since the development of the bacteria remains limited to the seat of the wound or the injection, it is *a priori* probable that the spasms are not excited immediately by the bacilli, but by a chemical poison produced by them during life. In fact, Brieger has lately succeeded in producing several alkaloid-like substances from tetanus-cultures, so-called toxines, which he terms tetanine, tetanotoxine, and spasmotoxine. All these substances are violent poisons, and, like strychnine, provoke the most violent tetanic spasms in the animals experimented on.

In regard to the method of infection in man, it occurs by far most frequently through open wounds (operation wounds, or small accidental injuries). Since the tetanus bacilli are found chiefly in the soil, we can readily understand that injuries of the feet in persons who go barefoot, or injuries of the hands in gardeners, farmers, etc., most frequently give rise to tetanus. Such cases were formerly called traumatic tetanus, in distinction from rheumatic tetanus, following exposure to severe cold, or idiopathic tetanus, which was thought to occur without any discoverable cause. In the cases last mentioned there was probably some wound infection which was overlooked (just as in erysipelas) on account of the smallness of the wound. It of course would not be impossible for the infection sometimes to take place in some other manner. *Tetanus neonatorum* is undoubtedly an ordinary wound tetanus, almost always arising from an infection of the umbilicus.

In Germany [and temperate latitudes generally] tetanus is a comparatively rare disease. Men are much oftener attacked than women. Tetanus is much more common in the tropics than in our climate. The frequency of the disease in negroes is well known. Tetanus is also not equally frequent at all times. Endemics and epidemics of tetanus have often been observed, especially in time of war. These have arisen in part from the unfavorable influence of certain external conditions, such as poor care, bad weather, sleeping on the wet ground, etc.

Clinical History.—In so-called "rheumatic" tetanus the symptoms usually begin soon after exposure to the exciting cause. There may be, however, an interval during which the patient feels perfectly well, or at most has only certain mild and indefinite premonitory symptoms, such as languor and headache. Similar prodromata may occur in the apparently idiopathic cases.

Traumatic tetanus seldom begins immediately after the injury has been received. Several days or even weeks may intervene previous to the outbreak of the disease. Here, too, there may be mild prodromata for a brief period preceding the graver phenomena. The patient's wound presents no specific appearances. Tetanus may be associated with either slight or severe injuries, whether treated carelessly or kept apparently aseptic.

The symptoms of the disease proper are the same in both rheumatic and traumatic tetanus. They usually begin gradually. Ordinarily, the first thing noticed is a feeling of rigidity and tension in the muscles of the face, lower jaw, and back of the neck. The stiffness spreads by degrees to the muscles of the back and abdomen. The disease is sometimes completely developed in a few hours, but sometimes not till after several days.

The persistent tension of the facial muscles gives the countenance a strange immobility. The brow is usually wrinkled, and the corners of the mouth are often drawn back in a "sardonic grin," or the face has a tearful expression from the deepening of the naso-labial folds and the drawing down of the corners of the mouth. Most prominent of all is the tonic spasm of the masseters, or trismus. The teeth are so firmly pressed together that it finally becomes impossible to open the mouth more than one or two millimetres. The eyes are staring, the pupils usually contracted. The muscles at the back of the neck draw the head somewhat backward, but in many cases it can still be moved quite well, although in other cases it is fixed by the spasm. The spinal column is bent forward, so that the trunk is convex anteriorly, permitting the hand to be passed between it and the bed—opisthotonos. The muscles of the back are hard and contracted. The epigastrium and the anterior part of the abdomen are flat. The abdominal muscles are as hard as a board. The lower limbs may be rigidly extended at the knee, and the adductors are also contracted, but as a rule the feet and toes are free from spasm. The arms generally can be quite freely moved, but the movements at the shoulder are usually decidedly impaired. Convulsive dysphagia, as seen in hydrophobia (*q. v.*), may occur, but it is rare (*vide infra*).

In many cases the continuous tonic spasm is occasionally interrupted by sudden and irregular paroxysms, during which all the affected muscles become still more tense. In severe cases this gives the whole body a violent shock, and makes the opisthotonos even more pronounced. In a very bad case the paroxysms are very frequent; in a mild case they are rare or almost indistinguishable. Sometimes they are apparently spontaneous, and sometimes they are evidently of reflex origin, being superinduced by external irritation. In severe cases the cause may be comparatively insignificant, such as a slight jar or noise.

If there are other nervous derangements, little is known about them—partly, no doubt, because it is seldom possible to make an extended examination of the patient. Sensation is said to have been impaired in some instances, but in others it is perfectly normal. The muscles affected by the spasms are usually the seat of severe pain. The cutaneous reflexes are almost always greatly exaggerated. In two cases which we saw very recently, the patellar reflex was much increased, and in one there was distinct ankle-clonus. Paralysis is extremely exceptional. There is often profuse perspiration. The forehead is usually studded with many beads of sweat. The intellect remains perfectly unclouded.

There is a special form of tetanus which must be briefly mentioned. It was first described by E. Rose, and is called "hydrophobic tetanus," or "cephalic tetanus." It occurs only in connection with injuries situated in the distribution of the cranial nerves—that is, in the face and head—and is characterized in most cases by violent spasm of the pharynx and œsophagus. This is in addition to the other ordinary phenomena of tetanus. The disease in many ways reminds one of hydrophobia. Another characteristic point is that in most cases there is facial paralysis on the injured side.

Tetanus, as a rule, gives rise to no disturbances referable to the internal viscera. In one case, however, in the Leipsic hospital, croupous pneumonia and acute nephritis came on a few days before the end. Often there are dyspnœa and a most harassing sense of thoracic oppression—symptoms due mainly to the convulsive rigidity of the muscles, by which the thorax is constantly maintained in the position it normally assumes during inspiration. Expectoration is impeded; and, finally, there may be such an accumulation of secretions in the mouth and air-passages as to cause a secondary diffuse bronchitis, or an inhalation pneumonia. Another occasional source of extreme dyspnœa is spasm of the glottis.

The pulse often remains normal for a long while, but it is usually accelerated, not infrequently reaching 120 or 160 beats a minute in severe cases. Such a pulse is small, and may be somewhat irregular. The temperature is at first usually normal, or slightly elevated (100°–102° [38°–39° C.]). Later it is almost sure to rise; and, as Wunderlich pointed out, it is often very high shortly before death—for instance, 107° to 111° (42°–44° C.). It is not rare for the temperature to keep on rising for a short time after death. No explanation of this terminal elevation of temperature has yet been furnished. It can not be the result of the increased production of heat occasioned by the muscular spasm, for in earlier stages the most violent convulsions are unattended by any such change. Authorities are, therefore, inclined to assume that at the last there is a paralysis of the centers which regulate the warmth of the body, just as is seen in other severe nervous disorders, such as meningitis, injury to the cervical portion of the cord, and uræmia.

Interesting observations have been made with regard to tissue-metamorphosis during tetanus. The excretion of urea is not increased. This fact agrees well with Voit's view, that muscular activity has no relation to the breaking down of albuminoids. Senator failed to find any increase of kreatine and kreatinine in the urine. Probably the production of carbonic dioxide is abnormally large in tetanus. At least, physiological considerations would strongly indicate this, although it has not yet been actually demonstrated. Occasionally traces of albumen and sugar have been detected in the urine. There is usually obstinate constipation, probably due to the persistent rigidity of the abdominal muscles; and, indeed, micturition is not a little impeded from the same cause.

It may be said, in regard to the general course of the disease, that there are severe and mild forms of the disease. What has been said above applies mainly to the severe form. In this, all the symptoms reach their extreme violence in a few days, the paroxysms occur in quick succession, and death usually takes place within a week or two. The fatal result is brought about by the suspension of respiration and by cardiac failure. Of course, the extreme difficulty of taking an adequate amount of food has an unfavorable influence. The bad cases seldom outlast the first week. If they do, there is some slight hope of recovery; the paroxysms may gradually become less frequent and less severe, until they finally cease altogether. The severer form, however, so rarely ends favorably that the prognosis is always very grave. The mild form, on the contrary, usually runs a much more favorable course. In it, all the symptoms are from the first much less severe. Often there is only more or less trismus, accompanied by no marked spasm in the muscles of the trunk, if any at all. There is little constitutional disturbance. The temperature is normal, and the prognosis is rather favorable. The disease may sometimes drag on for some weeks, but it often ends in complete recovery. It must not be forgotten, however, that what at first seems a mild case may develop into the severe form.

The anatomical changes in the nervous system in fatal cases are almost wholly negative. Further investigations are necessary to determine how far the finer changes in the motor cells of the spinal cord, which have repeatedly been found of late, are of pathological significance. Any small hæmorrhages, etc., have, if they be present, only a secondary significance.

Diagnosis.—In most cases, tetanus can be easily recognized from the peculiar convulsions and the general aspect of the disease. It might be confounded with acute meningitis, for this may cause rigidity of the neck and back; but here there are usually certain cerebral symptoms also present, such as headache and impairment of consciousness; and, on the other hand, in tetanus, trismus is an almost constant phenomenon, although exceptional in meningitis. Strychnine poisoning produces convulsions similar to those of tetanus, but they generally affect the extremities in a more marked degree. Hydrophobia is distinguished from tetanus by the ætiology, the absence of trismus, the predominance of the pharyngeal convulsions, and the greater distinctness of the individual paroxysms. The hysterical conditions similar to tetanus can usually be easily distinguished from genuine tetanus by the discovery of specific hysterical symptoms (anæsthesia, etc.).

Where trismus is the only symptom, we must guard against mistaking for tetanus the symptomatic rigidity of the jaws which occurs with severe sore throat, diseases of the teeth, or inflammation of the maxillary articulation.

Treatment.—Through the recent bacteriological investigations the treatment of tetanus seems to be directed into entirely new channels. Animals may be rendered immune to tetanus by inoculation with attenuated cultures of the specific germs just as they can be to many acute infectious diseases. The blood serum of such animals then contains substances ("antitoxine" or "alexine") which annul the action of the tetanus toxine (Tizzoni, Behring). A considerable number of experiments upon the therapeutic usefulness of the tetanus serum have also been tried on man, but the reports upon the therapeutic benefits are still very contradictory. It is, however, not only justifiable, but urgently desirable to inject the tetanus serum as early as possible in every severe case of tetanus infection.

If the physician can not obtain an antitoxic serum, he must look to the methods of treatment heretofore employed. We have ourselves treated all the cases we have seen in the last few years with salicylic acid (eight grains, gramme 0.5, every hour), and we believe that we have repeatedly seen a favorable influence from this remedy. Otherwise narcotics are to be used. Those chiefly recommended are subcutaneous injections of morphine, opium in large doses, and chloral, of which thirty grains (grammes 2) should be given two or three times a day, and the amount gradually increased. If deglutition be very difficult, the chloral may be given by enema. Bromide of potassium should also be mentioned (at least two and a half to four drachms, grammes 10–15, daily), and calabar-bean (a sixth of a grain of extract of physostigmine, gramme 0.01, three to five times a day). The above remedies diminish the irritability of the nervous centers. In curare we possess a means of lowering the excitability of the terminations of the motor nerves in the muscles. It has therefore been employed by many, but by few with success. It is difficult to say what the dose of curare is, inasmuch as the strength of different samples varies. The best way is to determine the strength of the solution to be employed by experimenting on some animal. Usually a one-per-cent. solution of curare in water is employed, and an amount equal to one quarter of the contents of a Pravaz's syringe is injected, the dose being gradually and cautiously increased. [Such a syringe contains about thirteen minims (0.8 gramme).]

It is very desirable to put the patient by himself in a darkened and quiet chamber. Nourishment should be liquid, and lukewarm stimulants, such as alcohol and camphor, should be given from the first. Protracted warm baths may be given cautiously. We know from personal observation that such baths are very grateful to some patients.

It need hardly be said that in traumatic tetanus the primary wound should receive careful attention. Since, according to recent investigations, the tetanus bacilli remain limited in their growth to the seat of the wound, it may be indicated in the beginning of tetanus, if possible, to amputate the wounded part or to excise the wound. Of course, from present experience, we can not promise a successful result.

CHAPTER VII

CONGENITAL MYOTONIA

(Thomsen's Disease)

In 1876, Thomsen, a Schleswig physician, described a peculiar disease which up to that time had escaped observation. He had had experience of it in himself and numerous members of his own family. Thomsen called it "tonic convulsions of the voluntary muscles," an appropriate but somewhat clumsy name, for which we suggested instead "congenital myotonia." Apparently the disease is very infrequent; but a considerable number of cases have already been reported in Germany, France, and Italy.

The disease is probably always congenital; at least, the symptoms invariably date from the earliest infancy. It is very often generic and hereditary; and males of the same family seem to suffer oftener, and also more severely, than do females. The essential symptom of myotonia is this: whenever any voluntary muscle has been inactive for a time and is then made to contract, it falls into a state of more or less persistent contraction, a mild sort of tetanus, so that it can not be immediately relaxed. It is obvious that this would interfere with any series of motions, and make voluntary movement difficult. The patient is not paralyzed at all, but he has a feeling of great resistance to be overcome in performing any act. Quick and accurate motions are often out of the question, so that, for instance, the patient can not perform military duty. It is noteworthy that the stiffness temporarily disappears after the patient has been moving his muscles for some time. On going up-stairs, the first steps are often very stiff and laborious, while succeeding ones grow easier and easier. Mental excitement invariably exerts a very unfavorable influence, exaggerating the stiffness of the muscles.

Upon physical examination, the observer is usually struck by the extraordinary development of the muscles. The size of the extremities, in particular, almost deserves the term "genuine muscular hypertrophy," although the strength is not always proportionately great. Certain extremely interesting variations in the electrical excitability of the nerves and muscles are, however, very remarkable. These were first studied in detail by Erb and given the name of "myotonic reaction." The motor nerves show quantitatively a normal faradic and galvanic excitability. All single shocks give short contractions, but on continued excitation (faradic current, labile galvanic current), a very characteristic persistence of the contraction occurs after the cessation of the irritation. The muscles are very easily excited by faradism, and, with rather strong currents, always show the above-mentioned persistence of the contraction. When the current is applied continuously peculiar wave-like, oscillating muscular contractions occur. On galvanic excitation of the muscles a slight increase of excitability is manifest. With rather strong currents the contractions are decidedly slow, tonic, and also persistent. They occur only on closing the current, not on opening. Finally we almost always see in the muscles a very remarkable phenomenon, discovered by Erb: rhythmical wave-like contractions occur in the muscles, under the action of a stabile current, which start from the cathode and go to the anode. The direct mechanical excitability of the muscles, on percussion, is usually increased, but this does not apply to the idio-muscular contractions. The reflexes, sensation, and, indeed, all other nervous phenomena, are normal.

These facts render it very probable that the cause of the disease is to be sought in the muscle itself, and that myotonia is due to a congenital peculiarity of the muscular system. This hypothesis is supported especially by the interesting histological discoveries of Erb on excised bits of muscle. He found a considerable increase in the volume of the individual fibers, whose transverse striation was often very fine and indistinct, at times the formation of vacuoles in the fibers, a great increase of nuclei, and a moderate increase of interstitial connective tissue. Nothing is known at present in regard to anatomical changes in the nervous system. Erb reports that, upon microscopic examination of minute particles of muscular tissue which were excised, he found marked hypertrophy of individual muscular fibers, and an increase in the number of nuclei in the sarcolemma.

The disease persists through life. The patient gradually becomes accustomed to it, and learns to conceal his misfortune as much as possible. There may be no constitutional disturbance. Sometimes there is melancholia. Therapeutic efforts have not yet been attended with much success. We might try cold sponging, with friction, gentle massage, and methodical exercise of the muscles.

[Eulenburg describes a congenital family affection, which he calls congenital paramyotone, allied to myotonia, where tonic spasm was produced by cold. Gowers has found the association of tonic spasm with ataxia—ataxic paramyotone.—K.]

CHAPTER VIII

CATALEPSY

Formerly catalepsy was regarded as a special form of disease, but at present the opinion is almost universally held that it is merely a symptom of several different diseases. As was mentioned on page 837, "cataleptic rigidity" is a term descriptive of that peculiar condition of the muscles in which the limbs maintain involuntarily any position into which the observer puts them. If we change the position of the members which are cataleptic, the patient does not make the slightest effort to alter the posture in which we leave them, however strange, awkward, and apparently insupportable, it may be. The limbs may be moved in this way almost like wax, and they are therefore said to exhibit a "waxy flexibility."

No real explanation of the cataleptic state has been given. We have not yet advanced beyond the study of the circumstances under which it appears, and of the associated phenomena. As has been said, the tonic muscular spasm of catalepsy is never very great, being little more than sufficient to overcome gravity and maintain the limb in the posture given to it. This shows that there must, in every case, be a due proportion in the vigor of the contractions of antagonistic muscles; and this relative force must vary with every change of position. How this continuous and remarkable regulation of motor nervous energy takes place is an unanswered question. Perhaps reflex action has something to do with it. A further interesting point is, that changes of position induced by electrical stimulation of the nerves or muscles are not permanent; when the stimulus ceases to act, the limb falls back into its old place.

Catalepsy occurs oftenest as a symptom of hysteria (see the following chapter). In this case it is usually associated with other disturbances, chief among which are impairment of consciousness and anæsthesia. The anæsthesia is especially marked in the muscles. For example, the patient will stand for an hour with arms extended, and yet experience not the slightest sensation of weariness. At last, however, the arms sink slowly down. Closely allied to hysterical catalepsy is hypnotic catalepsy, a condition which can be artificially produced by certain procedures in many hysterical subjects at will (see the next chapter). Charcot has reported cases where the lethargy of hypnotism could invariably be transformed into catalepsy by opening the previously closed eyes. These cases also presented, in addition to the catalepsy, the strange phenomenon called "suggestion." If the patient were put into any posture associated with some definite mental conception (such, for example, as the attitude of prayer, or that assumed in terror, or to express detestation), then the corresponding thoughts would come into the mind, as a hallucination, but with all the vividness of reality. Ample proof of this was visible in the expression of the face and in the whole bearing of the subject. An analogous fact has been observed by Duchenne, Lasègue, and ourselves—namely, that sometimes a hysterical person can be brought into the cataleptic state by artificial closure of the eyes (compare what is said in the next chapter about hysterical anæsthesia).

Catalepsy is also seen in many psychoses, particularly in certain grave forms of melancholia (melancholia attonita, katatonia), and sometimes in progressive general paralysis. (For particulars see works on insanity.) The cataleptic state may also develop in connection with grave organic cerebral disease, as in meningitis and apoplectic coma. It may finally be mentioned in passing that quite well-marked catalepsy is sometimes observed in young children of one or two years when they are ill. Probably they fall into a sort of stupor; or often it seems that they are rendered hypnotic, as it were, by the presence of strangers.

Catalepsy has been regarded as a special disease in those very rare cases where otherwise healthy persons are liable to "cataleptic fits." The condition comes on suddenly, unprovoked, and passes off spontaneously after a variable length of time. It is, however, very probable that such of these cases as are not hysterical are epileptoid, and deserve to be classed with genuine epilepsy.

Prognosis and Treatment.—Since catalepsy is a symptom of so many different diseases, it is impossible to make any general statements with regard to prognosis or therapeutics. The reader may gain some light from the remarks on the treatment of hysteria in the next chapter.

CHAPTER IX

HYSTERIA

Ætiology and Definition.—It is impossible to give a definition of hysteria that shall be at once brief and accurate, for the aspects of the disease are so manifold that there is no one symptom which can be called pathognomonic or even universally characteristic. Hysteria is therefore symptomatically not a well-rounded morbid unit, although the nature of all those pathological conditions which we term hysterical may very well be regarded from a single point of view. Hysteria thus has its peculiarities and its rules like any other disease. Only because its laws have been sought elsewhere than where they really are, has the claim often been made in the past that the morbid symptoms of hysteria were subject to no law.

The fundamental condition for a right understanding of hysteria seems to be that we should free ourselves from the old untenable idea that hysteria is a "general functional disease of the whole nervous system, where now this and now that portion of it may be disturbed in its functions." We regard hysteria as a disease which relates to the cerebral activity immediately associated with the psychical processes—that is, one may say, with a psychosis, but in the broader sense of the word; the disturbance affects not only the normal course of the psychical processes, but also their association with the purely corporeal processes of innervation. We therefore call any symptom hysterical which is due to a disturbance of the normal relations between the processes of our consciousness and our corporeity. Between our ideas of volition and our muscles, as between the excitations of our sensory nerves and our consciousness, there exist under normal conditions very definite and very well ordered connections. If there comes a loosening, a displacement, a false estimate in these connections, it gives rise to a "nervous" disturbance of motion or sensation, a "hysterical" symptom; but the starting-point of the disturbance always lies ultimately in the psychical domain. If, for example, the idea of volition suffers a morbid change in the sense that it can no longer arouse a corporeal motor excitation in any muscular territory, a "hysterical" paralysis follows. If the consciousness turns permanently from definite sensory impressions that come to it, we have to do with a hysterical anæsthesia. Later deductions will prove more plainly this characteristic psychogenetic origin of all hysterical symptoms.

In the meantime we may add to what has been said the following characteristic features of hysterical affections:

1. All hysterical disturbances, no matter how severe the functional nervous derangement attributable to them, are without visible anatomical basis. The best proof of this is the fact that any hysterical affection, however severe, may sometimes develop and then may recover completely in a very short time.

2. The hysterical affection is very often most intimately associated with discoverable causes of a psychical nature. Not only is its appearance and incipiency most closely linked with emotional excitement, but later on the mind is the main if not the only channel through which causes can operate to change the condition of the patient, whether favorably or unfavorably.

3. It is therefore evident that the origin of all hysterical disturbances must be sought in the most central portions of the nervous system—those regions which are most directly concerned in the mental processes. Hysterical phenomena are, however, exhibited in all parts of the nervous system, so far as these depend upon psychical processes or may be altered by them. The symptoms of hysteria are therefore more manifold than those of almost any other disease. Although so

manifold, certain symptoms predominate with such frequency as to be characteristic of hysteria, and therefore to be regarded as especially valuable in diagnosis. Following Charcot, we term such symptoms "hysterical stigmata." These symptoms are most constantly present, and therefore they can easily be found at any time.

4. Besides the permanent symptoms—the "stigmata"—we see in many cases of hysteria peculiar nervous attacks. These are also highly characteristic of hysteria, since they come on in forms which are seen in no other disease; but there are also patients who suffer from hysteria who nevertheless are never visited by attacks. The possibility of the occurrence of an attack is, however, always present. Very often the attacks are provoked by easily discoverable psychical causes.

After this brief introduction, turning to the special ætiology, psychical causes, as has been said, are of first importance. In numerous instances hysteria comes on as an immediate sequel to violent emotional excitement or to a "psychical trauma," if we may use such an expression. Hysterical convulsions or paralysis may be excited by great terror or violent anger or any unusual agitation. Here the efficient psychical cause is often hidden by some attendant circumstance. If, for example, a patient falls into the water, or gets burned, or tumbles downstairs, and thereupon develops hysteria, the mistake is often made of ascribing the subsequent nervous disease to catching cold, or to the injury received, although really it was the mental excitement which produced it. The various physical injuries (falls, blows, burns, etc.) play a far greater, and perhaps the greatest, part in the genesis of hysterical conditions. In such cases it is not the physical injury itself, but the terror it causes, or rather the whole disturbance of consciousness due to the trauma (terror, anxiety about the consequences, fright, etc.), which produces the hysterical symptoms. These nervous affections produced by physical injury (traumatic hysteria, traumatic neuroses) are of such great practical importance that we will devote special consideration to them later.

What is very remarkable in such cases is that the special attendant circumstances of the psychical disturbance often influence the localization of the hysterical phenomena: that part, to which attention was particularly directed at the time, not infrequently becomes, later on, the seat of the nervous disturbance. In hysterical joint-affections (page 821) the cause not infrequently proves to have been an injury to the particular joint now suffering from hysteria. A young girl, who was awakened at night by the smoke of her burning mattress, and who had a severe laryngitis from inhaling the vapors, exhibited later an indubitably hysterical paralysis of the vocal cords. In the case of another girl, who in jumping from a carriage fell upon her side, we afterward saw hemianæsthesia of the same side. Many such instances could be cited.

Certain hysterical cases, therefore, have for their obvious cause a single occasion of mental agitation; but in many others the disease is not thus abruptly excited. As, in poisoning, we can distinguish between the sudden action of a large dose and chronic cases where minute amounts of poison are absorbed daily for a long period, so hysteria may come on not only after a single violent shock, but also as the final consequence of psychical influences insignificant in themselves, but potent because of their frequent repetition or persistency. It is in cases of this sort that the causes do not become evident to the physician until he has gained the entire confidence of his patient; for the root of the trouble is often entwined about the most private affairs. Anxiety, sorrow, disappointed expectations, abandoned hopes, and, in brief, everything which can depress and overwhelm the mind—these are factors which may at last excite the functional nervous derangements of hysteria.

Finally, we must state that hysteria may often develop without any specially noticeable cause. We see this particularly in young people with a marked hereditary nervous taint. Here the ordinary daily demands upon a central nervous system, which had originally an abnormally weak constitution, sometimes suffice to disturb in a measure its normal equilibrium.

This leads us to a second extremely important point in the ætiology of hysteria. The blow which brings a feeble body to the ground rebounds without effect from a massive frame. Exactly the same may be said of the "blows" to which the nervous system is subjected. Few entirely escape these influences, but there are some "strong natures" who resist the psychical assault without wavering, while others have a feebly resistant ("inferior") nervous organization and are overpowered. We see, therefore, that the predisposition of different individuals to diseases of the nervous system varies. This fact is a very important one in the pathogenesis of all functional nervous disorders. In what this predisposition consists we do not know, being acquainted only with its results and with some of its causes.

In many cases this predisposition is hereditary. Hysteria is most prominent among those neuroses which attack different members of a family—one suffering from one disease and another from another (*vide* page 1141). It is also possible to acquire such a predisposition. At least it may be developed and fostered, on the one hand, or, on the other, checked and repressed. In these directions physical as well as psychical factors are of importance. Anything which weakens the constitution diminishes the resistant power of the nervous system. Among psychical influences, nothing favors the development of hysterical tendencies more efficiently thàn does a bad education. Hysteria is often ascribable to an irritability and feebleness of the nervous system thus engendered. The whims of the child are not controlled, its will is not strengthened, or its energy developed; its imagination is unsuitably and excessively stimulated, or its intellectual powers are overtaxed and prematurely ripened.

It is well known that hysteria occurs more frequently in the "feebler" female sex than among men; but it is by no means a rare thing for men to exhibit hysterical convulsions, paralysis, contracture, or other well-marked disturbances. Most cases occur between puberty and the end of middle life. It is quite common to see pronounced hysteria in children, especially such as are over eight or ten years old. The disease very frequently begins to develop a year or two previous to puberty. Nationality and race also seem to exert some influence—for instance, the severer forms of hysteria are decidedly more frequent in France than in Germany, but severe hysteria is by no means rare in Germany. The Jewish race are particularly subject to hysteria, and to nervousness in general.

One matter remains to be considered with regard to ætiology, to which a greatly exaggerated importance was formerly ascribed. It is the influence of disease of the sexual organs. The very name "hysteria" (ὑστέρα = uterus) reveals what the old view was, namely, that hysteria invariably originated in disease of the female genitals. Not to speak of the fact that the disease occurs in men and children, an unprejudiced consideration of the matter will show that the above assumption is entirely groundless even in regard to women. A large number of hysterical women present no anomaly of their sexual organs; and even if the latter be diseased, we are not justified in assuming at once that the hysteria is secondary to the sexual disorder. In these cases, also, we usually find, on careful inquiry, that psychical causes have been at work; and these are incomparably more potent in exciting hysteria than is malposition of the uterus or constriction of the cervical canal. It is true, however, that disease of the genital organs may depress the spirits more than some other diseases would, and so indirectly promote hysterical disturbances. In the same indirect way, menstruation, pregnancy, and parturition exert an important influence upon the development and course of hysteria. Sexual excesses or total abstinence from sexual indulgence also produce their effects indirectly through the mind.

THE SYMPTOMS AND MANIFESTATIONS OF HYSTERIA

Considering the great differences in the external morbid symptoms under which hysteria may manifest itself, it is not easy to give in a brief summary a comprehensive, intelligible, and yet accurate description of its clinical manifestations. We believe that we can best attain this end by speaking first of the most important and most frequent of the single symptoms of hysteria, by describing next the hysterical attacks, and finally by trying to evolve a general picture of the disease.

1. **The Hysterical Stigmata, especially Sensory Anæsthesia and Hyperæsthesia.**—In every case where the diagnosis of hysteria has already been established or where it is to be confirmed, we must examine the patient for certain symptoms, which, as we have said, are very common in hysteria, and in part so peculiar to it, that by themselves they are often of decisive diagnostic significance. These symptoms are termed "hysterical stigmata." A knowledge of them is the more important for the physician in that we often detect them only by a special examination directed to that end. Patients only exceptionally inform us as to these symptoms. It is not at all unusual for patients to have no inkling of their existence until after an examination.

The most important, because the commonest, of these hysterical signs relate to sensibility. The more carefully we examine, the more rarely will we meet with a case of severe hysteria in which in some sensory region we do not find manifest disturbances, especially a diminution of sensibility. We must therefore test accurately not only the sensibility of the whole surface of the body, but also the functions of all the special senses (sight, hearing, smell, and taste).

The sensory disturbances of the skin must first be mentioned. Not infrequently we find a diminution of sensibility over the whole surface of the body, especially a more or less complete analgesia. In such cases we can stick a pin in deeply anywhere, or pierce a fold of skin with a pin, without the patient's complaining of pain. As is well known, it often happens that hysterical patients, in order to appear interesting or from some other reason, produce upon themselves deep injuries and wounds; this is almost always connected with the patient's analgesia. Very often, however, the anæsthesia is not general, but it is limited to a definite region. We find complete anæsthesia of one arm or leg, or we find on the trunk or the extremities single areas of anæsthesia, in which the anæsthetic parts are bounded by the normally sensitive skin; these areas may take all possible forms, and may often be very peculiar. The sensibility to pain is almost always the most disturbed, even when the other forms of cutaneous sensibility may be quite unaffected, but changes in the sensibility to temperature, pressure, etc., may also occur. It may be added that in the severer forms of anæsthesia the deeper parts may also be insensitive, so that the so-called muscular sense is absent, and patients with their eyes shut have not the slightest idea of the position of the anæsthetic limbs. [The skin reflexes are usually diminished or absent over the anæsthetic regions. In hemianæsthesia the gagging reflex from irritating the back of the pharynx, palate, or tongue, may be absent on the anæsthetic side.—K.]

Besides cutaneous anæsthesia, disturbances in the sensibility of the other sensory organs (other "sensory anæsthesias") are among the commonest signs of hysteria. Sometimes the patients say, when asked, that they see indistinctly and dimly. If we examine the eyes we often find a loss of visual acuteness and a rapidly induced fatigue on using them. A limitation of the visual field is the most characteristic sign—that is, an anæsthesia of the peripheral portions of the retina. The degree of this limitation sometimes shows manifest differences for the different colors. Hysterical achromatopsia is a common symptom—that is, a partial or complete loss of the color sense. According to Charcot, in the hysterical the perception of violet usually first disappears, then green, and lastly blue and yellow; but we must not hold too strictly to all these rules; for, although the French investigators are otherwise deserving of great praise in adding to our knowledge of hysteria, in this respect they seem decidedly too schematic, and to have generalized too rashly. Diminution of the auditory acuteness in one or both ears is also not uncommon. Anæsthesia of smell and taste is still more common. Salt, quinine, vinegar, sugar, etc., either excite no sensation of taste at all, or the tongue is anæsthetic for one or another of these substances. The same holds for the sense of smell. All these symptoms are especially important in diagnosis, because in organic diseases they seldom reach such a degree as in hysteria.

The sensory anæsthesias above described may of course be combined in various ways in different cases, but none of these combinations is so characteristic and individual as hysterical hemianæsthesia, a symptom-complex which occurs almost solely in hysteria, and is therefore of distinct diagnostic value.

Hysterical hemianæsthesia is one of the most common symptoms of profound hysteria. It must often be sought for, inasmuch as the patient herself frequently has no suspicion of its existence until her attention is called to it. It seems just as if one half of the body had been entirely lost to consciousness; the patient does not know whether it is or is not capable of feeling.

In a typical and fully developed case the hemianæsthesia does actually affect just one half of the body. There are rudimentary forms; but in these complete ones, the boundary between the anæsthetic parts and the parts retaining normal sensitiveness accurately corresponds to the median line of the body. The skin on the affected side is entirely insensible to the prick of a needle or to heat. Often it seems somewhat blanched, and its blood-vessels seem to be constricted; at least the skin very often bleeds surprisingly little if wounded. The mucous membranes upon the abnormal side are all equally anæsthetic, including the conjunctiva, and the corresponding half of the buccal cavity and of the tongue. The deeper parts, such as the muscles and joints, are also almost invariably anæsthetic. The patient can no longer feel in what position the limbs of the affected side are; and if they are moved passively, no sensation is communicated to her. The organs of special sense are usually involved. Hearing is impaired upon the affected side; the corresponding half of the tongue can not taste; the corresponding nostril can not smell; and sight upon that side is affected in a peculiar manner. There is no hemianopsia, but a total amblyopia, or possibly amaurosis. If the amaurosis be not complete, we find at least a limitation of the visual field and the signs of the above-mentioned achromatopsia.

Apart from other hysterical symptoms with which hemianæsthesia as well as all the other sensory anæsthesias may be associated, we must mention here one symptom which is in close relation to anæsthesia, and which was first described by Duchenne under the name of "loss of muscular sense" (*perte de la conscience musculaire*). The patient, whose arm, for example, is anæsthetic, can not move it if she closes her eyes, although with the eyes open she can move it as well as ever. When the eyes are shut the arm remains motionless in the position it has previously occupied. If its position be altered by passive motion, the new position is maintained with equal persistency. There is, with the eyes shut, pronounced catalepsy. Duchenne referred this peculiar symptom to the loss of a special sense which he termed "muscular sense" (*conscience musculaire*). According to our present conception of hysteria it would be more correct to regard the symptom as purely psychical.

[The various anæsthesias and many other symptoms of hysteria might often be termed more properly "delusions of not-feeling." Patients with hysterical blindness, for example, when prisms are placed before their eyes, so that they imagine they are looking with the sound eye when they are really looking with the "blind" eye, can sometimes see very well. When hypnotized such patients can recall that they have been touched or pricked in the anæsthetic area, etc. This is often mistaken for deliberate simulation, but it is really another instance of the psychical nature of the disease.—K.]

The much-discussed and peculiar symptoms of transfer, as well as metalloscopy and allied symptoms, will be mentioned below.

Thus far we have spoken only of anæsthesia, but we very often find in the hysterical certain hyperæsthetic regions. These hyperæsthesias are so characteristic that we must reckon them among the hysterical stigmata, and must therefore look for them in every case. Sometimes the physician's attention is called to these places because they are the seat of constant slight or severe pain. In other cases the tenderness appears only upon pressure. The hyperæsthesia may be so great that the patient can scarcely bear the slightest touch. Of course, the hyperæsthesia is very closely connected with the condition of the patient's attention. If the thoughts be diverted, even firm pressure is often not at all noticed. We see clearly, then, that we usually have to do here with a "psychical hyperæsthesia."

The hyperæsthetic spots are either quite extensive or quite circumscribed, or even limited to one definite spot. They may lie near to or in the midst of anæsthetic areas. They are relatively rarest in the extremities and more common on the head and trunk, especially on the sternum, on the sides of the chest, under the mammæ, etc. Most common, and therefore especially characteristic, is the hyperæsthesia of the vertebral column, and, in women, of the lower abdominal region ("ovarian pain," "*ovarie*"). The latter term is convenient but by no means correct, since really we have only a tenderness of the soft parts in general, which is at once noticeable on deep pressure, and we do not have solely or chiefly a tenderness of the ovary. We usually find *ovarie* only on one side, oftener on the left than on the right. Analogous symptoms in men—tenderness in the groins or the testicles—are less common. Hyperæsthesia of the vertebral column, hysterical "spinal irritation," is even more important for diagnosis than *ovarie*. The hyperæsthesia affects either the whole spine or merely single portions of it, often only single vertebræ. It may attain so high a degree that the slightest touch of the skin over the vertebræ, or somewhat deep pressure, may cause the liveliest outcries and shrinking. This symptom is almost pathognomonic of hysteria.

The relations which these hyperæsthetic regions have to the hysterical attacks are very important, and they have therefore procured for them the name of "hysterogenous zones." We will speak further on this point in describing the hysterical attacks.

Hyperæsthesia in other sensory regions—abnormal sensitiveness of the eye, ear, etc.—also occurs. Thus certain cases of hysterical blepharospasm seem to be due to an immoderate sensitiveness to light. Unusually sharp hearing is noted, especially during the hysterical attacks. Here, too, belongs the aversion of many patients to certain odors and flavors, and the like.

We may also mention in this place hysterical pains. They can scarcely be separated strictly from hysterical hyperæsthesia; at least, almost all hysterical patients, who complain of pain (in the back, in the face, in the arms and legs, and very often "all over"!), state that these pains are increased by motion and pressure. The more the patient's general disposition is disturbed, the more he thinks of his pain, and the worse it becomes. If the patient is busied with other things, he forgets his pain entirely. Suggestive influences (*vide infra*) are of great effect. There can therefore be no doubt that these feelings of pain are of a purely psychical nature; they are, so to speak, "imaginary pains," hallucinations of pain. Hysterical pains sometimes become very important because they render the patient inactive. There are hysterical patients who lie the whole day on the sofa, because they can not stir a limb "on account of pain." Möbius and others have described such especially pronounced cases under the name of "*akinesia algera.*" In our opinion, there is no reason for wishing to make a separate disease of such cases, because such conditions often enough occur alone or associated in various ways with other symptoms in severe neurasthenia and hysteria.

2. **Hysterical Paralysis.**—Hysterical paralysis is frequently an immediate sequel to some violent mental excitement (for example, "paralysis from fright"); but it may come on gradually. Hysterical paralysis is indubitably of central origin. It is a paralysis of the will. The patient has lost the power to will a contraction of the affected muscles. One always has the feeling that the patient could move the paralyzed limb perfectly well if she only desired to; but she can not bring the will to bear on it, and this inability is the real trouble. It also seems a remarkable circumstance that hysterical paralysis often, but of course not always, affects only certain combined movements. Many patients, for instance, can move their legs very well in bed, but they can not walk a step ("*abasia,*" *vide infra*). We have seen a hysterical writing paralysis; the right arm was not paralyzed, but it utterly refused to make any attempt to write.

The extremities are most frequently paralyzed, particularly the lower limbs; but hemiplegia is not especially rare. A very common manifestation is loss of the power to stand or walk. The patient lies in bed or on a sofa, and sometimes while thus reclining she can flex and extend the legs very well; but as soon as she is urged to stand up or walk, the knees double up, the patient begins to tremble, the respiration grows rapid and convulsive, and there is not the slightest effort made to move the legs. In France the names "*astasia*" and "*abasia*" are used for these common hysterical symptoms. If only one leg be paralyzed, the gait is often very peculiar and characteristic. The sound limb makes long strides, while the paralyzed one is held perfectly rigid, and often is dragged along with a loud shuffling sound. The arms are much less often affected. The facial muscles are hardly ever paralyzed, although they may be involved in hemiplegia. We once saw hysterical diplopia.

In regard to the more precise form of the paralysis, both flaccid and spastic paralyses occur in hysteria. In many cases of hysterical paralysis the tendon reflexes may be so exaggerated—nay, there may even be, as we have repeatedly seen, such a lively ankle clonus—that one is at first disposed to think of actual spinal paralysis. In other cases, as in paralysis of an arm, the paralyzed limb hangs down completely flaccid. The paralyzed parts are often at the same time completely anæsthetic (a circumstance which may often be distinctive in diagnosis), but in other cases they are normally sensitive or even hyperæsthetic.

Hysterical paralysis of the vocal cords is very often seen. The voice is generally lost suddenly, and the patient can talk only in a whisper—hysterical aphonia. On laryngoscopic examination, we are often struck, at the outset, by the anæsthesia of the pharynx and its lack of reflex excitability. We find no trace of any anatomical lesion of the cords, but merely that they are paretic. The glottis can not be completely closed, and sometimes the vocal cords actually become wider apart upon every effort at phonation. The patient then speaks exclusively in a whisper.—Hysterical mutism quite rarely follows hysterical aphonia. The patients entirely lose voluntary control of their speech apparatus, and finally become completely dumb. We will also mention here hysterical stammering, although it should more properly be classed with the spasmodic hysterical symptoms. The voluntary quiet innervation of the muscles of articulation is affected, so that the speech is altered in the most peculiar way, by needless spasmodic attendant movements, grimaces, etc. We have very often observed such hysterical disturbances of speech (stammering or mutism), especially in children (as a result of fright). A loss of the power of writing may sometimes be associated with the dumbness in an interesting way.

Hysterical paralysis of deglutition is much less frequent than paralysis of the vocal cords. If there be dysphagia, it is often not an easy matter to determine whether it is due to paralysis or to spasm, which comes on at any attempt to swallow.

3. **Hysterical Contractures.**—Contractures may occur as isolated phenomena, or in combination with other symptoms, such as anæsthesia or paralysis. They are caused by unusually strong tonic muscular contractions. The starting-point of the irritation is certainly in the central nervous system. Sometimes the contractures are temporary, but often they are characterized by their intensity and their great persistency. The extremities suffer most frequently, although the trunk or the back of the neck may be affected. In the hands and feet, especially in the toes, flexor contractures predominate, but in the larger joints extensor contractures are the rule. Although many varieties occur, single forms of contracture are especially characteristic. As an example, we will refer to the accompanying illustrations (Figs. 180, 181).

The relations of hysterical contractures to the articular neuralgias have already been mentioned (page 821). They may occur in hemiplegic, paraplegic, or monoplegic form. They often follow a hysterical convulsive attack (*vide infra*). All hysterical contractures disappear completely in chloroform narcosis, after the use of an Esmarch bandage, and in deep sleep.

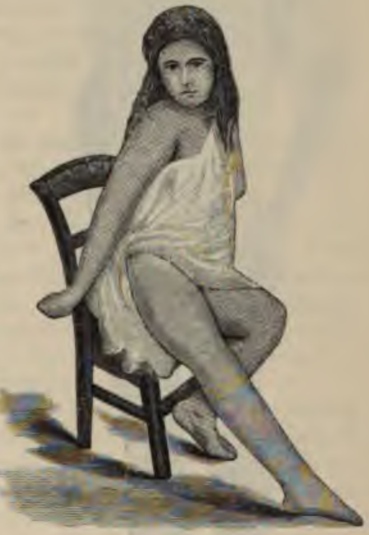

FIG. 180.—Hysterical contracture. (From Bourneville and Regnard's Iconographie de la Salpêtrière).

4. **Vaso-motor Disturbances. Secretory Disturbances. Symptoms in the Internal Organs.**—Besides the sensory and motor symptoms just described, other symptoms occur in hysteria which fall under the domain of the vaso-motor and secretory nerves; but in our opinion we should cite here with positiveness only those symptoms which we find to be dependent directly or indirectly upon psychical processes. In this domain, however, the credulity of many physicians has brought to light a mass of apparently wonderful and inexplicable observations, which are not proof against rigid criticism.

In the first place abnormal anæmia or abnormal fullness of the vessels (a cool, pale skin in the one, a hot, red skin in the other) is not infrequent, and points to vaso-motor influences. Since we know that the vascular nerves are influenced in a high degree by the emotions, we can probably assume a central origin for these symptoms. Hæmorrhages from internal organs are harder to explain; they are apparently not uncommon in hysteria, and they have often been referred to "nervous vaso-motor influences," but in our opinion we must be extremely guarded in such an assumption. We most frequently see hysterical hæmatemesis or hysterical hæmoptysis—that is, the evacuation of blood from the mouth, either with cough or in a way suggestive of choking. Inexperienced physicians have thus often been led to a false belief in dangerous pulmonary disease or gastric ulcer; but if we look more closely we shall find that the blood evacuated is of a clear raspberry-red color, is mixed with a good deal of mucus and saliva, and is of a thin fluid consistency. The whole amount is seldom more than two or three ounces (grammes 50–80). If we inquire more carefully as to its origin, we can usually find a source for the blood in the gums or in the oral or pharyngeal mucous membrane. There are often spasmodic chokings or hiccoughs, by which a hæmorrhage of the mucous membrane is mechanically produced. It is also certain that in many cases intentional deception on the part of the patients may lie at the bottom of the hæmorrhage. We must be extremely cautious in hæmorrhages from the genitals, from the skin, from the palms in the "stigmatized," etc. We once exposed a hysterical woman who had for years produced the most extensive gangrenous spots upon her skin by caustic soda!

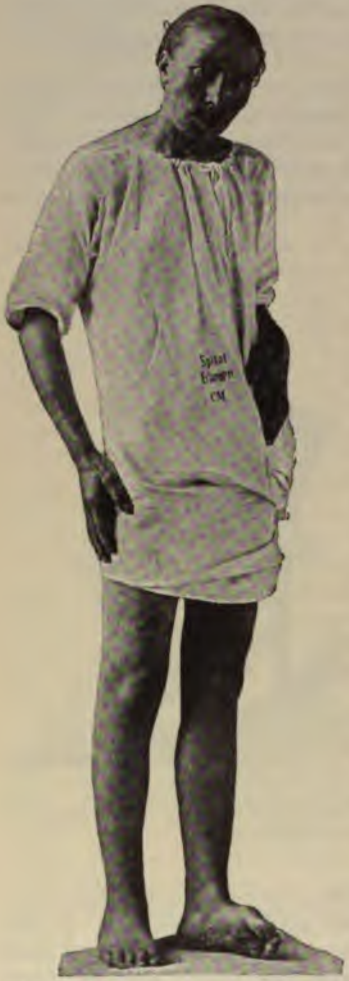

Fig. 181.—Contracted position of the left foot in traumatic hysteria. (Personal observation.)

Finally, among the vaso-motor disturbances we must mention one symptom whose significance, in our opinion, has not yet been satisfactorily explained—we mean hysterical fever. In severe hysteria, especially at the time of severe attacks and psychical disturbances, other observers, and we ourselves, have repeatedly seen high febrile temperatures, 106° (41° C.) and more; and these temperatures come on in a very irregular manner. In none of the cases under our own observation, however, could we wholly exclude the possibility of simulation—that is, of producing a rise of the mercury by rubbing and pressing on the thermometer. In all the measurements we made ourselves in the rectum the temperature was normal, while the high fever was always said to occur in our absence. We therefore recommend the greatest caution with reference to this point.

Anomalies of the secretory and excretory organs have also been met with in hysteria. Many patients have a remarkably dry skin, while others sometimes perspire very freely. The secretion of saliva is subject to similar modifications. Very remarkable observations have been made in a few cases with regard to hysterical ischuria; for days only a very small amount of urine has been passed, although there has been no retention. In one case of this sort, observed by Charcot, there was violent vomiting at the same time, and the vomitus was said to contain a comparatively large amount of urea (vicarious excretion). Such observations, which are very few in number, are at present not susceptible of an exact explanation, but the suspicion of simulation does not seem to us wholly excluded. Hysterical polyuria occurs more frequently than ischuria. A large amount of very light-colored urine of low specific gravity is excreted. This polyuria is in many instances merely the result of excessive ingestion of liquids. Polydipsia (excessive thirst, or probably, to speak more correctly, a morbid tendency constantly to drink water) is a very frequent symptom in hysteria, particularly at the close of a fit.

The digestive disturbances which many patients have are mainly such as have already been discussed on page 463, under the head of "nervous dyspepsia." Colicky pains, obstinate constipation, occasional diarrhœa, and similar symptoms, are by no means rare. Hysterical vomiting, especially, often plays a very great part, since it may very much impair the patient's nutrition. In other cases, of course, the patient's well-nourished appearance is in striking contrast with her statement that she "has to vomit everything up again." Hysterical tympanites also deserves mention. It is due to the accumulation of a large amount of air and gas in the primæ viæ. This may be in part the result of a sort of paralysis ("atony") of the muscular coat of the stomach and intestines, but another frequent cause is the swallowing of large amounts of air. Perhaps the patient does this on purpose. The prominence and tension of the abdominal walls may be so considerable as to simulate grave diseases, such as peritonitis, a tumor, or pregnancy. Doubts of this sort can always be dispelled by inducing anæsthesia with chloroform. It is possible to remove the gas completely in a short time by pressing upon the abdomen, or by introducing a long tube through the rectum.

Sometimes there are symptoms referable to the genital organs. It has been already pointed out that too much prominence was formerly given to the influence of sexual diseases in exciting hysteria. It is also true that nervous derangements of the genital organs, as of all the other organs, may be among the symptoms of hysteria. Many pains and hyperæsthetic conditions of the genital organs must in particular be so interpreted, while co-existing disturbances of menstruation are often to be regarded as co-ordinate symptoms. We can also readily understand that sexual relations often influence very excitable, hysterical individuals to no slight extent, as is indeed very frequently betrayed by the character of their hallucinations and their utterances when delirious.

5. **General Mental and Bodily Constitution of the Hysterical.**—If we regard hysteria as a disease which is largely psychical, which is the only correct view, it will not seem strange if the whole mental constitution and nature of the patient show special peculiarities. In many cases, therefore, the psychical condition of the hysterical is so characteristic that the physician can, from the patient's nature and demeanor, form his conclusions as to the form of her disease.

Hysterical persons are irritable and emotional, easily depressed, sensitive, whimsical, and subject to violent extremes of feeling. They are inclined to exaggerate their sufferings, exact a great deal of attention, and are anxious to excite sympathy. They have little energy or force of will, but they are sly and obstinate in carrying out any pet desire. Again, they can be very amiable and attractive if they take the fancy. They are often shrewd and active mentally, but hysteria is also not infrequently seen in dull and stupid persons.

This brief sketch represents many cases, as we have said, but not all. The patients to whom it refers as a rule present no very great disturbances, but complain merely of all sorts of general derangements, sometimes of one kind and sometimes of another, and yet are able to perform their daily duties tolerably well. A case of paralysis, contracture, or other important localized hysterical trouble may not present any marked mental peculiarities. Often, of course, the patient conceals them from the physician, and the psychopathic nature of the whole mental constitution ("character") is finally revealed only by a closer acquaintance with the patient and her previous life.

With regard to the general physical constitution of hysterical subjects, it has already been mentioned that any bodily weakness favors the development of the disease; and yet hysteria is by no means confined to the ill-nourished, weakly, and anæmic. On the contrary, many patients seem to be in blooming health and well nourished. In a severe case of hysteria, however, the effects of the disease on general nutrition may be very distinct. Little food is ingested, sleep is disturbed, digestion is affected (*vide infra*), and the bodily health is gradually undermined.

HYSTERICAL ATTACKS, CONDITIONS OF SPASM, ETC.

While the symptoms thus far described are largely of a permanent character, paroxysmal nervous symptoms are also very common in hysteria. The diagnosis of the entire morbid condition is often made certain by the onset of such hysterical attacks, which are often very characteristic, and their significance is readily perceived by the experienced physician. There are some cases of hysteria which run their course entirely free from these attacks, and present only certain permanent nervous symptoms, such as paralysis, contractures, anæsthesia, etc.; but there are also other patients in whom the hysterical attacks dominate the whole scene, and are sometimes the only manifestations of the disease. A hysterical attack is not infrequently the beginning of the trouble, especially in those cases where the disease is excited by fright, etc., and where the attack follows immediately upon the psychical excitement.

In regard to the severity and the variety of the hysterical attacks, they are so manifold that an exhaustive account of all the possibilities can not here be given; but certain features and details are so common in them, and recur so often, that a consideration of them is often in itself sufficient for a correct diagnosis.

The mildest form of the hysterical attack consists of the development of a feeling of distress, anxiety, vertigo, and especially of a loss of voluntary control of the body. The patient sinks on a bed or chair, closes the eyes, and becomes incapable of action or speech. Mild symptoms of motor irritation usually ensue, most commonly an acceleration of respiration, general tremor, winking of the eyes, etc. Mild spastic symptoms in the pharyngeal muscles and the diaphragm are not uncommon. There is very often a lively palpitation during the attack. The face is sometimes quite red, but in other cases pale.

In such mild attacks any impartial observer has decidedly the impression that the patient is humoring her inclinations. If, therefore, she be encouraged, or if cold water be thrown in the face or on the back without too great regard for her feelings, she usually soon regains her will-power and rapidly recovers.

In an unbroken series these mildest forms of attack pass over into the severer types, where the clouding of consciousness is greater and the symptoms of motor irritation are more severe. Complete loss of consciousness, so common in epilepsy, is scarcely ever seen in hysterical seizures, but there is very often a marked clouding of consciousness, and in the severe forms morbid alterations of consciousness (*vide infra*). If hysterical attacks occur which resemble those of epilepsy ("hystero-epilepsy"), we must think of the possibility of a combination of the two diseases; but usually the form of the hysterical convulsion is essentially different from that of the epileptic. In severe hysterical convulsions the convulsive movements are more varied, more extensive, and more complicated than in epilepsy. The arms make thrashing and thrusting movements, and often apparently quite well-co-ordinated movements. Patients strike the bed, and sometimes their own bodies, with clenched fists; they seize objects, such as the bedclothes or furniture, and cling fast to them. We also see clonic or tonic flexor and extensor spasms in the legs. The eyes are almost always rolled up, more rarely they converge or are turned to one side, and they often roll about. The lids are tightly closed or occasionally open. There is almost always trismus, and there is often grinding of the teeth. The trunk usually twists and turns the most of any part of the body. The patient often hits her head violently against the wall or the bed. The whole body may assume positions which a healthy person can scarcely imitate without special practice. The best known, and in fact one of the commonest and most characteristic, is the arched position (*arc de cercle*), of which the accompanying illustration (Fig. 182) gives an idea.

Many patients rest for a time on the floor supported only by the head and toes. At intervals they fling or roll their bodies to and fro, drum on the floor with their legs, throw themselves into the air, etc. Any one who has once seen such a " grand hysterical convulsion " will never forget the picture.

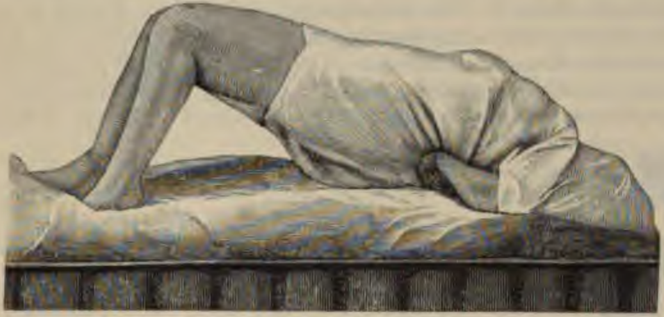

FIG. 182.—Hysterical *arc de cercle* (Bourneville and Regnard).

A different but yet a common form of hysterical convulsion is characterized by a pronounced implication of the respiratory muscles. The attack begins with a spasmodic acceleration of respiration, and the breathing becomes progressively shorter and more rapid. We have ourselves counted two hundred respirations a minute. Other peculiar spasms of the respiratory muscles, which occur in this way only in hysteria, are also common; hiccough, loud sobbing, grunting, etc. The muscles of the pharynx are usually implicated in producing these noises. Of course, all these spasmodic conditions may be joined in the most varied ways with spasms of the trunk and the extremities. We very often find during the hysterical attacks pronounced cataleptic states. If the patient lies in bed with the eyes closed and without marked convulsions, we can put the arms and legs into the strangest positions passively, and they are kept there for a long time (see Fig. 183).

One of the most essential factors which often gives the most characteristic stamp to the grand hysterical attack, is the relation which the spasms often have to certain co-existing abnormal conditions of consciousness. During the attack the patient is usually not unconscious, but she is under the dominion of morbid delusions, and these often mirror themselves in the outward movements. The patient is wholly dominated by a definite circle of ideas; she has hallucinations, and passes through some frightful or exciting event. All this is most strikingly manifested in her movements and her expression. Hence, in an attack the face often expresses terror, rage, threatening, concupiscence, serenity, etc. Very often the internal excitement breaks forth in words, and there is actual hysterical delirium, the most marked emotional utterances, etc. We often observe, and it is a fact of especial interest, that in hysterical attacks which have come on immediately after a severe fright, or some exciting event, the same scene to some degree is rehearsed over in the patient's consciousness in each subsequent attack. If, for example, hysterical attacks come on in a nervous child who has been attacked by a dog, we can not infrequently recognize again the cries of terror at the approach of the dog in each new attack, etc. The patients often talk continually to themselves during the attack, usually very rapidly, with frequent repetitions of the same phrase or word. We can often succeed in giving the delirium a definite direction by questioning the patient, or converse regularly with the patient during an attack, but usually the pure motor spasms again set in, or persist for some time in atonic form.

We can not go further into the numerous details of the symptoms of the grand attack ("*grande hysterie*"), for an accurate knowledge of which we must thank the observations of Charcot and the school of La Salpêtrière in Paris (Bourneville and Regnard, P. Richer). The descriptions of the French authors apply to the grand hysteria not infrequently seen in Germany. In regard to the whole picture of the attack the French neurologists distinguish several periods which correspond in general with the above-described conditions. The first period consists of severe epileptiform convulsions apparently associated with loss of consciousness.

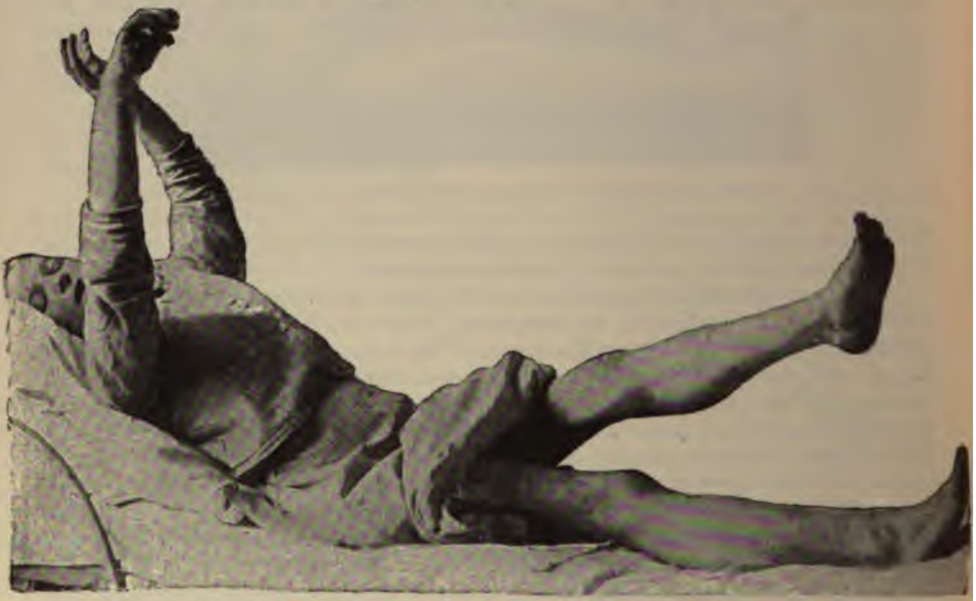

Fig. 183.—Hysterical attack with catalepsy. (Personal observation.)

Then comes the period of "contortions and grand movements" (clownismus), and finally the period of plastic positions and passionate attitudes (*attitudes passionelles*). The short and very significant expressions of this scheme are of practical value, but according to our experience one can only rarely expect sharply defined "periods" in the individual case; the grand hysterical attack is rather composed in the most varied ways of the different symptoms above mentioned.

There remains one essential trait needed to complete the picture of the hysterical attack, and that is the "suggestibility" of the patient, which is so peculiarly characteristic of hysteria. By "suggestion" we mean the artificial production of a definite psychical state, or a physical state dependent on the mind, by arousing the appropriate ideas. In the general characteristics of the mental constitution of many hysterical persons we have already had to point out how much and how often such patients let themselves be dominated by their imaginations. Suggestion is merely the artificial fostering of this psychical peculiarity and its special application. The more we try to foster and preserve this peculiarity, the more we leave uncorrected the false ideas which the patients have, the better we succeed at last in making the patients merely a shuttlecock for their ideas. Hence the physician's daily experience teaches that, as suggestion experiments are repeated, the patients more easily become susceptible to them, so that finally we can actually "do everything with them." There can scarcely be any difference of opinion as to whether such an experiment is entirely justifiable, medically and morally; and even the fostering of suggestion for therapeutic purposes is a two-edged sword whose action is not always rigidly within the physician's control.

Suggestion is most easy during the hysterical attack itself, especially in those forms where the patients speak, hear, and answer. As soon as we give the patients' ideas a definite direction, and tell them in a convincing tone that they are in a garden or a wood, picking flowers or fruit, that they are attacked, bound, lying on the edge of a precipice or the water, etc., we see in their bearing and speech that they think they are actually experiencing all these conditions in their delirium and hallucinations. The emotional utterances of fear, terror, joy, or aversion are then often expressed with astonishing art. In the same way we can suggest paralyses, contractures, or anæsthesias. The most interesting feature of it all is, that when the attack is over every trace of recollection of what happened during it is lost. The same patients who just now were so much excited by some definite idea, a few seconds later, as soon as the attack has ceased of itself or has artificially been brought to an end (*vide infra*), know nothing more about it. They have not even a vanishing recollection like that of a dream about what they have just said and done, even if it be expressly described; but it is still more remarkable that during the following attack they often remember very well what they have experienced actually, or in their imaginations in the past attack. In such cases we can truly speak of "double consciousness." Processes of the conscious waking life, however, often remain in the consciousness during the attack. We have already said that the actual event (scene of terror, etc.) which has given rise to the first onset of the attacks, is often revealed anew in the delirium of subsequent attacks.

Hypnotic symptoms are closely allied to the processes in suggestion. We can not give here a complete review of this field, which, as is well known, has been so much studied of late. The tendency of human nature to mysticism and the influence of suggestions, which may be potent not only with the patients but even with the investigating physicians, are the reasons why the false and the true have often been mixed in the study of hypnosis. We can say only that one great advance is now generally recognized, and that is, that most persons of scientific training have abandoned the hypothesis, once frequently accepted, of a special "magnetic power" (animal magnetism), by which the "magnetizers" could put their "mediums" into the "magnetic sleep" or other abnormal conditions.

Hypnosis is nothing more than the intentional artificial production of a hysterical attack, or a hysterical psychosis by suggestion—that is, by the action of definite ideas on the person to be hypnotized. Therefore, only those persons can be hypnotized in whom these ideas have a strong enough influence. No man can be hypnotized to whom the nature of hypnosis is clear. The essential feature of all hypnotic procedures is merely to produce in the most lively way possible the idea, "It will happen as the hypnotizer says." All other things—the fixation of the eyes on bright objects, the vibrations of a tuning-fork, etc.—are side issues, and serve merely to support the suggestion. In all easily hypnotizable persons the mere closure of the eyes and the remark, "Now go to sleep," are enough to produce the hypnotic sleep. Patients (and here we may actually speak of "patients") reach this suggestibility, of course, only after they have often been hypnotized; for the oftener the same action of an idea is produced the more easily it occurs—a law which follows also from many other experiments in the psychical domain. The different forms of hypnosis are not distinguishable from the various hysterical conditions. Hypnosis is an artificial hysterical symptom, designedly produced, and from this alone we note the danger of all hypnotic experiments if they are practiced by the ignorant. In this, too, it has often been shown that we can not become free again from the spirits which we call up.

The French physicians (Richer) distinguish four chief forms of the hypnotic state, which show, however, many transitions: 1. The cataleptic state, in which the limbs retain all the positions artificially given them (see the previous chapter). 2. The state of "suggestion," of artificially produced hallucinations. If we put the body passively into certain positions corresponding to definite acts, we can produce in the patient all the appropriate ideas with the distinctness of a hallucination. To this category belong the well-known hypnotic exhibitions where hypnotized adult men dance dolls, eat raw potatoes with an expression of enjoyment, etc. 3. The lethargic state, that is, a state of apparent unconsciousness, with the eyes closed, the muscles completely relaxed, and a marked increase of excitability in the muscles and nerves. A light pressure or a slight blow on a nerve, such as the facial, suffices to put all the muscles supplied by it into a tetanic contraction which outlasts the irritation. 4. By certain manipulations (for instance, rubbing over the vertex) we can change the lethargic state into one of hysterical somnambulism. The patients remain half unconscious, but answer automatically questions which are put to them, obey orders given to them, and sometimes show certain sensory hyperæsthesias. We see that all these forms are precisely identical with the different forms of the hysterical attack. Only the increased mechanical excitability of muscles and nerves is not yet fully explained, but the reported observations upon this point have not yet been absolutely confirmed. It is especially a question whether suggestion—that is, ideas which lead to unconscious muscular contractions—may not also play a part here?

We have thus far omitted to dwell upon an important point in the description of the hysterical attack—namely, the relation to it of "hysterogenous zones." We have mentioned above how often in hysterical patients certain parts of the body (the ovarian region, the sides of the chest, etc.) are extremely sensitive to pressure. It is by no means uncommon that, in spite of the patient's resistance, somewhat longer pressure on such a spot may excite a hysterical attack. On the other hand, we sometimes succeed in causing an existing attack to cease by pressure on the same zone. We are of the opinion that in these manipulations also ideas become potent as connecting links; for we may, as we have often demonstrated in the clinique, by suggestive persuasion aloud or by all possible procedures excite and then inhibit attacks in hysterical patients. In such cases we never have to do with physiological reflex processes, but always with the action of ideas suggested to the patient.

Finally, we must add that there are also milder forms of hysterical spasm which are limited to a definite muscular region, and are not associated with any marked clouding of consciousness. There are, for example, isolated spasms of the muscles of the neck, isolated respiratory spasms (spasmodic cough, etc.), not infrequently associated with the strangest noises, and isolated spasms in the arms and legs; the laryngeal muscles may also be affected (hysterical spasm of the glottis). Spasms of the diaphragm and other muscles of inspiration are quite common under the form of hysterical hiccough, which may sometimes last in the severest way for days and weeks. To spasmodic conditions in the muscles of the pharynx and œsophagus we refer the well-known symptom of so-called *globus hystericus*: the patients feel as if a ball were moving up and down in the throat.

We sometimes see spasmodic conditions which come on in a clonic fashion, or in single twitchings in this or that muscular territory; sometimes affect symmetrical groups of muscles; are not associated with disturbances of consciousness; cease during sleep, and are often easily cured by appropriate mental treatment. Such conditions have often been falsely called paramyoclonus, or myoclonia (*q. v*). Among the forms of hysterical spasm we may also reckon the peculiar attacks, essentially of a psychical nature, when more complicated morbid excitations of volition occur with or without the ordinary convulsive movements. In such attacks the patients begin to ejaculate the filthiest words of abuse (*coprolalia*), or to repeat every word they hear (*echolalia*), or to practice peculiar "impulsive" movements (tearing everything to bits, etc.). We see no reason to regard such conditions as a special disease under the name of "*maladie des tics*." We have repeatedly observed such conditions (especially in children) which were undoubtedly to be regarded as hysteria, and which could rapidly be made to disappear by appropriate psychical treatment. The severer forms of these conditions of course belong to the pronounced hysterical psychoses.

General Course of the Disease.—Our description of the symptomatology of hysteria has been confined to the most important and frequent phenomena; and yet even this meager outline shows what an infinite variety of shapes the disease assumes. 1. In one class of cases there are no severe symptoms whatever. The patient merely displays the general mental condition characteristic of hysteria: she is easily excited, prone to make much of her ills, has all sorts of symptoms, such as pain, cephalic symptoms, palpitation, dyspepsia, and dyspnœa, and these are aggravated by mental excitement, while at other times they may so nearly vanish that the patient does not appear to be ill. 2. A second class of cases has more severe disturbance, coming on after some unfavorable psychical or other influence (*vide supra*). The patient may have displayed a general hysterical tendency previously, or may have seemed perfectly well. Here we may observe all the symptoms above enumerated and described. There may be paralysis, spasm, contracture, anæsthesia, or paræsthesia. The individual symptoms may persist obstinately for weeks and months; but again they may vanish suddenly or give place to other disturbances. Psychical influences are unmistakably potent, not merely in the incipient stage but also in the further course of the disease. Any aggravation of the symptoms is often referable to emotional excitement. This is particularly true of the hysterical convulsions. In other cases, of course, the hysterical attacks come on apparently quite spontaneously, just like epileptic states. 3. The third class comprises the most severe forms of hysteria, with those nervous disturbances briefly outlined above. They are as complicated as they are puzzling, and form manifold combinations with all the other hysterical phenomena, including anæsthesia, contracture, and paralysis.

The entire duration of the disease varies greatly. The true root of all evil is the abnormal excitability of the nervous system, which always remains in unstable equilibrium; and often it is not possible to cure this. If not, the trouble lasts almost indefinitely. New manifestations of the disease succeed to periods of apparently perfect health. Usually the symptoms do not abate till quite late in life. There are, however, many instances of complete and permanent relief. This favorable termination is more especially to be hoped for where the patient comes into suitable and appropriate conditions of life, having some regular occupation which is not exposed to all sorts of unfavorable psychical influences. Many cases of hysterical disturbance, in previously healthy children or young adults, and due to some distinct cause, terminate comparatively soon, and never recur. It is never possible, however, to be sure that there will be no relapse, inasmuch as a single appearance of hysteria shows unmistakably that the nervous system is abnormally vulnerable to external impressions and the mental emotions excited by them.

Diagnosis.—An experienced physician is seldom greatly puzzled by hysterical affections. Although the disease may at first simulate some grave organic disorder, a careful physical examination and continued observation will almost invariably disclose the true character of the case. In the first place, there are never any such symptoms as would absolutely prove the existence of some organic lesion. For example, we never find marked atrophy or loss of electrical reaction in connection with hysterical paralysis. We should also regard the whole psychical behavior of the patient, the influence exerted upon her by emotional disturbances, the disappearance of the symptoms when the attention is diverted, and the ætiology of the illness—for instance, the development of the symptoms after some mental excitement or emotion. The discovery of specific hysterical symptoms, the so-called hysterical stigmata, sensory anæsthesia, hysterogenous zones, etc., are especially important. Many symptoms of hysteria, especially hysterical abasia, hysterical aphonia, hysterical hyperæsthesiæ, certain forms of spasm, hemianæsthesia, etc., are in themselves so characteristic that from them alone a correct diagnosis can usually be made at the first glance. The very circumstance that hysterical affections do not keep to the laws of nervous anatomy and nervous physiology, and that they often can not be brought into harmony with them, is of importance in their recognition; but, on the other hand, we must earnestly warn the physician against making the diagnosis of "hysteria" too readily. Pronounced hysterical symptoms are of course only rarely mistaken by an experienced physician, but very often all sorts of symptoms may be due to concealed affections, which can not be positively determined (certain cases of gastric ulcer, gall-stones, renal calculi, beginning cerebral diseases [especially multiple sclerosis.—K.], arteriosclerosis, etc.), and these symptoms may falsely be regarded as "hysterical," especially when we are actually dealing with nervous patients. Repeated careful objective examination and observation is therefore imperatively necessary. In doubtful cases the diagnosis can sometimes be rendered certain only by the result of treatment. In doubtful cases, however, the physician should not proceed too roughly or too rapidly, since he may only too easily cause harm.

Since an investigation of hysterical and epileptic attacks is of especial importance, we will give here a synopsis of the most important distinguishing features.

Epileptic Convulsions	Hysterical Convulsions
1. Sudden fall to the ground; often consequent external injuries.	1. Slow sinking to the ground; no external injuries.
2. Sudden pallor of the face. Often marked cyanosis later.	2. No striking pallor of the face. No marked cyanosis.
3. Pupils dilated; do not react.	3. Pupils react.
4. Convulsions according to the nature of cortical epilepsy in the form of convulsive twitching. Tongue often bitten.	4. Convulsions consist of extensive movements, striking out with the arms or legs, twisting of the body, etc. Respiratory spasms. No biting of the tongue.
5. Often one initial cry, then total loss of consciousness.	5. Hysterical disturbances of consciousness, but no complete loss of consciousness; spasmodic and continuous cries, laughter, weeping, etc. Talking during the attack, emotional disturbances, delirium, etc.
6. Convulsions rarely last longer than a few minutes, followed by deep sleep.	6. Attack may last from half an hour to an hour or more. Patient often wakes suddenly.
7. Suggestive and hypnotic procedures, and also external irritation during the attack, without any influence.	7. The attacks may easily be excited artificially at any time by hypnotic or other suggestive procedures, or they may be inhibited in like manner (pressure on definite parts of the body or the like). During the attack the convulsions may be influenced or even wholly suppressed by sprinkling with water, a cold douche, etc.
8. Other hysterical symptoms are absent.	8. Other hysterical symptoms may co-exist and often remain after the attack (hysterical hemianæsthesia, paralysis, contractures, etc.).

Treatment.—What has been said about the ætiology of hysteria at once suggests a possible method of prophylaxis. A watchful eye will often detect, even in childhood, the signs of abnormal nervous excitability, and in such a case the parent will make it his duty to impose a suitable physical and mental regimen, that graver disturbances may be averted.

If hysteria be already developed, the first and most important treatment is mental. There could be no greater mistake than to deride the patient or treat her as a malingerer; for hysteria is a disease, and its symptoms are just as independent of any conscious volition on the part of the patient as those of any other disease. It is, however, also absolutely essential to carry out the moral training, which the physician must institute with all the proper strictness and energy, because in this way alone can any good be accomplished. Therefore an absolutely positive diagnosis is the first essential in the successful treatment of hysteria. If the physician is in doubt as to the nature of the trouble (*vide supra*), he can not proceed with necessary inflexible consistency, and the therapeutic results are seldom satisfactory; but if the diagnosis is assured, then there is no need of making the slightest concession to the patient. Sometimes this most important indication can be fulfilled only after the patient has been withdrawn from the over-anxious and over-assiduous parents or relatives, and like unfavorable influences. In such cases, treatment in some institution will often be vastly better than the best care at home; and our own experience leads us to recommend most urgently that the eventful necessity of removal to an asylum should be constantly borne in mind with regard to aggravated cases. Often the mere dread of removal to such a place has a favorable mental influence.

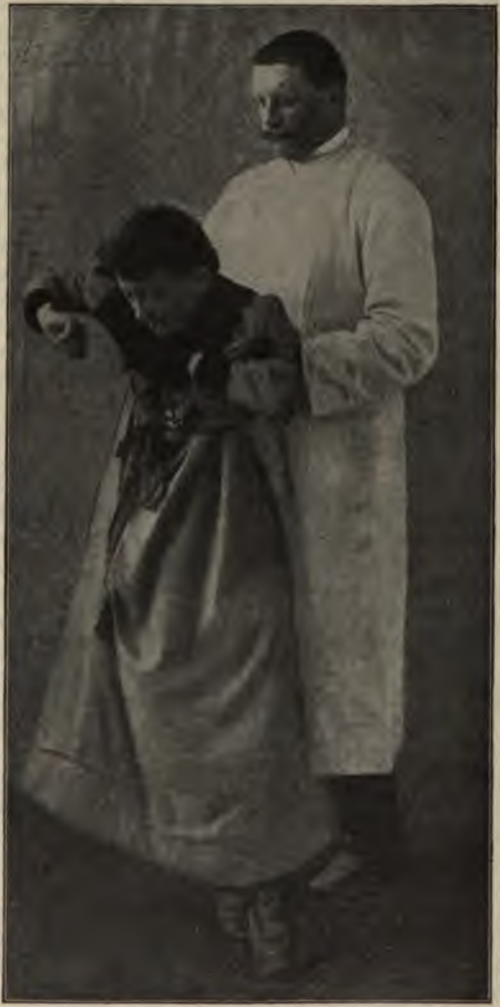

FIG. 184.—Hysterical abasia. Exercise in walking. (Erlangen Medical Clinique.)

Proper moral treatment achieves comparatively the best results where there is hysterical paralysis. When we are once certain that the paralysis is due to hysteria, the patient must be instructed how to regain by practice the lost power of the will over the paralyzed muscles. If the paralysis affect the lower extremities, as it usually does, the patient must be set on her feet, regardless of all her opposition and complaints, and kindly but most firmly required to try to walk.

Of course, at first she must be well supported (see Fig. 184). This exercise must be methodically gone through with several times a day. Gradually the patient's gait becomes more and more secure. She regains confidence in her own ability, and, having once begun to improve, usually makes rapid progress toward complete recovery. Every experienced physician can recall numerous instances where hysterical paralysis which had lasted weeks and months was cured in a few days by this mode of treatment. Faradization of the muscles, cold sponging, with friction and bathing, are excellent adjuvants; and the disagreeable element in these procedures of itself stimulates the patient to make every possible exertion to regain the use of her limbs.

When there is hysterical paralysis of the vocal cords, a similar training (energetic efforts to make the vocal cords tense and to produce a loud tone) will be found both practicable and efficient. Electricity is also of great value. It may be applied externally or within the larynx. Often the patient, terrified by the sudden pain it causes, recovers her voice at once. The same holds true of all hysterical disturbances of speech and of hysterical dumbness or mutism.

The treatment of hysterical contractures consists, first, of an effort to relax the contracture by massage and energetic passive motion. Faradism will be found of assistance here also. In order to remove the contracture permanently, the patient must be induced to exercise the muscles regularly by making voluntary movements. We sometimes have the greatest difficulty in getting the patient to make the first attempt to move. In such cases we can attain our end only by great patience and perseverance and by the suggestive aid of all sorts of manipulations (galvanism through the head, etc.).

The treatment of hysterical spasmodic conditions often causes greater difficulties. The main thing is to bring the patient to control herself at the beginning of the attack, either by an energetic appeal, by diverting the attention, etc. In many cases a sharp sensory irritation, douching with cold water, or a bath with cold affusions, acts to restore the energy of the patient's will and the control over the muscles, and thus to check the spasms. The dread of the repetition of the bath does its part in restraining the patients from giving themselves up unresistingly to a repetition of the attack. The electric current (strong faradism during the attack) may also act favorably in the same way. The action of such measures, however, often gradually weakens, the patients become accustomed to the cold baths, and they remain without effect.

The milder varieties of hysterical convulsion, such as hysterical hiccough or cough, are often controlled by a stern reproof. It is precisely in these cases that the moral effect of transfer to some institution frequently causes the abrupt disappearance of symptoms which have lasted for months. All the forms of hysterical spasm occurring in children especially are easily removed by proper treatment (especially isolation from the family!). The severe hysterical attacks, on the other hand, are often, of course, peculiarly obstinate, and they may resist for months, and even years, the most intelligent treatment. In all hysterical spasms limited to certain muscles methodical gymnastic exercises are often attended by the best results. We inhibit the abnormal motor innervations, and direct the will into other well-regulated paths: regular respiratory exercises in respiratory spasms, gymnastic exercises with the arms for twitching of the arms, etc.

Hysterical anæsthesia is best treated with the faradic wire-brush. The vigorous irritation restores the anæsthetic parts to the domain of consciousness. It should be said, however, that these cases may prove obstinate or relapse.

The most difficult of all hysterical cases to treat are those where the symptoms are not strongly pronounced, but where there is a general hysterical condition, expressing itself in a multitude of nervous derangements, such as palpitation, dyspepsia, and general debility, or in purely subjective symptoms, or in emotional tendencies. Such patients are often advanced in years, so that little is to be hoped for from moral training; and their circumstances may be unfavorable without our being able to remedy the situation. Even here, however, the physician may greatly benefit the patient by means of psychical influences, if he once gains her complete confidence. We should also consider all such remedies as invigorate the nervous system; electricity should be given, either in the form of general faradization, the faradic brush applied to the back and shoulders, or the galvanic current applied to the spinal column and the sympathetic nerve; and of still greater importance is a methodical cold-water treatment, either by sponging, bathing, or douches. Such patients are often vastly improved by sea-bathing in summer, or by going to the mountains.

The numerous internal remedies for hysteria are also of more use in these general conditions than where there are marked nervous disturbances in special parts of the body. In the latter, internal remedies do good only indirectly and in a psychical fashion by so-called suggestion, especially if the patient has great confidence in the virtues of the medicine or in the physician who prescribes it. This is the explanation of the frequent cases of rapid recovery after taking homœopathic and "electro-homœopathic" remedies, and those still more marvelous cures effected by means of holy water and relics.

Among the "anti-hysterical" agents contained in our medical thesaurus, asafœtida, valerian, and castoreum are the most famous; but probably few would at the present day claim that they possess any specific virtues. Perhaps the preparations of valerian are the most useful where there is hysterical excitement, as evinced by convulsions or palpitation. Bromide of potassium, arsenic, and other medicines which ordinarily exert a favorable influence upon the nerves, seldom accomplish any permanent good in hysteria, although often prescribed. We must utter an urgent warning against narcotics, especially morphine, since they do little good and there is great danger of developing the morphine habit in such patients.

If hysteria be complicated by some actual organic disease, the latter, of course, demands special treatment. Great benefit is hoped for by many from the cure of any uterine complaint which may be present. Cases are actually known where grave hysterical disturbance has vanished upon dilatation of a constricted cervical canal or rectification of a displacement; but there are numerous other instances on record where gynæcological treatment has proved entirely unavailing. There is hardly a doubt that, in the successful cases, the main benefit was due to the mental influence of the treatment. In general we would utter an urgent warning against needless gynæcological examinations and treatment in hysterical subjects. The patient's condition is often merely made worse by such procedures.

In general, we may certainly maintain that all methods of treatment of hysteria are efficient only when associated with the necessary psychical factor; that is, the patient's belief in the efficacy of the remedy, or, as we are accustomed to say at present, with *suggestion*. Hence any prescription, no matter how senseless it seems, may have the greatest effect as soon as the patient "believes in it"—that is, as soon as the psychical action of this belief is manifest. Hence it follows that in hysteria, as a rule, treatment either produces rapidly a brilliant success or it has no effect at all.

We must here mention two methods of treatment which have lately been much discussed—metallotherapy and treatment by hypnosis.

In regard to metallotherapy a French physician, named Burq, discovered years ago that by laying plates of metal upon a cutaneous surface affected by hysterical anæsthesia a remarkable result is sometimes produced. Almost at once sensation is restored to the immediate region, and often to a much larger area. Most of the cases have been those of hysterical hemianæsthesia. It is not every kind of metal which will prove effective, nor will the same kind affect all patients. It is said that iron is most frequently efficient; but sometimes copper, zinc, or gold is required. The process of determining the metal essential to each individual case Burq called "metalloscopy"; and he stated that this metal would also have the same effect if given internally! In 1876 a committee appointed by the Paris Société de Biologie tested these statements, at least with regard to the external application of metals—the idea of their internal administration having been pretty much abandoned—and confirmed them. Charcot also discovered many remarkable facts of a similar nature, which likewise soon received universal substantiation. The most remarkable of these phenomena is known as transfer. The return of sensation to the anæsthetic area, as a result of applying a metal plate, is accompanied by a simultaneous development of anæsthesia upon the opposite, previously normal side, in an exactly corresponding place. Sometimes sensation oscillates from one side to the other and back again, so that now one half of the body and now the other is alternately sensitive or anæsthetic. If the metal be placed at the start upon the normal skin, that part becomes anæsthetic, while the corresponding part upon the opposite side of the body regains its former normal condition.

It has also been discovered that other hysterical symptoms exhibit analogous phenomena. Transfer can sometimes be observed in hysterical amblyopia, achromatopsia, deafness, loss of smell and taste, contractures, and paralysis. Such transfers may be induced by various means other than metal plates. These are classed as æsthesiogenous remedies, and include large magnets, feeble galvanic currents, and static electricity. Vibrating tuning forks and sinapisms have also produced similar results. Hence it seems to us to be an indubitable conclusion that the whole group of symptoms just described has no peculiar position. These symptoms are simply the results of suggestion, produced in this case again by ideas. The similarity of the test as performed by the physician produces the similarity of the symptoms that ensue.

Of late the treatment of hysteria by hypnosis has acquired a far greater practical significance than metallotherapy; it is a mode of treatment which has been employed most extensively by the "school of Nancy" (Bernheim) and its adherents. If, during hypnosis, morbid states can be produced by suggestion, it naturally follows that morbid states can also be cured by suggestion. If by many well-known successes the hypnotizing physician has at the outset acquired the patient's confidence, as relics win the confidence of believers, the most beautiful results can of course be attained by hypnosis. There is no special peculiar principle in the hypnotic treatment. Any other efficient mode of treating hysteria rests on the same conditions and presumptions. Hypnosis has only the one great consequence, that it is artificially produced as a severe abnormal mental state in a patient who previously had not spontaneously fallen into this state. In that lies the sequel, which of course need not always be permanent, but which often enough has been most severe: the attempt to hypnotize a patient who is suffering from a slight hysterical affection has not infrequently been followed by the onset of a severe hysterical attack. The mischance will seldom happen to the physicians and magnetizers who practice hypnotism as a specialty, because their psychical influence on their patients is usually greater from the outset; but we should consider it a misfortune if hypnosis should come into too general use. That in this way apparently the most wonderful cures can often be obtained, is perfectly well established and not at all surprising; but the same cures can also be obtained in other ways, without running the risk of producing first that of which you would cure your patient, for to hypnotize means to make temporarily hysterical (see page 1185). Furthermore, it is not hard to prophesy that, as the knowledge of the peculiar nature of hypnosis becomes general, it will lose its halo with the patient, and with that it will lose its healing power.

CHAPTER X

NEURASTHENIA

(*Nervous Debility. Nervousness*)

Concept of the Disease and Ætiology.—The name of neurasthenia is given to a nervous symptom-complex which comes on in a great variety of ways. Its cause is not to be looked for in an organic disease but in a merely functional disturbance of the nervous substance. The nature of this functional disturbance is ordinarily characterized by the two symptoms of "*abnormal irritability*" and "*abnormal exhaustibility*"; but the phenomena of neurasthenia and its allied morbid states are by no means wholly exhausted by the combined concept of "*irritable weakness.*" We may also add that the entire nervous system does not by any means take part in this abnormal functioning. On the contrary, the more carefully we study and analyze the symptoms of neurasthenia, the conviction is forced upon us here, just as in hysteria, that in neurasthenia we have to do essentially only with an abnormal mental condition of the sick person, and that the countless physical complaints and symptoms of the neurasthenic are ultimately to be referred in the main to a disturbed ideational life.

Nervousness and neurasthenia are brief practical terms applied to certain forms of an abnormal mental constitution. In by far the greatest number of all cases this abnormal mental constitution, or at least its basis, is congenital. Neurasthenia of course does not always show itself in childhood, although it does often enough. It does not always develop to the same degree, varying according to the action which various external influences exert in making it more or less prominent. We can only rarely, however, wholly dispense with the ultimate assumption of a definite congenital "nervous" predisposition. Of course we must admit that neurasthenic conditions of exhaustion and irritation may also occur under the influence of persistent mental over-exertion or persistent and severe mental irritation in persons who were previously "perfectly well"; but in general we are usually inclined to attribute to these factors just mentioned merely the *rôle* of exciting causes; for every nervous system does not succumb to the same burden; one endures it without permanent harm, while another breaks down under it.

The congenital factor in the onset of neurasthenia is shown especially by a careful regard of hereditary conditions. Nervousness, neurasthenia, hysteria, degenerative insanity—these are closely associated morbid conditions, at the basis of which lies the inherited abnormal mental constitution, and these conditions are, therefore, unfortunately linked together in many families. Where the laity, from a natural dread of the worst forms, are disposed to believe in a fundamental distinction, the medical eye sees only quantitative differences in a long series of psychical conditions, which, without definite boundaries, join the normal psychical life on the one side and on the other pass into pronounced insanity.

It is our task here to describe merely the mild neurasthenic states, which far exceed in frequency all other nervous diseases. If it has been imagined that neurasthenia is a modern disease, this idea is incorrect. It is as old as our definite knowledge of the diseases of mankind, and if the increasing frequency of neurasthenia is often ascribed to the restless haste, the unrest and the excitement of "modern" life, a glance at the history of past centuries with their horrors and their disorders of war will render such a hypothesis extremely doubtful. The name "neurasthenia" is the only thing that is new or modern. This, and the better insight into the nature of the disease, we owe to the American neurologist

Beard (1880).* It is also true that all the exciting still active in their full force to-day, and that it can n observe neurasthenia with especial frequency in busi lations are attended by the excitements of hope and minds are constantly stirred by the eager strife of par scholars whose never quiet ambition forces them Neurasthenia develops often enough, however, in p the first through a congenital defect, without any can readily understand that a disease, which plays i tional life of those affected by it, should be material of education of its victims; that is, by the richness an life; but it is incorrect to call neurasthenia a disease or of the "higher classes." Neurasthenic conditions uneducated and in the lower classes, although they m somewhat different fashion.

The Symptoms of Neurasthenia. 1. **The Ment** most important changes in the ideational life of the r and ready appearance of ideas of an anxious charac refer to the patient's own corporeity, they have been chondriacal. Sometimes they seem to arise of them are produced by readily recognized causes (reading o medical works, cases of sickness and death in the ideas may constantly arise into consciousness often i form, and then dominate more or less all the rest of t number of neurasthenics are dominated by the thoug or already attacked by a serious organic disease. O pending attack of apoplexy, another heart disease, a t a fourth believes he has noticed positive signs of disea of the stomach, etc. The anxious ideas, however, may sometimes refer to the patient's nearest relatives rath are sometimes tormented by the constant dread that may fall sick. The ideas may also relate to externa anxious ideas are easily and incessantly associated. (anxious ideas on entering a large open space), the dr journey, of society, the fear of sitting under the chan of fire, and many other symptoms of a similar char neurasthenics.

Not infrequently such ideas of an anxious nature pecial severity and lead to actual attacks of anxiou physical symptoms as a consequence. Such attacks n sleep, drive the patient from his bed, and put him unrest and excitement.

We can readily understand that the ease with whi nature arise may keep the patient to some degree in a Hence such patients often complain: "I am always s me—everything, no matter how small, irritates me." are taken up by something else do they become quie seems perfectly normal.

These anxious ideas are by far the most frequent form of morbid ideas which are observed in neurasthe constantly arise in consciousness independently of ex take possession of the patient, so to speak, we give them the general and fit name of *imperative ideas*. The following are some of the especially frequent forms of imperative ideas: Many patients are distressed by the constant recurrence of wholly superfluous questions: Why is this so, why is it not so? What would happen if this or that should ensue? etc. (*Grübelsucht*, metaphysical or reasoning insanity, *folie raisonnante*). Other patients distress themselves with constant doubts, with the constant feeling of uncertainty whether this or that has been done correctly, whether they have not written something wrong, have not forgotten something, etc. (insanity of doubt). [Other patients are impelled to repeat certain words, or to perform acts a certain number of times.—K.] These and other imperative ideas, which occur in a pronounced fashion only in the severe forms of neurasthenia, form the transition to the special forms of neurasthenic insanity.

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* [The disease had previously (1867) been described under t Michigan, and the name itself is found in the first edition of Dungl

Although the morbid ideational life in most cases gives the mental constitution of the neurasthenic the characteristics of excitement and restlessness, there may be also a pronounced inhibition of the rest of the ideational life and consequently a depressed, melancholy disposition. The mental dispositions may sometimes vary in different forms. If there is in general an increased liveliness of the ideational life, the frequent and rapid transition from being "exalted to the clouds" to being "worried to death" may be especially striking.

The worst result of the disturbed ideational life, however, is the disturbance and inhibition of all other trains of ideas by the constant intrusion of the morbid ideas into consciousness. This is the chief reason why the neurasthenic is so often incapable of any persistent, methodical mental work, or at least why he can perform it only with the greatest effort. Sensations of bodily weakness (*vide infra*) are often added to the feeling of mental fatigue, among them being the inability to read or write continuously, sometimes associated with unpleasant subjective sensations of weakness or pressure in the eyes ("nervous asthenopia"). Of course in these disturbances of the power of mental work we must always make a sharp distinction between a congenital, primary, functional weakness and an impediment to mental work due merely to secondary causes.

[The special morbid fears like agoraphobia and the questionings and doubts are rarely met with in neurasthenia, and are usually indicative of a pronounced neuropathic taint of a degenerative type. They may exist without neurasthenia. Anxiety, mental depression, and a tendency to worry are much commoner and are of less serious import. Many neurasthenics, however, show hardly a trace of these mental disturbances, beyond a normal depression over their inability to perform the ordinary duties of life or their failure to regain their health. Their chief symptoms consist of inability to make any long-continued physical or mental effort and of physical weakness. As a result of over-exertion various physical symptoms—headache, insomnia, backache, anorexia, indigestion, etc.—ensue, without any marked mental symptoms. Neurasthenia seems, therefore, to be rather a condition of pathological fatigue, upon which the various mental symptoms above described may develop secondarily.—K.]

2. **The Physical Symptoms.**—Although the mental symptoms of neurasthenia are recognized only by the experienced physician as the special center of the disease, the neurasthenic himself, as a rule, claims to suffer from countless subjective symptoms, which may occur in almost every organ. By far the largest part of all these countless subjective sensations are in our opinion merely the result of the primary disturbance of the ideational life. We admit merely to the second place the possibility that there are independent functional nervous disturbances in other parts as well as the mental anomaly, and that these may lead to special bodily symptoms; but it is evident that most of these subjective symptoms have a psychogenic origin, since they almost always conform absolutely to the character

of the existing hypochondriacal ideas, and since they disappear entirely as soon as we succeed in driving these ideas out of consciousness.

In order to review the commonest neurasthenic symptoms we may very well classify them according to the organs to which the patient's fears generally refer them. The idea of a dreaded organic disease actually provokes the corresponding subjective sensations. Thus the "imaginary" diseases develop. If the patient's thoughts are occupied chiefly with the dreaded advent of a spinal disease, many symptoms apparently spinal ensue, especially pain in the back and unpleasant sensations along the spine, pain and paræsthesiæ in the legs, etc. The more the patient knows about actual diseases by experience or reading, the more manifold become his symptoms. Neurasthenic physicians are therefore the worst patients.—As we speak of a "spinal neurasthenia," so we can also speak of a "cardiac neurasthenia," a "gastric neurasthenia," etc. The cardiac neurasthenics, who live in constant anxiety about heart disease and its consequences, complain of palpitation, pressure, pain and "fluttering" in the cardiac region, vertigo, and distress for breath. Patients with gastric neurasthenia ("nervous dyspepsia," *q. v.*) have a feeling of pain and pressure in the stomach after taking any food, and therefore are afraid to eat anything that is at all indigestible, suffer from unconscious voluntary eructations, etc.

The long series of physical neurasthenic symptoms is by no means exhausted, however, with such subjective symptoms provoked by definite anxious ideas (or by "*auto-suggestion*," as the modern technical term is). In almost all cases a large number of physical symptoms are apparent, which we regard as the necessary (that is, the physiological) result of the general mental irritation. As in every healthy man every great psychical excitement is followed by a number of physical symptoms, it is plain that the abnormal state of excitement of the neurasthenic, coming on without sufficient external cause, may often have somatic results even in increased degree. Among these we may reckon first a large number of so-called "cerebral symptoms," such as headache, vertigo, and pressure and disturbance in the head, to which may of course be added many cerebral symptoms due to auto-suggestion. Among these belongs also the neurasthenic insomnia, one of the commonest and most important symptoms and a direct result of the mental irritation itself. In many cases the insomnia forms the center of the neurasthenic symptom-complex. We may then say that the fear of insomnia is the chief reason for the actual insomnia. Many vaso-motor symptoms are also a direct result of the general psychical irritation—palpitation, a rapid pulse, redness or pallor of the face and hands, increased sweating or persistent coldness of the hands and feet, etc. We may also add tremor and the not infrequent increase of the tendon reflexes. Finally, the loss of appetite which many patients show, the habitual constipation, and in other cases the tendency to diarrhœa, are certainly often most closely related to the psychical processes.

The liveliness of the motor innervations also depends in the main upon the general psychical state. While many neurasthenics, impelled by constant restlessness, exhaust themselves in long and useless walks, there is in others a great physical weakness, which is due to inadequate cerebral volitional innervation of the muscles, so far as it is not dependent upon actual functional abnormality or impaired general nutrition. Such patients are soon tired on walking, they can not work persistently, they sometimes feel so weak that they will scarcely leave their rooms, and they spend most of their time in lying on the bed or the sofa.

It is hard to decide whether neurasthenia exerts a direct influence upon the general bodily nutrition. We find neurasthenia in both full-blooded, corpulent persons and those who are frail and anæmic. Neurasthenia, however, very often exerts an indirect influence upon the patient's nutrition, since loss of appetite or fear of taking food gradually results in marked emaciation. We may mention that large amounts of phosphates are often found in the urine, the reason for which is still uncertain. [In most cases of neurasthenia there is defective metabolism. The urine is scanty, the uric acid is increased relatively to the urea, and in sexual neurasthenia there may be an excess of indican. Many neurasthenics, however, drink very little water.—K.]

We must also devote a few words to a form of neurasthenia which is of great practical importance—sexual neurasthenia. It usually develops in persons who have practiced masturbation in their youth, and, by reading or their own reflection, have fallen into the most exaggerated anxiety as to the supposed evil consequences of this vice. Such pitiable patients usually fear that they are affected past help with some disease of the brain or spinal cord, and suffer accordingly from a host of corresponding subjective symptoms; but the very common "psychical impotence" is still more significant. This is a sexual impotence which is the result of anxious ideas which disturb the sexual act and make it impossible. Other sexual disturbances are usually added to these, which are at least in part a direct result of the physiological hyperexcitation which has occurred in youth (frequent pollutions, urethrorrhœa, etc.). In sexual neurasthenia, therefore, the whole host of general neurasthenic symptoms above described are naturally grouped about this nucleus.

As has been indicated above, we are not justified in regarding all the physical symptoms occurring in neurasthenia as secondary psychogenic symptoms. Since neurasthenia is due to a general constitutional abnormal disposition of the nervous system, it is not strange if functional disturbances in other parts of the nervous system should occur along with the abnormal symptoms in the patient's ideational life. Thus neurasthenia may be associated with true neuralgia, or with migraine or other forms of headache. It is therefore an important and often a difficult task for the physician to analyze the whole aspect of the disease in the individual case, to recognize the purely psychogenous symptoms as such, and to separate from them any co-existing physical nervous symptoms. This distinction is of great importance, especially in regard to the treatment to be advised. [It may also be important to determine how far the neurasthenic condition may be the result of some pre-existing physical condition, which, by the attendant suffering, as of habitual headache, may have led to the general nervous exhaustion.—K.]

General Course and Prognosis of Neurasthenia.—The course of neurasthenia is governed in the main by the degree of general nervous predisposition that exists and the severity of the unfavorable influences that act upon the patient from without. If we are dealing with a patient who was previously in normal mental health and in whom a neurasthenic state of irritation or exhaustion has come on merely as an effect of unusual mental excitement or effort, such conditions of "acute neurasthenia" may wholly or mainly disappear under proper treatment and the removal of the injurious external influences. The condition is different in cases where the neurasthenic tendency has appeared quite early in the patient's whole mental nature. Here also we may often notice, of course, great variations in the prominence of the morbid symptoms; but an actual recovery is scarcely possible, since it would be equivalent to a complete change in the whole mental individuality; but in later life there may sometimes occur, even in such cases, a striking retrogression in the morbid symptoms. In the milder cases there is little outward evidence of derangement; the patient endeavors to hide his troubles, as his indefinite symptoms seldom gain much sympathy, and are apparently contradicted by his well-nourished and healthy appearance. In the severe cases, however, the patient's vigor is so much impaired that the disease acquires a grave aspect even for others than the patient, and fills them, as well as him, with infinite anxiety. The disease is never actually dangerous, and its existence only exceptionally prepares the way for more severe secondary nervous disease. Thus it happens that many neurasthenics, especially when external conditions are favorable, lead a comparatively endurable life and work hard and profitably in their calling.

Diagnosis.—Neurasthenia can usually be detected without difficulty, but the establishment of the diagnosis requires the exclusion of organic lesions of the nervous system. Every case, therefore, must be submitted to a thorough and careful examination. Grave cerebral diseases, such as incipient tumors or general paralysis, have been repeatedly mistaken for neurasthenia. One important point in diagnosis is always the ætiology, including both the outward circumstances and the presence or absence of a constitutional predisposition to nervous diseases.

We can not draw an absolutely sharp boundary, naturally, between neurasthenia, nervousness, and hysteria. In all these three conditions we are dealing, as has been said, with an abnormal nervous constitution, which is congenital or which has at least developed from outward circumstances on the basis of a congenital predisposition. This constitution is manifest in the first place in the purely psychical domain, and, in the second, in the relations between the mental and the corporeal life. If the abnormal state shows itself merely in slight mental irritability, sensitiveness with a tendency to anxious ideas and unfounded fears, and thus to the development of all sorts of subjective abnormal sensations referred, however, to the corporeity, we call it *nervousness*. If there is also associated a marked weakening of the capacity for mental effort and, in consequence of the defective or much inhibited mental energy, for physical effort as well—symptoms which, as a rule, are associated with increased anxiety and with consequent subjective sensations—we have pronounced *neurasthenia*. If, however, the relation between the mental processes and the corporeity has become so vacillating that there is a complete loosening of these relations (paralysis, anæsthesia), or that wholly abnormal states of mental irritation (spasms, attacks) have developed in consequence of the loss of all healthy mental inhibition, we have before us pronounced *hysteria*. The basis of all these three states is—figuratively expressed—the lack of all determined order and fixity in the course of all those processes which we include under the name of psychical processes in the broadest sense of the word.

Treatment.—As in hysteria, so also in neurasthenia, psychical or moral treatment is of prime importance; but here it must be a different kind from that necessary in hysterical cases. The neurasthenic requires sympathy. As soon as we recognize by means of a careful inquiry the hypochondriacal anxiety which is usually present, this is to be removed, as far as possible, by soothing persuasion on the basis of the thorough physical examination which has been made. The neurasthenic must be frequently examined and constantly encouraged and soothed. Herein, especially, is shown the physician's skill and personal influence. Every examination, which ends with the physician's assurance that he has found nothing to give occasion for any serious anxiety, has an extremely soothing and beneficial effect upon the patient. In so far as hypochondriasis is a prominent symptom, this moral influence may alone restore the patient to health. We should also pay attention to the patient's mental life otherwise. If the patient has been exposed to great strain and much excitement, we should of course procure a change. In such cases as these we prescribe mental rest, recreation, and abstention from work or any strenuous activity. Nothing, however, would be more improper than to try to make this prescription universal. On the contrary, it is absolutely necessary to prescribe resumption of regular and methodical work and occupation for many patients who, as a result of their mental disposition, do nothing but give way to their hypochondriacal broodings the whole day long. In this way alone can the thoughts of such patients be diverted, and the patients themselves be gradually brought back to take pleasure in their life and occupation. Of course we must beware of any over-exertion.

Besides the psychical or moral treatment, which must always take the first place, we must consider all the means at our command for "strengthening" the nervous system. In this respect there can be no doubt that the favorable results from physical methods of treatment in neurasthenia are to be referred in great, if not the greatest, part to their indirect psychical ("suggestive") influences. All methods of treatment must therefore be used methodically and continued long enough for the patient to remain for some time under the permanent personal influence of the physician. In this way alone can the patient have that mental schooling which he needs to make him conquer his own disease. What has been said explains why treatment in a sanitarium is so often desirable or even necessary for a neurasthenic.

Certain prescriptions as to diet are of importance in the physical treatment of neurasthenics. These are governed by the needs of the individual case.

For a corpulent patient treatment calculated to diminish obesity will sometimes be followed by decided improvement in the general condition and in bodily vigor. In those frequent instances where the patient is pale and thin, and very likely oppressed by nervous dyspepsia (see page 463), we should, on the other hand, make vigorous efforts to improve nutrition.* Definite instructions must be given in order that the patient may ingest a proper amount of food. Milk, butter, fresh meat, eggs, simple puddings, artificial foods, etc., are appropriate articles of diet. Often both the weight and strength improve rapidly. Any large amount of alcohol or of tobacco should be forbidden. Nothing is worse than to prescribe strong wine for a neurasthenic to "strengthen" him. We have countless times won a patient's gratitude by releasing him from such a prescription, which in his own experience he had found disagreeable (causing heat in the head, vertigo, etc.). Tea and coffee may be taken in moderation, if the patient is accustomed to their use. In regard to bodily exercise, we must again be guided by the condition of the individual. We would most earnestly warn the physician against the error, frequently committed, of driving weakly and debilitated persons to take long walks. For such, bodily rest is much more desirable; and fresh air may be enjoyed at the same time, if the patient sits or lies out of doors or drives. The sluggish and corpulent, on the other hand, often require persistent exercise. It is a good plan in many instances to employ the Swedish movement cure, or similar gymnastic exercises. [In many cases the treatment must be directed primarily toward increasing the nutrition of the patient and building up the general strength in every possible way—forced feeding, cod-liver oil, tonics, etc. To this we must add rest and a change of occupation or at least diversion. These are often more important than the psychical treatment, especially in the cases of pathological fatigue, where the morbid ideas are few or absent.—K.]

Of other other general remedies electricity and hydropathy are at present most in use. Electricity is warmly praised by many patients. The galvanic current is generally employed, and is applied either over the sympathetic nerve or along the spinal cord. Its use demands great caution. The current should not be too strong, and there should be no abrupt changes in it. Galvanism applied to the head is seldom well borne. Another very valuable mode of treatment was first practiced by

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* Playfair, Weir Mitchell, and certain other neurologists, have built up a special "method" of treating neurasthenia and allied conditions of nervous exhaustion; this consists in "over-feeding" the patient—that is, in introducing as large an amount of nourishment as possible into the system at the same time that complete bodily and mental rest is secured. Faradic electricity and massage are also daily employed. This mode of procedure is certainly excellent in many cases, but it must not be regarded as universally applicable. There are cases of neurasthenia for which it is not suitable.

Beard and Rockwell, and consists of general faradization. The patient is almost completely stripped, and places both feet upon a large, flat electrode, while the various parts of the body are stroked with another large sponge electrode; in place of this second electrode the "electrical hand" of the physician may be employed. The physician takes the second electrode in his left hand and allows the current to pass through his own body. Various institutions have lately begun to employ electrical baths; these also often seem to produce good results. In addition to peripheral galvanization and faradization of the nerves and muscles, it is advantageous to employ the faradic wire-brush, particularly on the back of the neck, along the spinal column, and over the shoulders and thighs. It is hard to say whether all these methods of electrical treatment have any specific action. The greatest part of the benefit obtained is undoubtedly of a suggestive nature; but there are few other methods by which such a suggestive effect can be so easily produced as by electricity, and therefore it is hard to dispense with it in the treatment of neurasthenics, in spite of what has just been said, since we often have to change our methods of treatment in so chronic a disease.

The hydro-therapeutic treatment may be quite well carried out at the patient's home, but a severe case will be better off in some well-conducted institution. Cold sponging, douches, hip-baths, lukewarm baths (or swimming), are all employed. Douches must not be applied to the head. If there is sexual disturbance, hip-baths of cold water are advisable. They should not be taken at night. Douching of the genitals and loins is also excellent. We can not here give a detailed description of the different hydro-therapeutic methods. The efficacy of hydrotherapy is due in part to its physical influence on the patient, which is undoubtedly favorable, but also, of course, in large part to suggestion, just as with all other forms of treatment. Subsequently sea-bathing will prove extremely beneficial for many patients. We would recommend the seashore especially for emaciated and anæmic subjects, who are frequently greatly benefited by the improved appetite and rest thus obtained. If the patient be well nourished, on the other hand, a journey on foot through the mountains, if made cautiously, may be very valuable. Massage is also much used as a general means of strengthening in nervous conditions. We can judge of its value from what has been already said. It is prescribed most advantageously in those forms of nervous trouble which are associated with painful sensations in the spine, the extremities, etc. Massage displays an excellent direct effect when there is habitual constipation.

In neurasthenia, internal remedies should be given only as indicated by the symptoms or in the way of suggestion. If there is anæmia, iron, quinine, or Fowler's solution is prescribed; if there is dyspepsia, some stomachic, such as dilute hydrochloric acid, pepsin, or some bitter. The constipation (see page 495) should be overcome mainly by diet, and laxatives should be given only in case of necessity. When there are vaso-motor symptoms (a feeling of heat, congestion, palpitation), we often prescribe ergotine, four to six one-grain (gramme 0.05) pills a day. In all states of nervous irritation bromide preparations are much used, a powder of potassic and sodic bromides, or bromine water. In nervous headaches, and also in other nervous states, antipyrine often has a good effect. Antifebrine and phenacetine, and many similar recent remedies, act in the same way.

The treatment of the insomnia which results from neurasthenia deserves a brief mention. It is usually a difficult problem. Since the insomnia is in most cases merely the result of the mental excitation, especially the onset of anxious ideas, the suggestive action of all the remedies that can be thought of plays of course the chief part, and the physician should take advantage of this circumstance. We must utter an urgent warning against the misuse of the stronger narcotics (chloral and morphine). The attempt should always first be made to secure sleep by a rational general treatment, or by some less injurious remedies. Often a warm bath for half an hour at bedtime soothes the patient and brings him sleep; and in other cases a wet cloth laid upon the head or back of the neck produces the same favorable result. Patients often report that general faradization at bedtime is an excellent soporific. Sometimes a moderate dose of alcohol may be tried as a true hypnotic. A glass of beer at bedtime often has a particularly good effect. If none of these means avail, our next resort should be the bromide of potassium. Very likely small doses of this have only a subjective effect, but there can be no doubt that a large dose, about a drachm (grammes 3–5) in a glass of water, does have a direct tendency to produce sleep. We may also mention as the best of the other hypnotics, sulphonal, twenty to thirty grains (grammes 1.5–2) in a large amount of water, in soup, [hot milk,] or tea, two or three hours before bedtime. Trional is equally to be recommended. These and other remedies, however, seldom give great satisfaction, and we must therefore rely mainly on general treatment.

CHAPTER XI

THE SO-CALLED TRAUMATIC NEUROSES (ACCIDENT NEUROSES)

As an appendix to the last two chapters on hysteria and neurasthenia, we must now—more from practical than from scientific reasons—speak in particular of a series of morbid states which are at any rate most closely related to the above-mentioned affections, but which yet show certain peculiarities. The morbid states in question develop as the result of various sorts of physical injuries (trauma, accidents). They were first noticed after railway accidents (the so-called "railway spine" and "railway brain" of English and American neurologists), and it was formerly the belief that the coarse material concussion of the central nervous system was to be regarded as the cause of the subsequent nervous symptoms. Later the name of "traumatic neurosis" was often used for these morbid states. By this we would imply that the nervous symptoms produced by the trauma do not depend upon coarse material injuries of the nervous system, but upon finer changes not yet made out anatomically. Soon, however, it was shown that in at least the great majority of these cases the material injury itself plays no other part than that of the exciting cause of a purely psychical change in the person affected by the injury. The material trauma is, as we express it, associated with a "psychical trauma." Among these we may reckon not merely the fright associated with the accident but the whole series of new ideas occasioned by the accident and entering most vividly into the consciousness of the injured person—anxiety about the further course of the injury, about the restoration of the capacity to work and earn a living, and also about obtaining any probable damages, etc. Sordid mental motives also are sometimes mixed with the others in consciousness, the desire for indemnification in money, for a better way of living, etc. From what has been said in the two previous chapters we can easily understand that this primary change in the ideational life of the injured person may be followed by a considerable number of apparently physical disturbances.

The type of disease thus arising is peculiarly characteristic. Sometimes the original trauma (fall, blow, etc.) is so severe that directly after it the well-known symptoms of cerebral or spinal concussion (loss of consciousness, general motor paralysis, etc.) may ensue with more or less severity. In other cases, however, the intensity of the trauma or the special seat of its action (fall on the sacrum, back, or side, injury of a limb, etc.) is such that there can be no question of a material cerebral concussion. Not infrequently there may be some organic injuries at first (contusion, hæmorrhage, fracture), but these of themselves are capable of perfect recovery.

The characteristic feature is that after the severe or slight initial material results of the injury have run their full course, there is not a complete cure, as might have been expected. The patient recovers, tries to get about again, and the improvement goes on up to a certain point; but, nevertheless, a number of symptoms remain which do not disappear, and which diminish or wholly destroy the victim's capacity for work. If we now examine such patients we find no signs of material injury either in the nervous system or in any other internal organ. What at once strikes us is a peculiar psychical change. The patients are dull, depressed, disinclined to any occupation, wholly without energy, and they have no more pleasure in intercourse with their families and friends. They brood constantly over their accident and its results. In very many cases the gloomy and listless disposition is mirrored in the patient's whole appearance, and especially in his facial expression. A practiced observer can often recognize the genuine "accident case" at the first glance (see Fig. 185). The sleep is disturbed and often rendered uneasy by dreams. Of subjective symptoms we must mention especially pains in the part which was hurt; these are commonest in the back, the sacral region, the sides of the chest, the hips, etc. Patients also complain of headache, vertigo, dullness, dim vision, tinnitus, spots before the eyes, loss of appetite, etc. Objective examination often shows a general motor weakness. Many patients can walk quite well alone, but they are soon fatigued; others walk only slowly and stiffly with support, complaining of pain in the back on walking, and therefore holding the hand on the back, etc. The nutrition of the muscles is usually good. The electrical excitability of nerves and muscles is completely normal. The reflexes are sometimes lively and sometimes weak, but never so much changed as we find them in actual organic diseases. Examination of the sensibility may reveal very marked changes, which do not occur in all cases. This examination must always extend to all the sensory organs. The skin over almost the entire body is often very insensitive to painful irritations, pin-pricks, the electric current, etc. There is analgesia. In some places we find complete anæs-

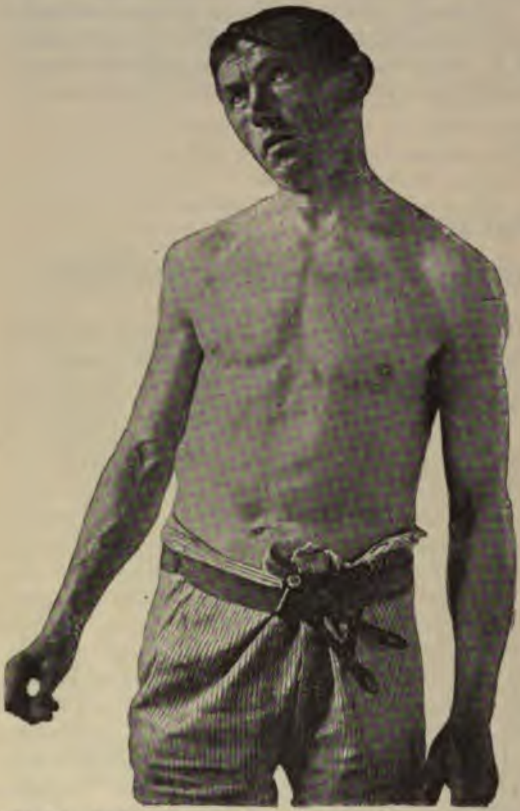

FIG. 185.—Accident case, with hysterical paresis and tremor of the right arm. (Personal observation.)

thesia, which may affect an entire extremity or only circumscribed parts of the extremities and the trunk. The boundary between the anæsthetic and the normally sensitive parts is indistinct or quite sharply defined, and the arrangement is often very peculiar, in a way that can never occur in organic nervous diseases. Examination of the eyes sometimes shows loss of visual acuteness, limitation of the visual field, and imperfect color perception. Very often the hearing is poor on one or both sides. The smell is often much blunted, and the taste completely lost, so that even quinine, vinegar, and substances of equally strong taste no longer excite any sensation of taste.

Besides the symptoms of impairment of sensation and motion just described, we often find also symptoms of sensory and motor irritation. Pain in the head, spots before the eyes, and tinnitus have been mentioned above. Hyperæsthesia of the skin is more characteristic—tenderness of the vertebral column, and particularly great tenderness of such parts of the body as were most affected by the original injury. Among the symptoms of motor irritation we may mention muscular contraction and muscular rigidity, again most common in the limbs most affected by the injury. A marked tremor is also very common.

Almost always the subjective symptoms are referred definitely to the spot which was first injured. If the injury has affected the head, the chief complaint is there; if one side of the trunk or one limb has been affected, most of the symptoms are manifested there; but of course the symptoms often extend beyond the part originally injured. If, for example, one shoulder receives a blow, the whole side, including the leg, etc., is often weak and painful. General nervous symptoms, especially the subjective feelings of vertigo and pressure in the head, are found in the majority of cases, even after peripheral injuries.

If we ask after the cause and the special nature of this peculiar affection, characterized chiefly by psychical disturbances and in the second place by subjective sensations of pain, sensory anæsthesia, and motor weakness, the purely material effects of the injury are in most cases not to be considered, as we have said. Although we at present believe that there are morbid states which we must actually regard as traumatic neuroses in the stricter sense of the word, and as permanent functional disturbances resulting from an actual material cerebral concussion, the assumption does not hold for the vast majority of all "traumatic neuroses," for the neurosis is certainly produced by the *ensemble* of psychical effects, associated with the trauma (accident), which have been described in detail above. The perplexity and frequent contentions about sick funds, insurance societies, and claims for damages, do their part in keeping the patient disturbed.

In laborers, who have accident insurance, and in whom the "traumatic neuroses" are far most frequently observed, we often find a peculiar obstinacy in their claims and their opinions in regard to a presumed right of damages after every accident. We may therefore assert that the traumatic neuroses have become decidedly more common as a result of the accident legislation.

[In America claims for "damages" against corporations are much more important factors in the ætiology of the traumatic neuroses than any claim for insurance. The various psychical influences associated with the litigation consequent upon such claims have given rise to the term "litigation neuroses." Although it is undoubtedly true that litigation aggravates, and sometimes perhaps originates, nervous conditions, it is equally true that we often see precisely similar conditions developing after an accident in which there was no possibility of making any claim for damages or insurance, and where the other psychical factors above referred to had only a slight influence. Furthermore, on considering a large number of cases, there seems to be some relation between the severity of the physical injury and the severity of the subsequent nervous conditions, even in the cases of genuine hysteria, although hysteria may at times develop after a very slight physical injury. The pain and other physical effects of the injury may, of course, if long continued, so far affect the general condition as greatly to aggravate the nervous sequelæ, but the influence of the actual material concussion is often under-estimated, and the " accident case " often receives less consideration than he really deserves.—K.]

Various factors therefore act in producing a condition which has the closest points of contact with general nervousness, neurasthenia, hypochondria, and hysteria. It would therefore be proper, now that our views as to the nature of the traumatic neuroses—a term, by the way, which we have always used merely in a symptomatic significance, and therefore justifiably—have become clear, to use this term merely as a symptomatic appellative and replace it by the terms " traumatic neurasthenia," " traumatic hysteria," etc. On the other hand, we must not forget that the neurasthenic and hysterical affections following accidents often have a certain peculiar stamp, a common type which we do not often find in ordinary hysteria and neurasthenia. The chief characteristics are the patient's peculiar psychical state, manifestly conditioned by the special external circumstances, his lack of courage and energy, his depression, the tendency to complain and be querulous, and also the great obstinacy with which these conditions persist and often resist any attempts at treatment. We know many cases where such patients, in spite of the fulfillment of every desire, have lived for years in a state of permanent neurasthenia, incapable of anything, and finally have fallen into a state of pronounced mental dullness. We may therefore still use the name " traumatic neurosis," or, better, " accident neurosis " or " accident psychosis," for such cases from practical reasons, if we only associate the correct meaning with it. In all those cases, however, where there are pronounced objective symptoms of hysteria (anæsthesia, paralysis, contractures at the seat of the injury), we should speak merely of hysteria, with the appropriate adjective, " traumatic " hysteria.

In the great majority of cases it is not at all difficult to recognize positively the " traumatic neuroses." The existence of gross material injuries can, as a rule, be easily excluded. It may be harder to decide whether there is real disease or simulation. In such cases we can not decide by single criteria—as has been supposed—but ultimately only by careful observation of the constitutional state (if possible, in a hospital) and by consideration of the general conditions (the patient's behavior, previous life, external circumstances, etc.). From our own experience we must in general state that conscious and intentional complete simulation is not very common, although, of course, it occurs often enough. Exaggeration and apparent simulation are of course very common, since the patients report symptoms, which careful investigation shows to be absent or at least not to be constant. Such a condition, however, is no proof of conscious simulation, since we see it every day in all the psycho-neuroses (hysteria, neurasthenia). A hysterical woman who claims that she can not leave her bed, and then walks very well when led by the physician; another who says she can not bear the slightest touch on her back, and then hardly feels the strongest pressure when her attention is diverted; a third who claims to have pain in the stomach after a swallow of tea, and soon after eats a beefsteak with relish—would not be called a " simulator " by any physician. A similar thing occurs of course in the accident neuroses, because they are precisely analogous conditions. It is of course very hard to decide the question, which is often of such practical importance, how far such neurasthenic-hypochondriacal patients have a claim for damages and pecuniary assistance. In such cases we are decidedly of the opinion that the examining physician should not be too generous, since it is necessary from a therapeutic point of view to cure the patient of his delusion that he has a serious acquired disease. We should therefore always try in the first place to bring about an amelioration or a recovery from the condition, and of course the chief stress is to be laid upon the proper psychical influence to be exerted on the patient; but we must also consider all the other methods of treatment (baths, sponging, electricity, gymnastics), with which we have become acquainted in the two preceding chapters. In this way we sometimes succeed undoubtedly in bringing about improvement and even recovery, but the outlook for the permanent success of the treatment is not in our experience very good. The accident neuroses apparently have an especially unfavorable prognosis, although, as we know, the same is unfortunately often enough the case in non-traumatic states of severe neurasthenia and hypochondria. In the accident cases, however, we often notice from the outset that they do not wish to be treated or cured. The incurability of their condition has become a matter of conviction to them, and their only interest is to obtain as large an accident annuity as possible. In most cases, at least, we should therefore advise against any continuous medical treatment, unless we have the impression that the patient will submit willingly and trustingly to the physician's directions. It seems to us of the greatest practical importance to "strangle" the traumatic neuroses "in the embryo," if possible, on their first development. Not infrequently we have seen accident cases who began to complain constantly of all possible nervous troubles after some slight external injury was evidently wholly cured. In such cases we should cut short such complaints as completely as possible from the outset and make the patient understand that he is now actually and completely well. The granting of a high accident annuity at first is therefore quite incorrect in such cases and makes recovery much harder, while the immediate and complete refusal is of the best influence. The patients are forced to resume their work and to forget their imagined symptoms. If a traumatic neurasthenia has continued for years, it is usually very hard to convince the patient that his complaints have no objective foundation.

[In many cases the patient is actually injured by resuming work too early. It is sometimes of decided advantage, especially in the early stages of the trouble, to prescribe a thorough "rest cure," with isolation, massage, forced feeding, etc., as prescribed by Weir Mitchell. The worries of litigation should be eliminated, if possible, by an early settlement of the case. It must be borne in mind that the various therapeutic procedures, which are often advisable in neurasthenic and hysterical conditions, such as massage, electricity, gymnastics, good nursing, travel, etc., are extremely expensive and often impracticable for those in poor circumstances. This is of importance when the physician is called upon to determine the amount of the damages or the accident annuity.—K.]

APPENDIX I

SUMMARY OF THE SYMPTOMS AND TREATMENT IN CASES OF POISONING

1. **Sulphuric Acid.**—Mucous membrane of mouth, throat, œsophagus, and stomach deeply corroded. In the worst cases rapid death ushered in by convulsions and asphyxia, or more rarely consequent upon perforation of the stomach. Usually the case is more protracted. The mouth and throat are whitened, or in severe cases blackened. They are soon attacked by an intense ulcerative inflammation. Deglutition is extremely painful, and there are most distressing choking and retching. The vomitus contains black lumps. Profuse salivation. Pain along the œsophagus. Abdomen usually distended and very tender on pressure. There may be intestinal discharges of a bloody character, resembling dysentery. Urine is generally scanty, and often contains albumen and blood. Collapse. Small and rapid pulse.

In mild cases, slow recovery, the necrosed tissues gradually sloughing off. Cicatricial stricture of the œsophagus frequently ensues and it may prove fatal. Neuralgia, hyperæsthesia, and various other nervous disturbances may also occur as sequelæ.

In fatal cases, the autopsy discloses necrosis, ulceration, and inflammation in the upper portion of the digestive tract. The lining of the stomach is usually coal-black. Well-marked parenchymatous degeneration of the liver and kidneys. Perhaps nephritis. In later stages, extensive cicatrices.

Treatment: If used at all, the stomach-pump must be introduced very cautiously, for fear of causing perforation. The best remedy to give at once is several teaspoonfuls of magnesia in water, or a few drops of liquor sodæ in mucilage. Later, the symptoms are to be combated with bits of ice, disinfecting mouth-washes and gargles, tonics, and cautious feeding with milk, eggs, etc. If stricture of the œsophagus develops afterward, an endeavor should be made to dilate it with bougies.

2. **Hydrochloric and Nitric Acids.**—Symptoms similar to those of sulphuric acid. The most prominent symptoms are the local ones, intense stomatitis, pharyngitis, etc. There is usually albuminuria, with casts and blood in the urine. Nitric acid sometimes stains the corroded spots, especially the angles of the mouth, yellowish; and the vomitus may have the same tinge (xanthoproteic acid). In poisoning from fuming nitric acid the inhaled vapors cause pulmonary symptoms. Prognosis and treatment as in case of sulphuric acid.

3. **Nitrous and Sulphurous Acid Fumes.**—Intense local inflammation of the air-passages. Violent dyspnœa, cough, abundant bloody or yellowish expectoration. There may also be grave nervous disturbance and collapse. ***Treatment:*** Symptomatic: sinapisms, narcotics, expectorants, and inhalations.

4. **Oxalic Acid.**—Local corrosive action similar to that of the other acids, only less severe. In severe cases collapse from cardiac paralysis. Apt to occasion certain nervous symptoms—formication, anæsthesia of the finger-tips, tonic and

clonic convulsions, trismus, and tetanus; later paresis. There is sometimes sugar in the urine, and nephritis may develop. Anuria is frequent, and is due to plugging of the tubules by calcic oxalate crystals. The treatment should be symptomatic, and should also include the administration of preparations of calcium—liquor calcis, calcii carbonas, or even egg-shells—to form insoluble calcic oxalate. Magnesia is also useful.

5. **Ammonia.**—The fumes affect the air-passages chiefly; the solution, the upper part of the digestive tract. The specific local effect is the production of an intense croupous inflammation of the mucous membrane. Accordingly, the symptoms are salivation, dysphagia, vomiting of strongly alkaline matter, and diarrhœa, or cough, dyspnœa, etc. In severe cases there is collapse, with rapid pulse, and such nervous symptoms as pain, paræsthesia, vertigo, convulsions, etc. *Treatment:* In fresh cases the stomach-pump. The cautious use of acids—for instance, acetic or citric. Also, symptomatically, emulsions of oil, bits of ice, and narcotics.

6. **Caustic Potash or Soda.**—Symptoms and treatment as in case of ammonia. In contrast to acid poisoning, it is to be noted that alkalies do not withdraw water and precipitate albumen, but dissolve it. The corroded spots are therefore not dry and brittle, but softened ("colliquation").

7. **Potassic Nitrate.**—Vomiting and diarrhœa. Severe abdominal pain. Collapse, with cold skin and rapid, thready pulse. Occasionally the pulse is slow. Nervous disturbances, such as painful muscular contractions, and, in severe cases, convulsions and coma. *Treatment:* Symptomatic; opium and other narcotics, stimulants (camphor, ether), and bits of ice.

8. **Chlorine Gas.**—Violent convulsive cough. Bloody expectoration. Spasm of the glottis. Dyspnœa. Darting pains through the chest. Sneezing and profuse flow of tears. In severe cases pneumonia. *Treatment:* Fresh air. Inhalation of warm aqueous vapor, or of ammonia to form ammonic chloride. Chloroform may also be tried, and narcotics.

9. **Iodine.**—1. *Acute iodism,* as seen, for instance, after the injection of large amounts of tincture of iodine into ovarian cysts: collapse, with pallor and cyanosis, and small and very rapid pulse. Vomiting. Often marked dyspnœa. Suppression of urine. Sometimes hæmoglobinuria. Later, the skin becomes very red. There is albuminuria; also sore throat, coryza, conjunctivitis, severe frontal headache, and cutaneous eruptions. 2. *Chronic iodism,* caused, for example, by long-continued internal administration of potassic iodide: coryza, conjunctivitis, sore throat. Gastric symptoms. Vertigo, headache, and similar nervous phenomena of a mild character. Acne or erythema, sometimes in the form of erythema nodosum. *Treatment:* In acute cases, white of egg and stimulants. Other than this, treatment must be symptomatic. Prophylaxis demands caution in the internal administration of iodine or its compounds. According to Ehrlich, the symptoms of iodism often disappear rapidly after the exhibition of about a drachm and a half (grammes 6) of sulpho-nitro-salicylic acid (sulphanilic acid).

10. **Bromine.**—1. *Acute poisoning* from the fumes of bromine excites the same symptoms as does chlorine gas. 2. *Bromism,* resulting from the long-continued use of potassic bromide, causes languor, debility, mental apathy, and impaired intellectual vigor. The reflexes are diminished, particularly the reflex irritability of the soft palate and pharynx. Anorexia. Diarrhœa. Impotence. Almost invariably a characteristic acne, the onset of which may be delayed by giving arsenic at the same time.

11. **Lead.**—(*a*) *Acute lead poisoning* produces severe gastro-enteritis. The best antidote is sulphate of sodium or magnesium; or phosphates, white of egg, and milk. In fresh cases the stomach-pump, or emetics and purgatives. Other than this, symptomatic treatment.

(*b*) *Chronic lead poisoning:* Seen in type-setters, type-founders, painters, potters, and others. General symptoms: Lead line on the gums, lead anæmia, and lead cachexia. Important groups of symptoms are: 1. *Lead colic:* Violent colicky pains, radiating from the umbilicus. Usually constipation, exceptionally diarrhœa. Abdomen concave and hard. Vomiting. Hard, slow pulse. Temperature usually normal. Urine sometimes contains a trace of albumen. Duration, one or two weeks. *Treatment:* If severe pain, opium and hot compresses. Atropine may also be tried. For constipation, enemata and gentle laxatives. Warm baths. 2. *Lead paralysis* (*vide* page 873): Paralysis of the laryngeal muscles due to lead poisoning has been reported. 3. *Saturnine encephalopathy:* Sudden development of grave cerebral symptoms; convulsions, coma, delirium, great mental uneasiness, and excruciating headache. Saturnine amaurosis. In severe cases, death. Cerebral lesions are very rarely found post mortem. *Treatment* is symptomatic. Lukewarm baths, with douching, narcotics, and stimulants. Later, potassic iodide. 4. *Lead arthralgia:* Most frequently attacks the knee. Also seen in the joints of the upper extremities. Sometimes associated with painful muscular contractions. Objective lesions are rarely seen. The treatment consists of warm baths and the administration of potassic iodide. It should be remembered that chronic lead poisoning may occasion gout and chronic interstitial nephritis. The reader is referred to the chapter describing these diseases.

12. **Copper** (*Blue Vitriol, Verdigris*).—(*a*) *Acute copper poisoning:* Copper taste. Vomiting of greenish matter, colic, tenesmus, and bloody stools. Also nervous symptoms, headache, vertigo, anæsthesia, paralysis, delirium. Collapse. Dyspnœa. *Treatment:* White of egg, milk, wood charcoal. Magnesia is also valuable. Yellow potassic ferrocyanide, milk sugar. (*b*) *Chronic copper poisoning* is rare. It occasions gastro-intestinal disturbance, colic, and a reddish or greenish discoloration of the hair.

13. **Zinc** (*Zinc Sulphate and Chloride*).—In acute poisoning, symptoms of severe gastro-enteritis, especially vomiting. Also albuminuria. *Treatment:* Albumen, tannin, and alkaline carbonates. *Chronic poisoning* (zinc fumes!): Fever, distress, vertigo, vomiting, metallic taste. General anæmia and emaciation.

14. **Mercury.**—(*a*) *Acute poisoning from corrosive sublimate:* The mucous membrane of the mouth, throat, œsophagus, stomach, and intestines is deeply corroded. Metallic taste. Vomiting. Diarrhœa with painful tenesmus. Ischuria or complete anuria. Collapse. Generally quickly fatal. *Treatment:* Milk, white of egg, reduced iron, narcotics. (*b*) *Chronic mercurial poisoning:* Seen in the makers of thermometers, scientific instruments, and mirrors. Rarely occasioned by the prolonged use of antisyphilitic remedies. The symptoms are anæmia, emaciation, with great muscular weakness, and gastro-intestinal disturbance. In the therapeutic use of mercury, besides the rarer mercurial enteritis, mercurial stomatitis is the most important toxic symptom; onset of foul-smelling necrosis of mucous membrane, with ulceration of mucous membrane of cheeks, jaws, gums, etc. *Treatment:* Immediate discontinuance of medication; potassic chlorate as mouth-wash and gargle.

Mercurial Tremor.—Tremor is common in chronic mercurial poisoning and is of special interest. It is usually preceded by a stage of pronounced mental irritability ("mercurial erethism"), and often some mental excitement, fright, etc., is the exciting cause of the tremor. The tremor itself is a pronounced intention tremor—that is, it is usually not present when the body is kept quiet, but it comes on with all movements, like the tremor in multiple sclerosis. The finer the movements the patient tries to make the more marked is the tremor. Mental excitement usually increases the tremor considerably. In severe cases the tremor is so great that the patients can not leave their beds. In rare cases there is also paralysis.

Treatment: Dietetic and hygienic. Iodide of potassium, hyoscine, and sometimes galvanism, are also useful.

15. **Phosphorus.**—(1) *Acute phosphorus poisoning*, as from matches: Violent epigastric pain, vomiting—the vomitus smells of phosphorus, and it may be phosphorescent. After these initial symptoms usually comes a period of comparative comfort, lasting two or three days. Then appear grave symptoms: jaundice, severe pain in the hepatic region and whole abdomen, hepatic enlargement, fever, feeble and rapid pulse, sometimes gastric or intestinal hæmorrhage, cutaneous ecchymoses, hæmaturia, epistaxis, or metrorrhagia. The intellect usually remains clear, except that just before death there may be drowsiness or convulsions. The urine contains albumen, blood, casts, bile-pigment, and sometimes leucine and tyrosine. No urea is excreted. Death occurs usually at the end of one or two weeks, but is may be more speedy. In mild cases the above symptoms are not strongly marked, and recovery may ensue. A very grave prognosis should be made in every case at the start. *Post-mortem appearances in acute phosphorus poisoning:* Jaundice. Dark blood. Numerous internal ecchymoses—for example, in the serous and mucous membranes and kidneys. Fatty degeneration of most of the internal organs, including the liver, heart, muscles, and kidneys. *Treatment:* In fresh cases, washing out of the stomach. Laxatives. As an emetic, sulphate of copper. The best antidote is old oil of turpentine (30–40 drops in mucilage). Oily substances should be avoided, as phosphorus is soluble in oil. Narcotics and other symptomatic remedies may also be indicated. (2) *Chronic phosphorus poisoning:* Necrosis of the lower jaw, less often of the upper jaw, extending from caries of the teeth. Necrosis of the bone with exuberant growth of osteophytes.

16. **Arsenic** (Arsenious Acid, Schweinfurth Green, Scheele's Green, Arsenical Wall-paper).—(1) *Acute arsenic poisoning:* Symptoms of intense gastro-enteritis, suggesting cholera. Violent vomiting. Rice-water stools. Severe abdominal pain. Nervous symptoms: Vertigo, headache, faintness, twitchings. Cardiac weakness. Cyanosis. Collapse. Not infrequently a cutaneous eruption resembling urticaria or eczema. Sometimes albumen and blood are present in the urine. Severe cases are fatal in one or two days. With regard to arsenical paralysis, *vide* page 875. *Treatment:* At first the stomach-pump or emetics—for example, sulphate of zinc. The best antidotes are freshly prepared ferric hydrate in water, two to four tablespoonfuls every fifteen to thirty minutes; ferrum oxydatum saccharatum solubile (P. G.), in teaspoonful doses; magnesia; and, best of all, the compound of magnesia and ferric hydrate known as "*antidotum arsenici*" (P. G.), of which a tablespoonful may be given every fifteen to thirty minutes. (2) *Chronic arsenic poisoning:* Acquired in arsenic works and glass factories, or from arsenical fabrics, papers, and flowers. Conjunctivitis, chronic gastro-intestinal catarrh, eczema, and cutaneous ulcerations. Anæmia and cachexia, falling out of the hair, loss of sleep. Treatment is purely symptomatic, except as regards prophylaxis. In poisoning with arseniuretted hydrogen, hæmoglobinuria and jaundice occur, with severe nervous symptoms.

17. **Alcohol.**—(1) *Acute alcoholic poisoning:* Unconsciousness; anæsthesia; pupils either dilated or contracted, usually not reacting to light; pulse small, sometimes slow; skin cold and clammy; vomiting; stertorous respiration. There may be delirium and clonic convulsions instead of coma. Such cases last three or four days. Death has been repeatedly observed. *Treatment:* Bathing and douching. Stimulants.

(2) *Chronic alcoholism.* (*a*) *Physical and mental debility:* Chronic catarrh of the throat, larynx, stomach, and intestines. Alcoholic tremor. Numerous

organic diseases, including cirrhosis of the liver, contracted kidney, heart-disease, cerebral disease, and neuritis.

(*b*) *Delirium tremens:* Usually a sudden onset, as in connection with some acute disease or after a surgical injury. Disordered intellect. Great restlessness, hallucination (vermin, etc.), excitement, and loss of sleep. *Treatment:* Bathing and douching. Injections of strychnine. The use of chloral and other narcotics should be cautious. Physical restraint should be avoided if possible. The patient may often be allowed to go about the room as he likes, if only he be watched. Alcohol should be given if collapse be threatened.

[(*c*) *Alcoholic insanity:* Marked mental confusion with loss of memory of recent events, failure to appreciate time or place, occasional hallucinations. In some cases marked depression or excitement, in other cases more systematized delusions, of persecution, marital infidelity, etc. In more severe cases pronounced mental failure. Conditions of physical weakness often associated (neuritis? *vide supra*, polyneuritic psychoses, page 894). *Treatment:* Abstinence from alcohol, strychnine, forced feeding, free elimination.—K.]

18. **Chloroform.**—Unconsciousness. Loss of sensibility and reflexes. Slow pulse. Pupils dilated. Failure of respiration, and finally of the heart. Danger of heart failure, especially in persons with weak hearts. *Treatment:* Artificial respiration. Injections of strychnine. Stimulants. Counter-irritation.

19. **Iodoform.**—(Repeatedly seen from the use of iodoform on wounds.) First of all, nervous symptoms: headache, vertigo, sleeplessness. Peculiar psychoses, maniacal attacks, delusions of persecution, refusal of food. In severe cases convulsions, deep coma. Very small, rapid pulse. *Treatment:* Symptomatic, by stimulants, baths, etc. Alkalies and atropine are recommended, but their action is doubtful.

20. **Carbonic-oxide Gas** (Illuminating Gas).—At first, vertigo, headache, throbbing in the temples, ringing in the ears, and spots before the eyes. The patient gradually becomes unconscious. Skin pale and cyanotic. Respiration intermittent. Subnormal temperature. The urine may contain albumen and sugar. The carbonic oxide may be demonstrated in the blood by means of the spectroscope. Its color is clear cherry-red (CO-hæmoglobine). Subsequently paralysis, disturbances of sensation and of speech. *Treatment:* Fresh air, artificial respiration, stimulants, transfusion.

21. **Sulphuretted Hydrogen.**—Headache, vomiting, diarrhœa. In severe cases, unconsciousness, dyspnœa, cyanosis, convulsions, and death. The blood is thin, fluid, and black (sulph-hæmoglobine). *Treatment:* Artificial respiration, fresh air, and the cautious inhalation of chlorine gas.

22. **Bisulphide of Carbon.**—(Workers in rubber factories.) Vomiting. Severe nervous symptoms. Incontinence of urine, atrophic paralyses, anæsthesia, mental disturbances, especially loss of memory, spasmodic conditions, etc. The red blood-corpuscles are destroyed; the black blood contains many flakes of pigment. *Treatment:* Symptomatic.

23. **Hydrocyanic Acid** (Potassic Cyanide; Bitter Almonds).—Characteristic odor of bitter almonds. In severe cases death may occur in a few minutes. If the course be more protracted, convulsive and extremely slow respiration, the expiratory act being much prolonged; the eyeballs protrude, and the pupils are somewhat enlarged and do not react to light. Cardiac weakness, cyanosis, unconsciousness. Twitching of the muscles. Trismus. *Treatment:* Merely symptomatic. Emetics, artificial respiration, cool douches, stimulants. Atropine may be tried; also, hydrated ferric oxide and chlorine water.

24. **Nitrobenzine** (Nitrobenzole, Oil of Mirbane).—Strong odor of bitter almonds. At first, dizziness. The skin soon assumes a bluish hue, rapidly increasing to the deepest cyanosis. Increasing anxiety, sense of suffocation, and gradual

loss of consciousness. In severe cases, death, ushered in by convulsions. In milder cases, gradual recovery. *Treatment:* Stomach-pump. Artificial respiration. Stimulants. In the Leipsic clinique, transfusion has worked admirably in two cases. The symptoms caused by aniline and the aniline dyes closely resemble poisoning from nitrobenzole.

25. **Carbolic Acid, also Lysol and the like.**—Corrosion of mouth, throat, and stomach. In mild cases, vertigo, headache, and collapse; in severe cases, coma, preceded in rare instances by symptoms of cerebral irritation. Contracted pupils. Vomiting. Pulse slow at first, then rapid. The urine has a dark, olive-green color. Sometimes hæmoglobinuria and nephritis. Carbolic acid is very readily absorbed from the rectum, so that great caution should be employed in its use as an injection. *Treatment:* Stomach-pump. Slaked lime and water. Large doses of sulphate of sodium are especially to be recommended. [Alcohol is an antidote. Administer three or four ounces of whisky at once, then wash out the stomach with water containing sulphate of sodium. In desperate cases, venesection followed by infusion of normal salt solution has been successful.—V.]

26. **Atropine** (*Belladonna*).—Dryness of the mouth and throat. Excessive thirst. Dizziness and headache. Peculiar mental disturbances: hallucinations are particularly frequent. Pupils very widely dilated. Cutaneous erythema resembling that of scarlet fever. In severe cases, pulse enormously accelerated, with violent pulsation in the arteries. Convulsions may occur. Nervous symptoms persist for some time. *Treatment:* The following physiological antidotes should be tried: physostigmine (eserine), pilocarpine, and morphine.

27. **Digitalis.**—Vomiting. Diarrhœa. Pulse very slow (forty beats per minute, or less). Dyspnœa. Symptoms of collapse. Cold extremities, muscular tremor. Somnolence. In the worst cases, sopor and death. Even the milder cases are protracted. *Treatment:* Emetics, stomach-pump. Tannin. Camphor, strong *café noir,* ether, ammonia. Counter-irritation.

28. **Nicotine** (Smoking; Tobacco Juice; Tobacco Enemata).—1. *Acute:* Pulse small and slow, syncope, sense of oppression, salivation, vomiting. In severe cases, loss of consciousness, tetanic spasms, both pulse and respiration intermittent. 2. *Chronic* (from excessive use of tobacco): Palpitation, irregular action of the heart, paroxysms of asthma and angina pectoris. Tremor, muscular weakness. Loss of sleep. Sometimes there are symptoms of ataxia ("nicotine tabes" seen in cigar makers). Amblyopia, scintillating scotoma. Gastric disturbance, chronic catarrh of the pharynx and larynx. *Treatment:* In acute cases, stimulants. Chronic poisoning necessitates the giving up of tobacco. Further treatment is symptomatic.

29. **Strychnine.**—Violent tetanic reflex convulsions. Exaggeration of the cutaneous and tendon reflexes. Trismus. Opisthotonos. Pulse small and very rapid. The convulsions come in paroxysms, with intervals between them. The intellect is usually perfectly clear. Recovery occurs only in mild cases. *Treatment:* Emetics, stomach-pump. Tannin. Tincture of iodine. Castor-oil. The convulsions are to be combated by morphine, chloroform, chloral, or potassic bromide. Curare has also been tried.

30. **Coniine** (Hemlock).—In severe cases convulsions, then general paralysis, especially of the respiratory muscles, loss of consciousness, and death. Pupils dilated. In milder cases confusion, muscular weakness, vomiting, and diarrhœa. *Treatment:* Emetics, tannin, stimulants.

31. **Morphine** (Opium).—1. *Acute:* Begins with fatigue, headache, darkening of the visual field, nausea, vomiting. Then coma comes on with slow, stertorous, sometimes irregular respiration. Muscles completely lax. Pupils usually very small. Pulse often slow, but in other cases rapid and small. Toward the end of life Cheyne-Stokes's respiration. In milder cases only vomiting, stupor,

headache, etc. *Treatment:* Sulphate of zinc, or some other emetic; stomach-pump. [One-per-cent. solution of permanganate of potassium.—V.] Tannin. *Café noir.* Atropine may be tried as a physiological antidote. Stimulants (camphor, ether) are best, also cold baths with shower-baths; artificial respiration. 2. *Chronic* (morphine habit): Emaciation, anæmia, headache, vertigo, wakefulness. Tremor. Mental disturbance. Unconquerable longing for morphine; and, if this be denied, the appearance of grave symptoms. To break up the morphine habit is almost impossible except in hospitals and special asylums. The withdrawal of the drug is abrupt according to the practice of some, and gradual according to others. For particulars consult monographs.

32. **Ergot** (Ergotine).—1. *Acute:* At first nausea, vomiting, colic, and diarrhœa. Then vertigo, headache, and muscular weakness. Pulse slow. In severe cases, sopor, disturbance of respiration, and sometimes death. *Treatment:* Emetics and purgatives. Tannin. Ether, camphor, and *café noir* as stimulants. 2. *Chronic ergotism:* Gastric symptoms, vertigo, languor, cardiac weakness. The nervous disturbances are, however, of especial importance. Of these, paræsthesia has long been recognized. Recently attention has been attracted to the great resemblance of the nervous symptoms to those of tabes dorsalis; and there is, moreover, an anatomical change in the posterior columns of the cord. Convulsions and psychical phenomena are also observed. A second form of chronic ergotism is called gangrenous ergotism. It results in dry gangrene of the hands and feet. A line of demarcation forms and the gangrenous parts slough off. The process may be attended by fever and pyæmia. The probable explanation is that the minute blood-vessels become spasmodically contracted and thrombi form under the influence of the poison. The different symptoms are due in part to the different constituents of the ergot. The best known are sphacelinic acid, which is probably the cause of gangrenous ergotism and ergotine tabes; cornutine, which causes severe convulsive symptoms (convulsive ergotism) and uterine contractions; and finally ergotinic acid. The *treatment* of chronic ergotism is purely symptomatic.

33. **Poisonous Mushrooms.**—1. *Poisoning from morels:* Fresh morels ("morcheln" or "lorcheln") contain a poison which is readily soluble in hot water, and which evaporates completely if the morels be dried. Morels that have been dried or parboiled are therefore perfectly harmless, but the fresh ones are poisonous. The symptoms are nausea, vomiting, diarrhœa, headache, coma, and, above all, hæmoglobinæmia and hæmoglobinuria (*q. v.*), associated with which is a hæmatogenous icterus. In severe cases death occurs, ushered in by convulsions. *Treatment* is symptomatic, and includes the administration of emetics, purgatives, and stimulants. 2. *Poisoning from the red agaric (amanita muscaria).* This contains the poisonous alkaloid muscarine. Gastric symptoms and diarrhœa. Mental excitement, delirium, tetanic and epileptiform convulsions. A rapid pulse, small pupils, disturbed vision from spasm of accommodation, sweating, salivation, and in most of the severe cases sopor and death. *Treatment:* Emetics, etc. Atropine, which acts as a physiological antidote to muscarine. Also tannin, and stimulants. 3. *Poisoning from bulbous mushrooms (amanita phalloides),* confused with young button mushrooms (champignons). Digestive disturbances, later jaundice, somnolence, coma. The autopsy shows fatty degeneration of the liver, kidneys, and stomach, as in phosphorus poisoning.

34. **Poisoning from Sausages** (*Botulismus*).—This sometimes occurs as the result of eating partially decayed sausages. The symptoms are pain in the stomach, nausea, vomiting, colic, and diarrhœa. There are also marked feebleness, præcordial anxiety, and dyspnœa; vertigo, headache, somnolence; and very often disturbance of vision (amblyopia, spots before the eyes), and, what is surprising, ptosis. In severe cases, dysphagia, as a result of more or less complete paralysis

of the tongue and the constrictors of the pharynx. The mouth is dry. The heart is feeble; this and the general prostration and malnutrition may prove fatal. The cases are usually protracted, rarely being very acute. The active principles (alkaloids of putrefaction) have lately become known in part. In sausage and also in meat poisoning the most important substance is ptomato-atropine, a substance which acts almost exactly like atropine. *Treatment:* Emetics, purgatives (calomel), stimulants, and, if indicated, artificial feeding.

35. **Poisoning from Meat.**—In repeated instances severe symptoms have been occasioned by eating tainted meat, or possibly that obtained from animals which had been diseased. These symptoms are certainly due to the products of putrefaction, substances which act partly like muscarine, partly like atropine, neurine, methylguanidine, etc. The special poison is not yet fully known. Probably there are several poisons, either chemical or organic and infectious. The usual symptoms are vomiting and diarrhœa. The case may closely simulate cholera. In most instances certain nervous phenomena are also observed—wakefulness, delirium, headache, and changes in the pupils. There may be roseola, wheals or erythema. Frequently there is a high fever, but sometimes the temperature is subnormal. The pulse is small and slightly accelerated, although it may occasionally be slower than normal. There is a sense of thoracic oppression. The cases are often protracted. Tendency to relapses. Death may occur. Post mortem, there is usually found an intense and often hæmorrhagic enteritis with secondary changes in the spleen, kidneys, lungs, and other organs. *Treatment:* Symptomatic: calomel, emulsions, stimulants, and baths. Food should be given cautiously.

36. **Poisoning from Fish.**—The eating of tainted fish has likewise caused grave disturbance. The symptoms vary. Usually there are pain in the stomach, præcordial anxiety, vertigo, dryness of the throat, aphonia, and labored respiration. There may also be disturbance of vision, amblyopia, and colored vision, or paralysis of the motores oculi and of accommodation. In severe cases there may be dysphagia and general paresis. Sometimes dyspnœa and cardiac weakness are observed. The cases are frequently very tedious. Here, too, there are many active poisonous products of putrefaction, among them one which acts like muscarine. *Treatment* similar to that recommended in the two preceding paragraphs.

37. **Poisoning from Mussels** (*Mytilus Edulis*).—This is also common. A drawing feeling in the neck, a blunt feeling in the teeth, crawling and burning in the arms and legs, dullness in the head, mental excitability, feeling as if everything were light, as if the patient must fly; in the later stages dilated, immobile pupils, difficult speech, paresis and ataxia of the muscles; also nausea, skin eruptions (urticaria), fall of temperature. In severe cases death may ensue in a few hours. At the autopsy we find severe enteritis, enlargement of the spleen, and often a peculiar speckling of the liver. The poison of mussels has been isolated, and it is called mytilotoxine.

38. **Poisoning from Cheese.**—Vomiting, colicky pains, diarrhœa, vertigo, sense of thoracic oppression, headache, languor, and disturbance of vision. *Treatment* as in paragraphs 35 and 36.

APPENDIX II

TABLE OF WEIGHTS AND MEASURES

Table of Relation of U. S. Fluid to Metric Measure

Minims.		Cubic Centimetres.	Minims.		Cubic Centimetres.	Fluid Drachms.		Cubic Centimetres.	Fluid Ounces.		Cubic Centimetres.
1	=	.06	30	=	1.85	4	=	14.79	4	=	118.24
2	=	.12	40	=	2.46	6	=	22.18	6	=	177.39
5	=	.31							8	=	236.53
10	=	.62	**Fluid Drachms.**		**Cubic Centimetres.**	**Fluid Ounces.**		**Cubic Centimetres.**	12	=	354.82
15	=	.92	1	=	3.70	1	=	29.57	16	=	473.11
16¼	=	1.00	2	=	7.39	2	=	59.10			
20	=	1.23	3	=	11.09	3	=	88.67			

Table of Relation of Troy Weight to Grammes

Grains.		Grammes.	Grains.		Grammes.	Drachms.		Grammes.	Ounces.		Grammes.
⅛	=	.008	8	=	.52	1	=	3.89	1	=	31.1
⅙	=	.011	10	=	.65	1½	=	5.83	1½	=	46.6
¼	=	.016	15	=	.97	2	=	7.77	2	=	62.2
½	=	.032	15.43	=	1.00	3	=	11.66	3	=	93.3
1	=	.065	20	=	1.29	4	=	15.55	4	=	124.4
2	=	.13	30	=	1.94	6	=	23.3	6	=	186.6
4	=	.26	40	=	2.59				8	=	248.8
5	=	.32	61.73	=	4.00						
6	=	.39									

Table of Comparison between Centigrade and Fahrenheit Scales

Degrees Centigrade.		Degrees Fahrenheit.	Degrees Centigrade.		Degrees Fahrenheit
25	=	77	36	=	96.8
26	=	78.8	37	=	98.6
27	=	80.6	38	=	100.4
28	=	82.4	39	=	102.2
29	=	84.2	40	=	104
30	=	86	41	=	105.8
31	=	87.8	42	=	107.6
32	=	89.6	43	=	109.4
33	=	91.4	44	=	111.2
34	=	93.2	45	=	113
35	=	95			

THE METRIC SYSTEM IN MEDICINE

Old Style.		Metric.	
♏ j or gr. j	=		06 grm.
f ʒ j or ʒ j	=	4	"
f ℥ j or ℥ j	=	32	"

The decimal *line*, instead of *points*, makes errors impossible. As .06 (drug) is less than a grain, while 4. and 32. (vehicle) are more than the drachm and ounce, there is no danger of giving too large doses of strong drugs.

C. c. (cubic centimetres), used for grms. (grammes), causes an error of five per cent. (excess).

A teaspoonful is usually five grammes; a tablespoonful, twenty grammes.

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